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Original Article

Physical activity, eating patterns, and insulin resistance in obesity

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Abstract

Background Unhealthy eating patterns and physical inactivity are associated with obesity. Insulin resistance, an early sign of type 2 diabetes mellitus, is common in obese individuals.

Objective To assess for an association between physical activity and eating patterns to insulin resistance in obese female adolescents.

Methods Subjects were 77 obese female adolescents aged 13-15 years. Peripheral blood specimens were obtained for measurements of fasting blood glucose, insulin, and the homeostatic model assessment (HOMA) index. Subjects were considered to be in a state of insulin resistance for HOMA index ≥3.16. Physical activity recalls for 7 x 24h were performed to obtain information on subjects' energy expenditure, as well as intensity and duration of physical activity. Data on nutrients intake was also obtained using 7 x 24h food recalls. Fat intake was categorized into saturated fatty acid (SAFA), unsaturated fatty acid (UNSAFA), monounsaturated fatty acid (MUFA), and polyunsaturated fatty acid (PUFA).

Results Subjects spent most of their time on light activities, 23.5 (SD 2.0) hours/day. Those with insulin resistance spent slightly more time doing light activities. Conversely, they spent less time in vigorous activities, a mean difference of 4.96 (95%CI 0.61 to 9.31) minutes/day (P=0.01). Subjects who spent less than 1 minute/day in heavy activities had higher odds for insulin resistance, (OR 3.14; 95%CI 1.20 to 8.50; P=0.02). There were no relationships found between nutrients intake, such as energy, protein, fats (SAFA, UNSAFA, MUFA, and PUFA), or carbohydrates, and insulin resistance.

Conclusion In obese female adolescents, physical inactivity is associated with insulin resistance, while nutrients intake is not associated with insulin resistance. **[Paediatr Indones. 2014;54:82-7.]**

Keywords: *insulin resistance, obesity, physical activity, energy, protein, fat, carbohydrate, female adolescents*

Obesity and overweight are increasing, both in developed and developing countries. The prevalence of overweight children in the United States increased from 4% in the early 1970s to 15% in 1999-2000.[1] In the Asia-Pacific region, the prevalence of overweight and obesity ranged from less than 5% in India to 60% in Australia. The prevalence in China was only one-third that of Australia, but a 400% increment of prevalence has been observed over the past 20 years.[2] In Indonesia, the 2007 National Basic Health Research reported that the national prevalence of under-fives was 12.2%, with weight-for-height standard deviation scores above 2.0. In adults (≥15 years), the prevalence of BMI above 27 kg/m^2 was 10.3%, with about 18.8% of these having central obesity (waist circumference of >90 cm for males and >80 cm for females). A greater prevalence of central obesity was seen in females (29%) compared to males (7.7%).[3]

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Obesity is known to be a risk factor for diabetes mellitus and cardiovascular diseases.[4] Together, they are parts of a condition called metabolic syndrome, which is strongly associated with insulin resistance.[5] Several studies have attempted to elucidate the relationship between lifestyle, obesity, and insulin resistance. *The Framingham Offspring Cohort Study* reported that intake of total fiber, cereal fiber, fruit fiber, and whole grains was negatively related to insulin resistance, and that glycemic index and glycemic load were positively related to insulin resistance.[6] Fat intake, especially of saturated fatty acids, was associated with insulin resistance, however, intake of mono-unsaturated fatty acid was not.[7]

Although some studies reported benefits of physical activity on insulin sensitivity, findings remain inconclusive.[8-10] To date, there have been few studies on the association between food intake, physical activity, and insulin resistance in Indonesia. This study was conducted to assess for an association between physical activity and eating patterns to insulin resistance in obese female adolescents.

Methods

We conducted a cross-sectional study involving 6 junior high schools in the city of Yogyakarta in 2007. We screened 2,120 female students aged 13 to 15 years to identify obese female adolescents.

Obesity was defined as BMI-for-age $\geq 95^{th}$ percentile, based on the 2000 Centers for Disease Control reference standard.[11] Out of 136 obese female adolescents screened, 77 consented to participate in the study. Subjects were interviewed to identify their daily activities and eating patterns for 7 x 24 hour-recall periods. Blood specimens (± 5 mL) were collected in EDTA-anticoagulant following fasting for at least 8 hours. Anthropometric measurements, as well as food and activity recalls were performed by six students from the School of Nutrition and Health, Gadjah Mada University Medical School, who underwent training and standardization of measurements prior to data collection. Blood sampling was performed by laboratory technicians.

This study was approved by Medical and Health Research Ethics Committee, Gadjah Mada University Medical School.

Subjects' weights and heights were measured at school, while wearing light clothing, and without shoes, socks, or hats. Weight was measured using an electronic digital scale (*Camry*) that had been calibrated by the Metrology Institution to an accuracy of 0.1 kg. Height was measured using a microtoise with an accuracy of 0.1 cm. Measurements were done in triplicate and averaged. BMI was calculated using the formula: weight (kg)/[height $(m)^2$].

Laboratory assessments were performed in the Clinical Pathology Laboratory of Gadjah Mada University, Yogyakarta. Fasting blood glucose was measured by hexokinase test, and insulin was measured using an immunoassay. Insulin resistance was assessed using the HOMA index: 1/405 x [fasting insulin concentration (mU/mL) x fasting glucose concentration (mg/dL)]. A HOMA index of ≥ 3.16 was considered to indicate insulin resistance.[12]

All subjects were interviewed to recall all activities performed and their duration, e.g., breakfast 6:00-6:15am, walking to school 6:30-7:00am, sitting in class 7:00-9:00am, playing football 9:00-9:30am, sleeping 10:00pm-5:00am, in the previous 24 hours. Interviews were done seven times, i.e., six recalls for six working days and one recall for one weekend day. This data was converted into metabolic equivalent (MET). Units of MET represented the intensity of the physical activity. Higher MET values indicated higher intensity physical activity. One MET unit was equivalent to resting metabolic rate (RMR) or the activity of sitting quietly.[13] Duration of activities was recorded in minutes. Therefore, the unit of energy expenditure in a certain time period was expressed in METs-min. The average of the seven physical activity recalls was the daily energy spent in METs-min. All activities were then classified into an intensity group, i.e., light, medium, or heavy. An activity was classified as light for <3 METs, medium for 3-6 METs, and heavy for >6 METs.[13]

We used 24-hour food recalls to assess the types and amounts of food consumed within the previous 24 hours. To have a better estimate of the amount of food consumed, we employed the help of food models and household measures, such as spoons, drinking glasses and plates. The required frequency of recalls for quantification of energy, protein, fat, and carbohydrate intake was estimated using the formula introduced by Beaton *et al.*:[14]

$n = (Z_\alpha \times Cv_w/D_o)^2$

[n: number of day(s) required; Z_α: normal deviation for the percentage of times the measured value should be within a specified limit (usually 1.96); Cv_w: coefficient of intra-individual variation; D_o: tolerance].

The CV_w value from fat (39.3 %) was used because the variation of fat intake was the highest. With a tolerance of 30%, the minimum number of days required for analysis was six days. Data on food intake were converted to nutrient intake using *NutriSurvey for Windows* that was adapted for use in Indonesia.

Independent T-test was used to compare physical activity and nutrients consumed between subjects with and without insulin resistance. Data were reported in mean differences (95%CI). Odds (95%CI) for insulin resistance in high-risk subjects were also reported. Pearson's correlation was used to analyze nutrient intake and HOMA index.

Results

Insulin resistance was found in 44 out of 77 obese female adolescents (57%). Clinical and demographic characteristics of subjects are presented in **Table 1**. Subjects' physical activity recalls are shown in **Table 2**. These obese adolescents spent most of their time doing light activities for a mean 23.5 (SD 2.0) hours/day. Although not statistically significant, subjects with insulin resistance spent more time doing light activities (**Table 2**).

There was no significance difference in energy expenditure between the insulin resistant and the non-insulin resistant groups. However, subjects with insulin resistance spent less time doing heavy intensity activities, compared to those without insulin resistance, [mean difference of 4.96 (95% CI 0.61 to 9.31) minutes/day; (P=0.03)] (**Table 2**). On average, subjects spent only 4.75 (SD 9.74) minutes/day on heavy intensity activities, and 63.6% of them did heavy intensity activities for less than 1 minute/day. These individuals had a higher risk for insulin resistance [OR 3.14; (95%CI 1.20 to 8.50); (P=0.02)].

There were no significant differences in energy intake between the two groups, including protein intake, either in grams or % energy, or in types of fats consumed (**Table 3**). Correlation analysis did not reveal associations between intake of calories, protein, fats, or carbohydrates to the HOMA index (**Table 4**).

Table 1. Clinical and demographic characteristics of subjects

Characteristics	Insulin resistance (n=44)	Non-insulin resistance (n=33)
Mean age (SD), years	13.82 (0.85)	13.56 (0.96)
Mean BMI (SD), kg/m^2	30.18 (2.63)	29.09 (2.40)
Mean HOMA index (SD)	5.25 (3.82)	2.30 (0.68)
Mean fasting insulin (SD), mU/mL	22.86 (12.10)	11.13 (3.39)
Mean fasting glucose (SD), mg/dL	91.14 (21.69)	84.73 (11.02)

Table 2. Pattern of physical activities in subjects with and without insulin resistance

	Insulin resistance (n = 44)	Non-insulin resistance (n = 33)	Mean difference (95%CI)	P value*
Mean energy expenditure (SD), Mets-min/day	1,915.26 (123.87)	1,938.98 (116.15)	27.78 (-31.76 to 79.07)	0.40
Mean duration of heavy activities (SD), min/day	2.63 (7.34)	7.59 (11.76)	4.96 (0.61 to 9.31)	0.03
Mean duration of medium activities (SD), min/day	23.172 (9.40)	30.51 (29.32)	7.34 (-6.13 to 20.81)	0.28
Mean duration of light activities (SD), hrs/day	23.57 (0.49)	23.38 (0.50)	0.19 (-0.42 to 0.03)	0.09

* Independent sample T-test

Table 3. Pattern of food intake in subjects with and without insulin resistance

	Insulin resistance (n = 44) mean (SD)	Non-insulin resistance (n = 33) mean (SD)	Mean difference (95%CI)	P value
Energy, kcal	1,115.48 (284.36)	1,179.19 (337.97)	-63.72 (-205.18 to 77.75)	0.37
Fat				
Total amount intake, g	39.82 (12.45)	45.10 (15.39)	-5.28 (-11.60 to 1.05)	0.10
% energy	32.01 (5.79)	34.13 (4.64)	-2.12 (-4.57 to 0.32)	0.09
Carbohydrates				
Total amount intake, g	150.25 (43.01)	152.46 (44.13)	-10.02 (-22.16 to 17.74)	0.83
% energy	53.85 (6.48)	51.96 (5.33)	1.89 (-0.79 to 4.57)	0.18
Total protein				
Total amount intake, g	39.10 (10.17)	41.21 (13.41)	-2.11 (-7.46 to 3.24)	0.44
% energy	14.20 (2.43)	13.96 (2.10)	0.24 (-0.81 to 1.30)	0.65
Saturated fatty acids				
Total amount intake, g	17.65 (7.88)	18.79 (6.77)	-1.14 (-4.55 to 2.27)	0.51
% energy	13.80 (3.50)	14.63 (3.57)	-0.83 (-2.45 to 0.80)	0.31
Unsaturated fatty acids				
Total amount intake, g	17.42 (5.98)	17.95 (5.33)	-0.54 (-3.16 to 2.08)	0.68
% energy	13.81 (3.05)	14.23 (3.33)	-0.42 (-1.88 to 1.03)	0.56
Monounsaturated fatty acids				
Total amount intake, g	10.17 (3.63)	10.73 (2.97)	-0.56 (-2.10 to 0.98)	0.47
% energy	8.06 (1.97)	8.50 (1.66)	-0.43 (-1.28 to 0.41)	0.31
Polyunsaturated fatty acids				
Total amount intake, g	7.25 (3.11)	7.23 (2.90)	0.02 (-1.37 to 1.41)	0.98
% energy	5.75 (1.91)	5.74 (2.11)	0.01 (-0.90 to 0.93)	0.98

Table 4. Pearson's correlation of energy intake vs. HOMA index

Pattern of energy intake	HOMA	
	r	P value
Energy intake, kcal	-0.17	0.15
% energy from protein	0.04	0.74
% energy from fat	-0.14	0.23
% energy from unsaturated fat	-0.03	0.78
% energy from saturated fat	-0.12	0.29
% energy from carbohydrates	0.09	0.39

Discussion

We found a higher prevalence (57%) of insulin resistance in obese adolescents than that observed (25%) in French adolescents.[12]

Although daily energy expenditure did not significantly differ between the insulin and non-insulin resistant groups, we observed that subjects with insulin resistance had, on average, a shorter duration of heavy intensity activities compared to those without insulin resistance. An American study showed a significant, positive correlation between daily energy expenditure and insulin sensitivity, as assessed by a euglycemic insulin clamp, a more accurate method to assess insulin sensitivity than the HOMA index.[15] However, that method is impractical for use in an epidemiological study.

Physical exercise lowers fasting insulin levels and increases insulin sensitivity through increased glucose transport into muscle cells and increased glycogen deposits in muscles to replace the glycogen lost in exercise. Another beneficial effect of physical exercise is the increase in fat-free mass that increases muscle volume and glucose transfer to muscles. Weight loss due to physical activity decreases insulin resistance in obese adolescents.[16] As a result, physical inactivity was associated with insulin resistance. Although not statistically significant, we observed that subjects with insulin resistance had longer durations of sedentary activities.

A study reported the association between energy intake and insulin resistance, suggesting that that a negative energy balance would increase insulin sensitivity.[17] However, we found no significance difference in energy intake between the insulin resistant and non-insulin resistant groups.

Protein intake, in this case, high protein intake, can decrease glucose response. A study on hyperinsulinemic obese men and women showed a significant glucose

response with a high protein diet (27% protein, 29% fat, and 44% carbohydrate) compared to a standard diet (16%protein, 27% fat, and 57% carbohydrate). In our study, protein intake did not correlate with insulin resistance.[18] Protein-containing foods stimulate insulin by different means. Lavigne *et al.* found a correlation between protein intake and insulin sensitivity. Cod protein prevented insulin resistance in muscles of obese rats, as cod protein has a higher glucose disposal rate compared to that of casein and soy protein.[19] Milk and whey have an ability to rapidly increase insulin secretion, despite causing a low glucose response (glycemic index = 30, insulinemic index = 90).[20]

We did not observe any association between fat or carbohydrate intake and insulin resistance, neither in absolute amounts nor in percentage of energy derived from fat or carbohydrates. Vessby *et al.*[7] compared a saturated fatty acid diet to a monounsaturated fatty acid diet (SAFA diet: 17% energy; MUFA diet: 14% energy; PUFA: 17% energy) (MUFA diet: saturated fatty acid 8% energy; monounsaturated fatty acid 23% energy; polyunsaturated fatty acid 6%). The SAFA diet caused a decrease in the insulin sensitivity index (Si) of 10% ($P < 0.05$), but the Si remained the same for those on the MUFA diet.[7] Previous studies have shown that type of fat consumed daily affected serum lipid concentration. Patients with type 2 diabetes mellitus had a larger proportion of saturated fatty acid and lower linoleic acid compared to normal individuals. Diet also affected the composition of fatty acid in muscles, which is also influenced by physical activity and muscle fiber composition.[7,21]

Insulin sensitivity is determined by fat oxidative capacity. Accumulation of fatty acid metabolites in the cytosol, such as diacylglycerol and ceramide, disturbs the first step in insulin signaling. Diacylglycerol inhibits early signaling events through promoting serin phosphorylation from Insulin receptor substrate 1 (IRS-1), while ceramide is known to inhibit insulin stimulation to Akt (protein kinase B). In active individuals, an increase in the ability to oxidize muscle fat and fatty acid metabolites while exercising, is recognized as an important factor in the association between muscular fat oxidative capacity and insulin sensitivity.[22]

In conclusion, we observe that longer duration of heavy intensity physical activity is associated with better insulin sensitivity. In obese female adolescents, physical inactivity is associated with insulin resistance, while no association is found between nutrients intake with insulin resistance.

References

1. Lederman SA, Akabas SR, Moore BJ. Editors' overview of the conference on preventing childhood obesity. Pediatrics. 2004;114:1139-45.
2. Asia Pacific Cohort Studies Collaboration. The burden of overweight and obesity in the Asia-Pacific region. Obes Rev. 2007;8:191-6.
3. Report on national basic health research (Riskesdas). Jakarta: Institute of Research and Development (Badan Penelitian dan Pengembangan Kesehatan), Ministry of Health, Republic of Indonesia; 2007. p.50-3.
4. Sharma AM, Chetty VT. Obesity, hypertension and insulin resistance. Acta Diabetol. 2005;42:s3-8.
5. Huang PL. A comprehensive definition for metabolic syndrome. Dis Model Mech. 2009;2:231-7.
6. McKeown NM, Meigs JB, Liu S, Saltzman E, Wilson PW, Jacques PF. Carbohydrate nutrition, insulin resistance, and the prevalence of the metabolic syndrome in the Framingham Offspring Cohort. Diabetes Care. 2004;27:538-46.
7. Vessby B, Uusitupa M, Hermansen K, Riccardi G, Rivellese AA, Tapsell LC, *et al*. Substituting dietary saturated for monounsaturated fat impairs insulin sensitivity in healthy men and women: the KANWU study. Diabetologia. 2001;44:312-9.
8. Janssen I, Leblanc AG. Systematic review of the health benefits of physical activity and fitness in school-aged children and youth. Int J Behav Nutr Phys Act. 2010; 7:40.
9. Ferguson MA, Gutin B, Le NA, Karp W, Litaker M, Humphries M, *et al*. Effects of exercise training and its cessation on components of the insulin resistance syndrome in obese children. Int J Obes Relat Metab Disord. 1999;22:889-95.
10. Bell LM, Watts K, Siafarikas A, Thompson A, Ratnam N, Bulsara M, *et al*. Exercise alone reduces insulin resistance in obese children independently of changes in body composition. J Clin Endocrinol Metab. 2007;92:4230-5.
11. Kuczmarski RJ, Ogden CL, Guo SS, Grummer-Strawn LM, Flegal KM, Mei Z, *et al*. 2000 CDC growth charts for the United States: methods and development, National Center for Health Statistics. Vital Health Stat 11. 2002;246:1–190.

12. Klein-Platat C, Drai J, Oujaa M, Schlienger J, Simon C. Plasma fatty acid composition is associated with the metabolic syndrome and low-grade inflammation in overweight adolescents. Am J Clin Nutr. 2005;82:1178–84.
13. Ainsworth BE, Haskell WL, Whitt MC, Irwin ML, Swartz AM, Strath SJ, *et al.* Compendium of physical activities: an update of activity codes and MET intensities. Med Sci Sports Exerc. 2000;32:S498-504.
14. Beaton GH, Milner J, Corey P, McGuire V, Cousins M, Stewart E, *et al*. Sources of variance in 24-hour dietary recall data: implication for nutrition study design and interpretation. Am J Clin Nutr. 1979;32:2546-59.
15. Schmitz KH, Jacobs DR Jr, Hong CP, Steinberger J, Moran A, Sinaiko AR. Association of physical activity with insulin sensitivity in children. Int J Obes Relat Metab Disord. 2002;26:1310-6.
16. Reinehr T, Kiess W, Kapellen T, Andler W. Insulin sensitivity among obese children and adolescents, according to degree of weight loss. Pediatrics. 2004;114:1569-73.
17. Assali AR, Ganor A, Beigel Y, Shafer Z, Hershcovici T, Fainaru M. Insulin resistance in obesity: body-weight or energy balance? J Endocrinol. 2001;171:293-8.
18. Farnsworth E, Luscombe ND, Noakes M, Wittert G, Argyiou E, Clifton PM. Effect of a high-protein, energy-restricted diet on body composition, glycemic control, and lipid concentrations in overweight and obese hyperinsulinemic men and women. Am J Clin Nutr. 2003;78:31–9.
19. Lavigne C, Tremblay F, Asselin G, Jacques H, Marette A. Prevention of skeletal muscle insulin resistance by dietary cod protein in high fat-fed rats. Am J Physiol Endocrinol Metab. 2001;281:E62–71.
20. Nilsson M, Stenberg M, Frid AH, Holst JJ, Björck IME. Glycemia and insulinemia in healthy subjects after lactose-equivalent meals of milk and other food proteins: the role of plasma amino acids and incretins. Am J Clin Nutr. 2004;80:1246–53.
21. Borkman M, Storlien LH, Pan DA, Jenkins AB, Chisholm DJ, Campbell LV. The relation between insulin sensitivity and the fatty-acid composition of skeletal-muscle phospholipids. N Engl J Med. 1993;328:238-44.
22. Rimbert V, Boirie Y, Bedu M, Hocquette JF, Ritz P, Morio B. Muscle fat oxidative capacity is not impaired by age but by physical inactivity: association with insulin sensitivity. FASEB J. 2004;18:737-9.