

# Correlation Between Serum Insulin and Features of Metabolic Syndrome in Thais

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## Abstract

Several clinical and metabolic abnormalities, i.e. central obesity, hypertension, impaired glucose tolerance or diabetes and dyslipidemia often cluster together and are commonly found in patients with atherosclerotic cardiovascular disease. Hyperinsulinemia and insulin resistance are often evident in subjects with these metabolic abnormalities, so called insulin resistance or metabolic syndrome. In the present study, we looked into the correlations between serum insulin or index of insulin sensitivity and various clinical and metabolic abnormalities. Subjects consisted of 103 males and 118 females. Oral glucose tolerance test was performed on all subjects. Homeostasis model assessment of insulin sensitivity (HOMA-S) was used to determine insulin sensitivity. In males, HOMA-S was found to be significantly correlated with BMI, plasma glucose, insulin, triglycerides and waist circumference. Male subjects in the highest quartile of HOMA-S also had significantly higher systolic blood pressure compared to those in the lowest quartile. In females, HOMA-S was significantly correlated with BMI, blood pressure, plasma glucose, insulin, triglycerides, HDL-cholesterol, waist circumferences and waist-hip ratio. However, after adjustment for BMI, correlation between HOMA-S and blood pressure in women was no longer statistically significant. We, therefore, concluded that correlations between serum insulin or index of insulin sensitivity with certain metabolic abnormalities also existed in Thai subjects. Some of these correlations seem to be at least in part dependent on obesity.

**Key word :** Insulin Resistance, Metabolic Syndrome, Atherosclerosis, HOMA-S

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A cluster of clinical and metabolic abnormalities, i.e. central obesity, hypertension, impaired glucose tolerance and dyslipidemia (increased VLDL-triglycerides and decreased HDL-cholesterol) are commonly found in patients with atherosclerotic cardiovascular disease<sup>(1)</sup>. Insulin resistance, i.e. resistance to insulin action, which usually refers to defects in insulin-mediated glucose disposal, has been shown in many studies to be linked to these metabolic and clinical features<sup>(2,3)</sup>. Reaven<sup>(4)</sup> has coined the term "syndrome X" to describe these clinical and metabolic features and has hypothesized that insulin resistance is the basis of this syndrome. However, there seem to be some inconsistencies of these correlations among different populations. The correlation between insulin and coronary heart disease seems to be more controversial and both cross-sectional and prospective studies have yielded contradictory results<sup>(2)</sup>. This study, therefore, looked into the associations between serum insulin and other features of clinical and metabolic abnormalities, or the so called metabolic syndrome in the Thai population.

## SUBJECTS AND METHOD

### Subjects

Subjects included adult males and females, aged 40 years old or more, who had no underlying diseases and were not receiving any medications which might affect insulin sensitivity or lipid profile. The body weight and height were measured while the subjects were fasting and in light clothes. The blood pressure was measured with a standard mercury sphygmomanometer in the sitting position after the subjects had been at rest for at least 15 minutes and the mean values of two measurements taken at 10-min intervals were used. The waist circumference was measured at the umbilical level. The hip circumference was measured at the widest part over the buttocks or trochanter region.

### Oral glucose tolerance test

Oral glucose tolerance test was performed on all subjects with 75 grams of glucose. The test was done in the morning between 8-10 a.m. after an overnight fast. Samples were collected every 30 minutes for measurements of glucose and insulin. Baseline samples were also collected for measurements of serum cholesterol, triglycerides and HDL-cholesterol.

### Laboratory assessment

Plasma glucose was measured by the glucose oxidase method using the Beckman Glucometer 2. Serum insulin was measured by radioimmunoassay (CIS Biotechnology). Assessment of insulin sensitivity was calculated from the homeostasis model assessment (HOMA-S) using fasting glucose and insulin values as described previously<sup>(5)</sup>. Serum total cholesterol (TC), HDL-cholesterol (HDL-C) and triglycerides (TG) concentrations were determined by colorimetry.

### Statistical analysis

Data were expressed as mean  $\pm$  SD. Correlations among variables were analysed using Pearson and Spearman Rho's correlation. Comparisons among variables in subjects with different glucose tolerance status were done using one-way analysis of variance.

## RESULTS

There were 221 subjects included in this study, 103 males and 118 females. The mean age  $\pm$  SD was  $52.6 \pm 9.6$  years. The body mass index (BMI) was  $24.3 \pm 3.2$  kg/m<sup>2</sup>. The systolic blood pressure (SBP) was  $128.4 \pm 18.7$  mmHg, diastolic blood pressure (DBP)  $81.2 \pm 9.5$  mmHg, waist circumference (WC)  $82.5 \pm 8.5$  cm, waist-hip ratio (WHR)  $0.85 \pm 0.07$ . The mean serum cholesterol level was  $219.4 \pm 16.2$  mg/dl, triglycerides  $138.2 \pm 95.7$  mg/dl, HDL-C  $49.2 \pm 12.5$  mg/dl. The mean calculated LDL-C was  $143.5 \pm 32.7$  mg/dl. The mean plasma glucose and insulin levels from oral glucose tolerance test are shown in Table 1. Fasting insulin levels and HOMA-S have been shown to be significantly correlated with postload insulin at all points measured, including area under the curve of insulin ( $P < 0.01$ ). Fasting insulin and HOMA-S were also significantly correlated with fasting and 1 hour-postload glucose ( $P < 0.01$ ).

Both fasting insulin and HOMA-S were significantly correlated with various parameters associated with features of insulin resistance syndrome, namely body mass index, blood pressure, plasma glucose, triglycerides, HDL-cholesterol and index of central obesity, i.e. waist circumference and waist hip ratio, with HOMA-S showing stronger correlations than fasting insulin or insulin measured at other time-points. We, therefore, chose HOMA-S to represent the index of insulin sensitivity in our study. Table 2 and 3 show correlations between

**Table 1. Plasma glucose and serum insulin levels at baseline and after 75 grams oral glucose tolerance test.**

Variables	Time (minutes)				
	0	30	60	90	120
Glucose (mg/dl)	89.9 ± 14.7	160.1 ± 32.6	181.8 ± 48.0	173.9 ± 51.3	153.7 ± 52.2
Insulin (μU/ml)	21.1 ± 9.7	102.4 ± 64.3	126.9 ± 88.6	146.4 ± 107.8	142.3 ± 112.2

**Table 2. Correlations between quartile of HOMA-S and various clinical and metabolic parameters in males.**

Variables	Quartile of HOMA-S				P value	
	1st	2nd	3rd	4th	Not adjusted	Adjusted for BMI
Age (yr)	54.2 ± 9.9	56.6 ± 10.8	52.2 ± 9.3	50.9 ± 10.1	NS	NS
BMI (kg/m <sup>2</sup> )	22.9 ± 1.7	23.3 ± 2.6	24.2 ± 3.1	26.1 ± 2.4	< 0.01	-
SBP (mmHg)	123.9 ± 13.4	133.8 ± 21.1	130.8 ± 16.8	133.7 ± 18.4	NS	NS
DBP (mmHg)	80.4 ± 7.1	84.9 ± 10.5	80.0 ± 9.0	84.9 ± 9.8	NS	NS
FPG (mg/dl)	86.6 ± 9.6	87.4 ± 6.9	93.2 ± 9.0	104.1 ± 30.3	< 0.01	< 0.01
1 h-PG (mg/dl)	172.8 ± 41.3	178.3 ± 38.4	187.4 ± 41.3	219.7 ± 68.5	< 0.01	< 0.01
2 h-PG (mg/dl)	133.4 ± 37.2	143.2 ± 39.4	140.2 ± 38.2	183.7 ± 81.5	0.03	0.02
Fasting insulin (μU/ml)	11.4 ± 3.0	18.5 ± 2.3	23.8 ± 3.3	36.7 ± 10.1	< 0.01	< 0.01
1 h-insulin (μU/ml)	99.1 ± 43.8	120.8 ± 68.8	154.3 ± 76.1	218.9 ± 167.7	< 0.01	< 0.01
2 h-insulin (μU/ml)	112.4 ± 66.9	113.6 ± 69.2	144.9 ± 105.3	215.6 ± 208.9	< 0.01	0.09
TC (mg/dl)	220.3 ± 29.2	225.5 ± 45.2	216.0 ± 39.1	202.5 ± 33.9	NS	NS
TG (mg/dl)	143.7 ± 73.7	133.3 ± 57.0	191.6 ± 201.9	183.9 ± 97.3	0.04	NS
HDL-C (mg/dl)	42.3 ± 9.3	46.1 ± 11.7	43.7 ± 12.2	40.9 ± 8.0	NS	0.04
WC (cm)	83.1 ± 6.3	83.9 ± 6.8	87.2 ± 7.2	90.4 ± 7.1	< 0.01	NS
WHR	0.89 ± 0.05	0.88 ± 0.05	0.91 ± 0.05	0.90 ± 0.04	NS	NS

HOMA-S, divided into quartile, with various parameters of metabolic syndrome in males and females. In males, HOMA-S has been shown to be significantly correlated with BMI, plasma glucose, insulin, triglycerides and waist circumference. Male subjects in the highest quartile of HOMA-S also had higher systolic blood pressure compared to those in the lowest quartile ( $P=0.01$ ). In females, HOMA-S was significantly correlated with BMI, blood pressure (both systolic and diastolic), plasma glucose, triglycerides, HDL-C, waist circumference and waist-hip ratio. However, after adjustment for BMI, correlation between HOMA-S and blood pressure in women no longer existed.

Table 4 demonstrates the clinical and metabolic parameters of subjects with normal glucose tolerance (NGT), impaired glucose tolerance (IGT) or diabetes mellitus after 75-gram glucose load. According to the World Health Organization Provi-

sion Criteria<sup>(6)</sup>, there were 82 subjects (37.1%) who had IGT and 36 (16.3%) who fulfilled the criteria for diabetes. Subjects who had diabetes were significantly older and had significantly higher BMI, systolic blood pressure, waist circumference and waist-hip ratio than those with either NGT or IGT ( $P < 0.05$ ). Moreover, subjects with diabetes had higher fasting and 120-minute postload insulin than the other two groups ( $P < 0.05$ ). Although the subjects with diabetes had higher triglycerides and lower HDL-cholesterol levels compared to those with IGT or normal glucose tolerance, these did not reach statistical significance.

## DISCUSSION

The results of the present study demonstrated the association between hyperinsulinemia and several clinical and metabolic parameters associated with atherosclerotic cardiovascular disease.

**Table 3** Correlations between quartile of HOMA-S and various clinical and metabolic parameters in females

Variables	Quartile of HOMA-S				P value	
	1st	2nd	3rd	4th	Not adjusted	Adjusted for BMI
Age (yr)	53.2 ± 11.3	50.1 ± 7.1	50.4 ± 7.8	54.1 ± 9.9	NS	NS
BMI (kg/m <sup>2</sup> )	22.8 ± 3.0	24.1 ± 3.2	25.1 ± 3.3	25.8 ± 4.3	< 0.01	-
SBP (mmHg)	118.6 ± 18.7	129.7 ± 19.0	126.3 ± 19.7	130.9 ± 18.4	0.03	NS
DBP (mmHg)	74.3 ± 9.3	83.6 ± 8.2	80.7 ± 10.6	81.1 ± 7.6	0.03	NS
FPG (mg/dl)	82.8 ± 6.4	86.4 ± 10.0	86.1 ± 6.8	94.0 ± 13.3	< 0.01	< 0.01
1 h-PG (mg/dl)	161.3 ± 35.1	169.0 ± 44.0	173.2 ± 46.0	196.2 ± 42.7	< 0.01	< 0.01
2 h-PG (mg/dl)	142.4 ± 36.2	152.9 ± 38.5	150.5 ± 46.5	181.0 ± 64.6	0.06	0.05
Fasting insulin (μU/ml)	10.1 ± 3.4	16.4 ± 1.9	21.7 ± 2.0	30.7 ± 5.8	< 0.01	< 0.01
1 h-insulin (μU/ml)	79.7 ± 34.0	86.5 ± 37.4	141.9 ± 80.0	122.8 ± 52.5	< 0.01	< 0.01
2 h-insulin (μU/ml)	89.2 ± 35.0	125.1 ± 66.7	170.7 ± 116.6	167.4 ± 103.9	< 0.01	< 0.01
TC (mg/dl)	223.6 ± 30.9	219.7 ± 27.9	221.6 ± 39.4	224.8 ± 40.0	NS	NS
TG (mg/dl)	102.2 ± 45.9	107.4 ± 39.0	125.4 ± 64.5	129.7 ± 66.3	0.04	NS
HDL-C (mg/dl)	58.0 ± 10.3	57.2 ± 13.2	51.4 ± 12.1	50.9 ± 9.9	0.01	0.03
WC (cm)	75.2 ± 6.5	79.1 ± 7.5	78.1 ± 6.5	84.3 ± 8.8	< 0.01	0.02
WHR	0.81 ± 0.06	0.81 ± 0.06	0.81 ± 0.05	0.85 ± 0.06	0.02	0.08

**Table 4.** Clinical and metabolic parameters in subjects with normal glucose tolerance (NGT), impaired glucose tolerance (IGT) or diabetes after 75-gram oral glucose tolerance test.

Parameters	NGT	IGT	DM
Number of subjects (%)	103 (46.6)	82 (37.1)	36 (16.3)
Age (yr) a,b,c	49.9 ± 8.4	53.4 ± 9.3	58.8 ± 10.9
% female	48.5	54.9	63.9
BMI (kg/m <sup>2</sup> ) b,c	23.8 ± 2.8	24.0 ± 3.2	26.6 ± 3.8
Systolic BP (mmHg) b,c	126.0 ± 18.3	128.2 ± 18.9	136.2 ± 18.1
Diastolic BP (mmHg)	80.6 ± 9.4	81.1 ± 9.7	83.0 ± 9.3
Fasting insulin (μU/ml) b,c	20.5 ± 9.3	19.6 ± 8.2	26.3 ± 12.1
1 h-insulin (μU/ml)	136.0 ± 75.9	110.4 ± 62.5	138.3 ± 150.0
2 h-insulin (μU/ml) a,b,c	106.1 ± 65.3	157.1 ± 94.7	212.6 ± 191.1
HOMA-S b,c	4.4 ± 2.1	4.3 ± 2.0	6.9 ± 3.7
Cholesterol (mg/dl) c	213.3 ± 35.9	223.7 ± 34.2	227.2 ± 39.4
Triglycerides (mg/dl)	124.9 ± 111.6	146.0 ± 79.9	158.2 ± 73.8
HDL-C (mg/dl)	50.1 ± 12.5	49.2 ± 13.2	46.4 ± 10.5
WC (cm) b,c	81.2 ± 7.7	81.8 ± 9.0	87.6 ± 7.3
WHR b,c	0.84 ± 0.07	0.85 ± 0.07	0.89 ± 0.05

a ; P &lt; 0.05 between NGT and IGT

b ; P &lt; 0.05 between IGT and DM

c ; P &lt; 0.05 between NGT and DM

These results were in accordance with several other studies done in various ethnic groups<sup>(2)</sup>. However, the strength of associations between various components of metabolic syndrome was somewhat different. In our study, we use homeostasis model assessment of insulin sensitivity (HOMA-S) as an index of insulin sensitivity. This model has been shown to

be useful to assess insulin sensitivity, especially in epidemiological studies<sup>(5)</sup>. We have demonstrated the correlations between HOMA-S and various components of metabolic syndrome, i.e. body mass index, blood pressure, dyslipidemia (increased triglycerides and decreased HDL-cholesterol levels), plasma glucose (both fasting and post glucose load)

and index of central obesity (waist circumference and waist hip ratio), many of which showed stronger correlations in females than in males. When the data were compared between subjects in the lowest and highest quartile of HOMA-S, the difference between these parameters became more apparent. The correlation between HOMA-S and body mass index was highly significant in both sexes and addressed the close relationship between insulin and obesity. Our study also demonstrated the correlation between serum insulin and blood pressure, especially in women. However, the correlation was not statistically significant when BMI was taken into account.

The relationship between insulin resistance, obesity and dyslipidemia seems to be at least in part dependent upon the degree of visceral adiposity<sup>(7-9)</sup>. Decreased insulin action on adipocytes increases lipolysis, thereby releasing free fatty acids. Free fatty acids released from visceral fat can enter directly into the portal circulation and serve as a substrate for both hepatic triglyceride synthesis and gluconeogenesis<sup>(10,11)</sup>. Free fatty acids in turn can affect insulin sensitivity both at the hepatic level and at muscular level *vis* Randle cycle<sup>(11)</sup>. Recent evidence has also suggested that leptin, a hormone produced by adipocytes, was also significantly associated with insulin resistance<sup>(12)</sup>. In clinical practice, waist circumference and waist-hip ratio may be used as an index of visceral or central obesity and these two parameters have been found in most studies, including ours, to be closely correlated with insulin resistance.

The relationship between insulin resistance and hypertension is somewhat more controversial. Insulin levels have been shown to be significantly correlated with blood pressure in many ethnic groups, including Caucasians and Japanese, but the

correlation was weak in African Americans or Pima Indians<sup>(13-19)</sup>. In some studies, the correlation was independent of obesity, while in others obesity seemed to play an important role. In our study, the correlation between insulin and blood pressure seemed to be at least in part dependent on obesity.

Our study also demonstrated that subjects with impaired glucose tolerance or who had diabetes mellitus were more insulin resistant than those with normal glucose tolerance. Both fasting and post-glucose load insulin levels were highest in those who had diabetes mellitus. Subjects with diabetes mellitus also had other features of insulin resistance, i.e. higher BMI, blood pressure, waist circumference and waist hip ratio. They also tended to have higher triglyceride and lower HDL-cholesterol levels, but this did not reach statistical significance. These data suggested that insulin resistance becomes increasingly more severe in subjects with normal glucose tolerance through impaired glucose tolerance and diabetes.

Perhaps one of the mostly debated issues about insulin resistance is whether insulin resistance or hyperinsulinemia is an independent risk factor for atherosclerotic cardiovascular disease. Many prospective studies have been done to look into this question but have come to different conclusions<sup>(20-30)</sup>. Some studies have found that insulin was an independent risk factor while others have found that the association of insulin and coronary heart disease was no longer present when other factors, namely lipids and body mass index were taken into consideration. Differences in ethnic groups and methods to assess insulin sensitivity may also contribute to these inconsistent results<sup>(31, 32)</sup>. Whether insulin resistance is an independent risk factor of coronary heart disease in the Thai population needs to be further investigated.

## REFERENCES

1. DeFronzo RA, Ferrannini E. Insulin resistance: a multifaceted syndrome responsible for NIDDM, obesity, hypertension, dyslipidemia, and atherosclerotic cardiovascular disease. *Diabetes Care* 1991;14:173-94.
2. Stern MP. The insulin resistance syndrome. In: Alberti KGMM, Zimmet P, DeFronzo RA, Keen H, eds. *International Textbook of Diabetes Mellitus*. London: John Wiley & Sons, 1997: 255-83.
3. Modan J, Halkin H, Almog S, et al. Hyperinsulinemia: a link between hypertension, obesity and glucose intolerance. *Hypertension* 1985;75:809-17.
4. Reaven GM. Role of insulin resistance in human disease. *Diabetes* 1988;37:1595-607.
5. Haffner SM, Miettinen H, Stern MP. The homeostasis model in the San Antonio Heart Study. *Diabetes Care* 1997;20:1087-92.
6. Alberti KG, Zimmet PZ. Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: diagnosis and classification of diabetes mellitus provisional report of a WHO consultation. *Diabet Med* 1998;15:539-53.
7. Fujioka S, Matsuzawa Y, Tokinaga K, et al. Contribution of intra-abdominal fat accumulation to the impairment of glucose and lipid metabolism in human obesity. *Metabolism* 1987;36:54-9.
8. Abate N. Insulin resistance and obesity: the role of fat distribution pattern. *Diabetes Care* 1996;19:292-4.
9. Garg A. Insulin resistance in the pathogenesis of dyslipidemia. *Diabetes Care* 1996;4:387-9.
10. Ferrannini E, Barrett EJ, Bevilacqua S, et al. Effects of fatty acids on glucose production and utilization in man. *J Clin Invest* 1983;72:1737-47.
11. Groop LC, Saloranta C, Shank M, et al. The role of free fatty acid metabolism in the pathogenesis of insulin resistance in obesity and non-insulin dependent diabetes mellitus. *J Clin Endocrinol Metab* 1991;72:96-107.
12. Donahue RP, Prineas RJ, Donahue RD, et al. Is fasting leptin associated with insulin resistance among nondiabetic individuals? *Diabetes Care* 1999;22:1092-6.
13. Ferrannini E, Buzzigoli G, Bonadonna R, et al. Insulin resistance in essential hypertension. *N Eng J Med* 1987;317:350-7.
14. Pollare T, Lithell H, Berne C. Insulin resistance is a characteristic feature of primary hypertension independent of obesity. *Metabolism* 1990;39:167-74.
15. Collins VR, Dowse GK, Finch CF, et al. An inconsistent relationship between insulin and blood pressure in three Pacific Island populations. *J Clin Epidemiol* 1990;43:1369-78.
16. Saad MF, Lillioja S, Nyomba BL, et al. Racial differences in the relation between blood pressure and insulin resistance. *N Eng J Med* 1991;324:733-9.
17. Miura K, Nakagawa H, Nishijo M, et al. Plasma insulin and blood pressure in normotensive Japanese men with normal glucose tolerance. *J Hypertens* 1995;13:427-32.
18. Asch S, Wingrad DL, Barrett-Conner EL. Are insulin and hypertension independently related? *Ann Epidemiol* 1991;1:231-44.
19. Denker PS. Fasting serum insulin levels in essential hypertension. A meta-analysis. *Arch Intern Med* 1992;152:1649-51.
20. Pyorala K. Relationship of glucose tolerance and plasma insulin to the incidence of coronary heart disease: results from two population studies in Finland. *Diabetes Care* 1979;2:131-41.
21. Pyorala K, Savolainen E, Kaukola S, et al. Plasma insulin as coronary disease risk factor: relationship to other risk factors and predictive value during 9 1/2 year follow-up of the Helsinki Policeman study population. *Acta Med scand* 1985;701 (suppl):38-52.
22. Welborn TA, Wearne K. Coronary heart disease incidence and cardiovascular mortality in Busseton in reference to glucose and insulin concentrations. *Diabetes* 1979;2:154-60.
23. Ducimetiere P, Eschwege E, papoz L, et al. Relationship of plasma insulin levels to the incidence of myocardial infarction and coronary heart disease mortality in a middle-aged population. *Diabetologia* 1980;19:205-10.
24. Fontbonne A, Charles MA, Thibault N, et al. Hyperinsulinemia as a predictor of coronary heart disease mortality in a healthy population: the Paris Prospective Study, a 15-year follow up. *Diabetologia* 1991;34:356-61.
25. Despres J-P, Lamarche B, Mauriege P, et al. Hyperinsulinemia as an independent risk factor for ischemic heart disease. *N Eng J Med* 1996;334:952-7.
26. Hargreaves AD, Logan RL, Elton RA, et al. Glucose tolerance, plasma insulin, HDL-cholesterol and obesity; 12-year follow-up and development of coronary heart disease in Edinburgh men. *Atherosclerosis* 1992;94:61-9.
27. Welin L, Eriksson H, Larsson B, et al. Hyperinsulinemia is not a major coronary risk factor in elderly men: the study of men born in 1913. *Diabetologia* 1992;35:766-72.
28. Ferrara A, Barrett-Conner E, Edelstein SL. Hyperinsulinemia does not increase the risk of fatal cardiovascular disease in elderly men or women without diabetes: the Rancho Bernardo Study,

- 1984-to 1991. Am J Epidemiol 1994;140:857-69.
29. Bonora E, Willett J, Kiechl S, et al. U-shaped and J-shaped relationships between serum insulin and coronary heart disease in the general population. The Bruneck Study. Diabetes Care 1998;21:221-30.
  30. Folsom AR, Rasmussen MI, Chambless LE, et al. Prospective associations of fasting insulin, body fat composition, and diabetes with risk of ischemic stroke. The Atherosclerosis Risk in Communities (ARIC) Study Investigators. Diabetes Care 1999;22:1077-83.
  31. Wingard DL, Barrett-Conner EL, Ferrara A. Is insulin really a heart disease risk factor? Diabetes Care 1995;18:1299-304.
  32. Stern MP. The insulin resistance syndrome: the controversy is dead, long live the controversy! Diabetologia 1994;37:956-8.

## การศึกษาความสัมพันธ์ระหว่างซีรัมอินซูลินและการเปลี่ยนแปลงทางเมตาบอลิกในคนไทย

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การเปลี่ยนแปลงทางคลินิกและเมตาบอลิกหลาย ๆ อย่าง เช่น ความดันโลหิตสูง, โรคอ้วน, ความทนกลูโคสผิดปกติ หรือโรคเบาหวาน และระดับไขมันที่ผิดปกติ มักพบร่วมกัน และพบได้บ่อยขึ้นในผู้ที่เป็นโรคหัวใจและหลอดเลือดภาวะดื้อต่ออินซูลิน หรือการมีระดับอินซูลินในเลือดสูงมักพบในคนเหล่านี้ ทำให้มีผู้เรียกกลุ่มอาการเหล่านี้ว่า กลุ่มอาการที่เกี่ยวข้องกับภาวะดื้อต่ออินซูลินหรือกลุ่มอาการทางเมตาบอลิก คณะผู้วิจัยได้ทำการศึกษาความสัมพันธ์ระหว่างระดับอินซูลินหรือดัชนีที่แสดงความต้องการอินซูลินต่อการเปลี่ยนแปลงทางคลินิกหรือเมตาบอลิกในคนไทยทั้งสิ้น 221 ราย (ชาย 103 ราย, หญิง 118 ราย) โดยอาสาสมัครทุกรายได้รับการทดสอบความทนกลูโคสโดยการรับประทานกลูโคส 75 กรัม ในเพศชายพบว่าดัชนีที่แสดงความต้องการอินซูลินมีความสัมพันธ์กับดัชนีความหนาของร่างกาย, ระดับน้ำตาล, ระดับอินซูลินที่เวลาต่าง ๆ, ระดับไขมันไตรกลีเซอไรด์ และเส้นรอบเอว ในเพศหญิง ยังพบความสัมพันธ์นี้กับระดับความดันโลหิตด้วย อย่างไรก็ตาม เมื่อนำค่าดัชนีความหนาของร่างกายมาพิจารณาด้วย พบว่าความสัมพันธ์ระหว่างอินซูลิน และระดับความดันโลหิตไม่นัยสำคัญทางสถิติอีกต่อไป ดังนั้น จึงสรุปได้ว่าระดับอินซูลินมีความสัมพันธ์กับการเปลี่ยนแปลงทางเมตาบอลิกหลายอย่างในคนไทย แต่ความสัมพันธ์บางอย่างอาจเกี่ยวข้องกับดัชนีความหนาของร่างกาย

**คำสำคัญ :** ภาวะดื้อต่ออินซูลิน, กลุ่มอาการทางเมตาบอลิก

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