# Metabolic Syndrome in Obese Thai Children and Adolescents

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***Background:*** *The prevalence of childhood obesity in Thailand is increasing. Obese children are at risk of metabolic syndrome.*
***Objective:*** *To determine the prevalence of metabolic syndrome in obese Thai children with various degrees of obesity and its association with severity of obesity, insulin resistance and C-reactive protein.*
***Material and Method:*** *A cross-sectional study of 89 obese Thai children and adolescents was conducted at the Department of Pediatrics, Faculty of Medicine, Ramathibodi Hospital, Mahidol University. Family histories of diabetes mellitus, hypertension, obesity and dyslipidemia were assessed. Anthropometry and cardiovascular risks including levels of fasting blood sugar, oral glucose tolerance test, insulin, C-reactive protein (CRP) and lipid profile were determined. Metabolic syndrome was defined using International Diabetes Federation criteria adjusted for age and sex. Univariate and logistic regression analysis were used for identification of the independent associated factors.*
***Results:*** *The overall prevalence of metabolic syndrome in the present study was 16.9%. The percentages of metabolic syndrome in subjects with moderate, severe and morbid obesity were 10.5, 23.1 and 22.2 respectively. Univariate analysis revealed that metabolic syndrome had a statistically significant association with insulin level over 25 microIU/mL, homeostasis model for assessment of insulin resistance (HOMA-IR) equal to 3.16 or more and CRP over 3 mg/L. Logistic regression analysis revealed that only insulin level over 25 microIU/mL was independently associated with metabolic syndrome (OR 7.24; 95% CI: 2.01-26.10).*
***Conclusion:*** *The prevalence of metabolic syndrome is high among obese Thai children and adolescents. Prevention and proper management of metabolic syndrome including treatment of obesity should be considered in obese children.*

***Keywords:*** *Metabolic syndrome, Insulin resistance, Obesity, Children, Adolescent, Thai*

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Childhood obesity and obesity related comorbidities are increasing worldwide and rank among the most important public health problems facing the world today[1,2]. The important obesity related comorbidities are metabolic and cardiovascular complications. Metabolic syndrome (MS) has been described since 1988 as a cluster of potential risk factors for atherosclerotic cardiovascular disease and type 2 diabetes mellitus in adults[3,4].

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The World Health Organization (WHO), the National Cholesterol Education Program (NCEP) Adult Treatment Panel (ATP) III and the International Diabetes Federation (IDF) have recently provided the clinical definitions and criteria of MS[5,6]. These criteria, although similar in their metabolic components such as obesity, dyslipidemia, hyperglycemia and hypertension, differ in the individual threshold values. Key factors in MS pathogenesis are visceral fat and insulin resistance. Many studies in obese patients concerning these factors have shown that free fatty acid (FFA) accumulation in the liver, fat cells, pancreas and skeletal muscle are associated with insulin resistance. Hyperinsulinemia increases triglyceride synthesis and lipolysis resulting in FFA accumulation which worsens insulin resistance and generates vicious

cycle. Hyperinsulinemia also increases renal sodium absorption and sympathetic tone, leading to hypertension. Moreover, pro-inflammatory cytokines from adipose tissue such as interleukin 6, plasminogen activator inhibitor-1, tumor necrotic factor α (TNFα) and C-reactive protein (CRP) are released and play roles in pathogenesis of atherosclerosis and cardiovascular disease[7,8]. Although MS is particularly important in adults, the pathological process and risk factors associated with its development have been shown to begin during childhood[9-15]. MS in adulthood was strongly associated with the presence of MS in childhood, which was also an independent predictor of type 2 DM in adulthood. Previous studies in many countries found a high prevalence of MS among obese children and adolescents which increased with the severity of obesity[16-20]. In Thailand, the prevalence of MS increases in the adult population but data in children and adolescents are scarce[21-23].

In this context, the present study aimed to determine the prevalence of MS in obese children with various degrees of obesity and its relation to severity of obesity, insulin resistance and C-reactive protein.

## Material and Method

### *Study population*

This cross-sectional study was undertaken in the nutrition clinic, Department of Pediatrics, Faculty of Medicine, Ramathibodi Hospital, Mahidol University during the period January-December 2007. The study group consisted of 89 obese children and adolescents (49 males and 40 females). Subjects were eligible if they were healthy, aged between 4-18 years and had body mass index (BMI, weight in kilograms divided by the square of height in meters) above the 95th percentile for their age and sex[24]. Exclusion criteria were the use of medication that altered blood pressure or glucose or lipid metabolism and secondary obesity. The protocol was approved by the ethic committee of the Faculty of Medicine, Ramathibodi Hospital, Mahidol University. Written informed consent from parents was obtained.

### *Procedures*

The subjects were evaluated after a 12-hour, overnight fast. Family histories of diabetes mellitus, hypertension, obesity, and dyslipidemia were assessed. Their weight and height were measured and their BMI were calculated. Blood pressure was measured using auscultation method with mercury sphygmomanometer. Waist circumference (WC) was measured with subjects in standing position, placing the metric strip at the midpoint between the lower ribs and the iliac crests after the normal exhalation. Baseline fasting blood samples were obtained from subjects for measurements of glucose and lipid profile by an enzymatic method, insulin by chemiluminescence immunoassay and C-reactive protein (CRP) by immunonephelometry. An oral glucose tolerance test was then performed with the administration of 1.75 g of glucose per kg of body weight (maximal dose 75 g).

### *Definition of severity of obesity*

The severity of obesity was classified by using the percentage of ideal (the 50th percentile) weight for height into mild (> 120 to 140%), moderate (> 140 to 160%), severe (> 160 to 200%) and morbid obesity (≥ 200%).

### *Definition of metabolic syndrome and its components*

MS was defined by using the International Diabetes Federation criteria adjusted for age and sex[25]. MS was diagnosed by the presence of abdominal obesity plus any 2 of the following; hypertriglyceridemia, low high-density lipoprotein cholesterol (HDL-C), hypertension and impaired fasting glucose or impaired glucose tolerance test. For each of the components of MS, the following cut off values were used: hypertriglyceridemia, triglyceride ≥ 150 mg/dL; low HDL-C, HDL-C < 40 mg/dL; hypertension, systolic blood pressure ≥ 130 mmHg and /or diastolic blood pressure ≥ 85 mmHg; impaired fasting glucose, fasting glucose ≥ 100 mg/dL; impaired oral glucose tolerance test, 2-hour glucose ≥ 140 mg/dL; and abdominal obesity, WC ≥ the 90$^{th}$ percentile for age and sex[26]. Hypercholesterolemia (total cholesterol ≥ 200 mg/dL) and high low-density lipoprotein cholesterol (LDL-C ≥ 130 mg/dL) were defined according to the American Academy of Pediatrics[27].

### *Definition of insulin resistance (IR)*

IR was determined through homeostasis model for assessment of insulin resistance (HOMA-IR) which was calculated using the following equation: [fasting glucose (mmol/L) x fasting insulin (microIU/mL)]/22.5. HOMA-IR value of 3.16 was chosen as the cut-off point to define IR[28]. Hyperinsulinemia was diagnosed when fasting insulin was more than 25 microIU/mL[29,30].

### *Definition of high CRP[31,32]*

The concentration of CRP above 3 mg/L, which has been related to high risk for cardiovascular diseases, was defined as high CRP.

***Statistical analysis***

Data were summarized with descriptive statistics, reported as raw number and proportion for categorical variables; as mean and standard deviation for numerical variables. Comparison of categorical variables was accomplished using Chi-square (substituting Fisher-exact testing when expected cell sizes were less than 5). Univariate and logistic regression was performed to identify variables that were significantly related to the odds of having MS. The results were reported as odds ratios with 95 percent confidence interval. Statistically significant differences were assumed if the p-value was < 0.05. Data were processed with SPSS software version 14.0.

**Results**

Anthropometric and laboratory data were shown in Table 1. For both sexes, the mean age was 11.25 $\pm$ 2.74 years; the mean BMI was 30.09 $\pm$ 6.26 kg/$m^2$ which corresponds to severe obesity; and the mean WC was 91.36 $\pm$ 12.97 cm. All of these parameters were not significantly different between sexes (p-value > 0.05). All subjects in the present study had abdominal obesity. The percentages of mild, moderate, severe and morbid obesity were 3.4, 42.7, 43.8 and 10.1 respectively. The authors classified severity of obesity into 2 groups, *i.e.* group 1 (mild and moderate obesity, n = 41) and group 2 (severe and morbid obesity, n= 48) as shown in Table 2. Most of the subjects had acanthosis nigricans (83.1%).

The prevalence of metabolic and CVS risk factors in this population were as follows (Table 3): hypertension, 20.2%; hypercholesterolemia, 30.3%; high LDL cholesterol, 38.2%; low HDL cholesterol, 28.1%; hypertriglyceridemia, 13.5%; impaired fasting glucose, 3.4%; impaired glucose tolerance, 10.5%; hyperinsulinemia, 27.3%; IR (HOMA-IR $\geq$ 3.16), 58.4% and high CRP, 42.7%. The prevalence of hyperinsulinemia, IR and high CRP were higher in group 2 than group 1 (p-value < 0.05).

The overall prevalence of the MS was 16.9% (Table 3) and its prevalence increased with age (p-value = 0.027) (Table 4). The syndrome was present in 10.5%, 23.1% and 22.2% of children classified as moderate, severe and morbid obesity, respectively. No subject with mild obesity met the criteria for this syndrome. There were no statistically significant differences of MS prevalences among the severity groups (p-value = 0.09). Univariate analysis revealed that insulin level > 25 microIU/mL, HOMA-IR $\geq$ 3.16, and CRP > 3 mg/L

**Table 1.** Anthropometric and laboratory data

| Features | Male | Female | Total |
|---|---|---|---|
| Number (%) | 49 (55.10) | 40 (44.90) | 89 (100) |
| Age (years) | 11.19 $\pm$ 2.76 | 11.31 $\pm$ 2.76 | 11.25 $\pm$ 2.74 |
| Anthropometric data | | | |
| Weight (kg) | 72.14 $\pm$ 29.19 | 69.26 $\pm$ 19.01 | 70.85 $\pm$ 25.03 |
| Height (cm) | 151.26 $\pm$ 16.82 | 149.76 $\pm$ 14.45 | 150.59 $\pm$ 15.73 |
| Waist circumference (cm) | 93.71 $\pm$ 14.40 | 88.49 $\pm$ 10.45 | 91.36 $\pm$ 12.97 |
| % Weight for height | 169.59 $\pm$ 32.29 | 165.58 $\pm$ 21.89 | 167.79 $\pm$ 28.02 |
| BMI ($kg/m^2$) | 30.32 $\pm$ 7.14 | 29.81 $\pm$ 5.05 | 30.09 $\pm$ 6.26 |
| SBP (mmHg) | 117.04 $\pm$ 13.10 | 113.25 $\pm$ 13.36 | 115.34 $\pm$ 13.23 |
| DBP (mmHg) | 75.16 $\pm$ 8.18 | 73.15 $\pm$ 9.13 | 74.26 $\pm$ 8.63 |
| Laboratory data | | | |
| TC (mg/dL) | 184.55 $\pm$ 28.18 | 183.95 $\pm$ 32.25 | 184.28 $\pm$ 29.90 |
| HDL-C (mg/dL) | 45.06 $\pm$ 8.09 | 44.63 $\pm$ 8.41 | 44.87 $\pm$ 8.19 |
| LDL-C (mg/dL) | 118.47 $\pm$ 25.74 | 119.13 $\pm$ 26.00 | 118.76 $\pm$ 25.71 |
| TG (mg/dL) | 109.92 $\pm$ 74.21 | 103.05 $\pm$ 53.50 | 106.83 $\pm$ 65.46 |
| FBS (mg/dL) | 84.67 $\pm$ 7.07 | 85.80 $\pm$ 8.36 | 85.18 $\pm$ 7.66 |
| OGTT 2 h (mg/dL) | 114.13 $\pm$ 19.07 | 121.21 $\pm$ 21.32 | 117.34 $\pm$ 20.31 |
| Insulin (microIU/mL) | 18.42 $\pm$ 13.12 | 20.98 $\pm$ 10.48 | 19.57 $\pm$ 12.00 |
| HOMA-IR | 3.86 $\pm$ 2.75 | 4.51 $\pm$ 2.52 | 4.15 $\pm$ 2.65 |
| CRP (mg/L) | 4.11 $\pm$ 4.72 | 4.26 $\pm$ 4.25 | 4.18 $\pm$ 4.49 |

Mean $\pm$ SD, p-value > 0.05

had statistically significant association with MS (Table 5). Logistic regression analysis revealed that only insulin level > 25 microIU/mL was independently associated with MS (odds ratio 7.24, 95% CI 2.01-26.10) (Table 6).

## Discussion

Since the age specific reference of WC for Thai children has not been established, the authors used the Japanese reference which were also Asian and similar in terms of body size to the Thai population. All subjects in the present study had abdominal obesity according to the Japanese reference[26]. Moreover, the mean WC of all subjects was higher than the upper limit of adult values. Therefore, all subjects were diagnosed as abdominal obesity. Regarding the degree of obesity, the authors used the percentage of ideal body weight for height instead of BMI because the criteria for severity classification based on BMI are not available. Most of the presented subjects were severely obese because Ramathibodi Hospital is a tertiary care and referral center.

The universal standard diagnostic criteria for pediatric MS have not been established. Therefore, the authors used the IDF criteria, which is practical for clinicians, to define the MS in children and adolescents. The prevalence of MS among obese children and adolescents based on IDF criteria in the present study is close to previous studies which were based on IDF/ modified IDF criteria[33,34]. However, the prevalence of MS in some studies are different from the present study, which may be due to differences in terms of ethnic groups, cut-off values, degree of obesity and age groups.

The prevalence of MS increased with age. These results have not been reported in recent studies. The authors speculate that the earlier appearance of metabolic risk clusters and the longer time of exposure lead to the greater chance of developing coronary

**Table 2.** Severity of obesity

| Severity of obesity | Group 1 | | Group 2 | |
|---|---|---|---|---|
| | Mild | Moderate | Severe | Morbid |
| Number | 3 | 38 | 39 | 9 |
| % | 3.4 | 42.7 | 43.8 | 10.1 |

**Table 3.** Metabolic and cardiovascular risk factors by severity of obesity

| Risk factors | Total (n = 89) | Group 1 (n = 41) | Group 2 (n = 48) | OR (95% CI) | p-value |
|---|---|---|---|---|---|
| Age ≥ 10 years | 63 (70.8%) | 27 (65.9%) | 36 (75%) | 1.56 (0.62-3.89) | 0.34 |
| Hypertension | 18 (20.2%) | 6 (14.6%) | 12 (25%) | 1.94 (0.66-5.75) | 0.26 |
| Presence of acanthosis nigricans | 74 (83.1%) | 31 (75.6%) | 43 (89.6%) | 2.77 (0.86-8.93) | 0.08 |
| Total cholesterol ≥ 200 mg/dL | 27 (30.3%) | 9 (22%) | 18 (37.5%) | 2.13 (0.83-5.48) | 0.11 |
| LDL-C ≥ 130 mg/dL | 34 (38.2%) | 14 (34.1%) | 20 (41.7%) | 1.38 (0.58-3.27) | 0.46 |
| Triglyceride ≥ 150 mg/dL | 12 (13.5%) | 3 (7.3%) | 9 (18.8%) | 2.97 (0.74-11.63) | 0.11 |
| HDL-C < 40 mg/dL | 25 (28.1%) | 10 (24.4%) | 15 (31.3%) | 1.41 (0.55-3.60) | 0.47 |
| Impaired fasting glucose | 3 (3.4%) | 0 (0%) | 3 (6.3%) | 0.52 (0.43-0.64) | 0.24 |
| Impaired glucose tolerance | 9 (10.5%) | 2 (5.0%) | 7 (15.2%) | 3.41 (0.67-17.47) | 0.17 |
| Insulin > 25 microIU/mL | 19 (27.3%) | 1 (2.4%) | 18 (36.5%) | 24 (3.03-89.92) | < 0.001 |
| HOMA-IR ≥ 3.16 | 52 (58.4%) | 18 (43.9%) | 34 (70.8%) | 3.10 (1.29-7.45) | 0.01 |
| CRP > 3 mg/L | 38 (42.7%) | 10 (24.4) | 28 (58.3%) | 4.34 (1.74-10.84) | 0.001 |
| Metabolic syndrome | 15 (16.9%) | 4 (9.8%) | 11 (22.9%) | 2.75 (0.80-9.43) | 0.09 |

**Table 4.** Metabolic syndrome by age

| Age (years) | < 6 | 6-10 | 10-16 | > 16 | Total |
|---|---|---|---|---|---|
| MS + | 0 (0%) | 1 (4.3%) | 12 (20.0%) | 2 (66.7%) | 15 (16.9%) |
| MS - | 3 (100%) | 22 (95.7%) | 48 (80.0%) | 1 (33.3%) | 74 (83.1%) |

MS + = Presence of metabolic syndrome, MS - = Absence of metabolic syndrome, p-value = 0.027

**Table 5.** Factors related to metabolic syndrome: univariate analysis

| Factors | OR (95% CI) | p-value |
|---|---|---|
| Sex | 0.78 (0.25-2.42) | 0.673 |
| Age ≥ 10 years | 7.14 (0.88-57.47) | 0.058 |
| Family history of DM | 1.16 (0.33-4.05) | 1.00 |
| Acanthosis nigrican | 0.77 (0.19-3.17) | 1.00 |
| TC ≥ 200 mg/dL | 0.80 (0.23-2.80) | 0.77 |
| LDL-C ≥ 130 mg/dL | 0.53 (0.16-1.83) | 0.31 |
| Insulin > 25 microIU/mL | 9.60 (2.8-32.82) | < 0.001 |
| HOMA-IR ≥ 3.16 | 5.83 (1.22-27.68) | 0.01 |
| CRP > 3 mg/L | 3.29 (1.02-10.61) | 0.04 |

**Table 6.** Factors related to metabolic syndrome: logistic regression

| Factors | OR (95% CI) | p-value |
|---|---|---|
| Insulin > 25 microIU/mL | 7.24 (2.01-26.10) | 0.003 |
| Age ≥ 10 years | 5.87 (0.64-53.73) | 0.117 |
| CRP > 3 mg/L | 3.16 (0.85-11.71) | 0.085 |

disease and MS. Since the present study is cross-sectional, the authors suggest a cohort study to support this hypothesis.

Previous studies found the relation between severity of obesity and MS prevalence[2,16,17]. The prevalence of MS in the present study did not significantly increase with the degree of obesity. However, the present study has limitation since most of the present subjects were severely obese and all of them had central obesity.

The authors found the association between MS and insulin resistance, hyperinsulinemia and pro-inflammatory marker (CRP). Relation between MS and insulin resistance as well as hyperinsulinemia may be explained by pathophysiology of MS that hyperinsulinemia is the key factors of this syndrome.

From the hypothesis[35,36], obesity is characterized by a state of low grade inflammation at all ages. Chronic subclinical inflammation in children is associated with metabolic dysfunction, which can lead to insulin resistance and MS. In correspondence with the present results, children with MS had a higher CRP level than those without. The authors propose that CRP may be a good indicator of metabolic derangement and cardiovascular risk.

Limitations of the present study are small sample size, no age specific reference of BMI for Thai children, and no control group. In addition, the authors did not have information on various factors such as pubertal staging and duration of obesity. Further study, particularly a longitudinal one in larger populations, is needed to identify long term outcome of MS in children. The present study demonstrates a significant relationship between obesity, insulin resistance, a pro-inflammatory marker and MS. For clinical application, the authors suggest that insulin level and CRP should be assessed in severely obese children to identify high risk groups of MS and maintain close follow-up.

## Conclusion

The prevalence of MS is high among obese children and adolescents. The level of fasting insulin level above 25 microIU/mL was independently associated with MS. Prevention and proper management of MS should be considered in obese children.


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## Potential conflicts of interest

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## *กลุ่มอาการเมตาบอลิกในเด็กและวัยรุ่นที่มีภาวะอ้วน*

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***ภูมิหลัง****: ความชุกของโรคอ้วนในเด็กและวัยรุ่นเพิ่มขึ้นอย่างรวดเร็ว และเด็กอ้วนมีความเสี่ยงต่อการเกิดกลุ่มอาการเมตาบอลิกมากขึ้น*

***วัตถุประสงค์****: เพื่อหาความชุกของกลุ่มอาการเมตาบอลิกในเด็กอ้วนที่ระดับความรุนแรงต่าง ๆ กัน และศึกษาความสัมพันธ์ระหว่างกลุ่มอาการเมตาบอลิกกับปัจจัยดังนี้ คือ ระดับความรุนแรงของโรคอ้วน ภาวะดื้อต่ออินซูลิน และระดับ C-reactive protein (CRP)*

***วัสดุและวิธีการ****: เป็นการศึกษาแบบ cross-sectional study ในเด็กอ้วนที่มารับการรักษาที่โรงพยาบาลรามาธิบดี จำนวนทั้งหมด 89 คน ทำการเก็บข้อมูลประวัติครอบครัวเกี่ยวกับโรคเบาหวาน ความดันโลหิตสูง โรคอ้วน และไขมันผิดปกติ จากนั้นชั่งน้ำหนัก วัดส่วนสูง และวัดรอบเอว รวมทั้งมีการเจาะเลือดตรวจ fasting blood glucose, oral glucose tolerance test, insulin, CRP, และระดับไขมันในเลือด (lipid profile) หาความชุกของกลุ่มอาการเมตาบอลิกโดยใช้เกณฑ์ของ International Diabetes Federation (IDF) ปรับตามอายุและเพศ วิเคราะห์ข้อมูลโดยใช้ univariate และ logistic regression*

***ผลการศึกษา****: ในการศึกษานี้ ความชุกของกลุ่มอาการเมตาบอลิกเท่ากับ ร้อยละ 16.9 โดยพบกลุ่มอาการนี้ได้ในเด็กอ้วนปานกลาง, อ้วนมาก และอ้วนรุนแรง คิดเป็นร้อยละ 10.5, 23.1 และ 22.2 ตามลำดับ เมื่อวิเคราะห์โดยใช้ univariate พบว่า ปัจจัยที่มีความสัมพันธ์กับกลุ่มอาการเมตาบอลิกอย่างมีนัยสำคัญทางสถิติ ได้แก่ ค่าอินซูลินที่มากกว่า 25 microIU/mL, ค่า HOMA-IR ตั้งแต่ 3.16 ขึ้นไป และค่า CRP ที่มากกว่า 3 mg/L และเมื่อวิเคราะห์ด้วย logistic regression พบว่าค่าอินซูลินที่มากกกว่า 25 microIU/mL เป็นปัจจัยเดียวที่มีความสัมพันธ์กับกลุ่มอาการเมตาบอลิกอย่างมีนัยสำคัญ odds ratio 7.24 (95% CI 2.01-26.10)*

***สรุป****: พบความชุกของกลุ่มอาการเมตาบอลิกในเด็กและวัยรุ่นที่มีภาวะอ้วนได้สูง เด็กอ้วนจึงควรได้รับการรักษาอย่างเหมาะสมเพื่อป้องกันกลุ่มอาการนี้*