Original Article

Insulin, lipid profiles and measures of fatness in Taiwanese women in relation to duration of residence in Australia

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This study investigated the relationships between measures of fatness and blood insulin and lipids in Taiwanese females living in Taiwan (n=97) or Australia (n=100), and examined the effect of length of residence in Australia on these relationships. Fasting glucose and lipids were determined by Reflotron and fasting insulin using Microparticle Enzyme Immunoassay; insulin resistance (IR) was identified by HOMA. There were no significant inter-country differences in crude plasma insulin or HOMA-IR between Taiwan and Australia (51.7±42.2 vs. 45.0±29.0 pmol/L and 1.43±1.21 vs. 1.29±1.00, respectively, all $p > 0.05$), but when insulin and HOMA-IR were adjusted for waist circumference, they were greater in Taiwan (45.7±1.6 vs. 38.0±1.6 pmol/L and 1. 26±1.59 vs. 1.13±1.59, respectively, all $p < 0.05$). Subjects living in Australia greater than 5 years had higher insulin and HOMA-IR values than those with less than 5 years residence (50.0±32.3 vs. 32.4±10.5 pmol/L and 1.45±1.00 vs. 0.90±0.28, respectively, all $p < 0.01$), even after adjustment for all measures of fatness. Subjects in Australia > 5 years have 6 (CI, 1.3-27.9) times the risk of having insulin > 50 pmol/L; the increased risk being confined to generally and/or centrally obese women. Measures of central obesity and general obesity were positively associated with HOMA-IR in both countries (r = 0.23, $p < 0.05$ and 0.27 $p < 0.01$, Taiwan, 0.43 and 0.43, both $p < 0.01$, Australia). Taiwanese females living in Australia initially appear to have a more favorable state of IR than those in Taiwan, but insulin resistance is associated with length of residence in Australia, particularly among the obese.

Key Words: insulin resistance, diet, obesity, blood lipids, Taiwanese, Taiwanese immigrants in Australia

Introduction

Insulin resistance (IR) is a common pathologic state in which target cells fail to respond to ordinary levels of circulating insulin, leading to hyperinsulinemia. Both IR and hyperinsulinemia are known to be associated with the metabolic syndrome which has a number of definitions, the most common of which involves a cluster of abnormalities comprising glucose intolerance, dyslipidaemia, high blood pressure and impaired fibrinolytic activity.[1] The metabolic syndrome is associated with increased risk of developing type 2 diabetes mellitus (T2DM) and cardiovascular disease (CVD).[1]

There is increasing evidence that obesity plays a key role in the development of the metabolic syndrome[2] and that body fat distribution is associated with IR and CVD risk.[3, 4] However the strength of this association varies by other factors such as age[5], and the relationship between regional fat and IR may be different among ethnic groups. Chinese populations have higher proportions of body fat when compared to body mass index (BMI)-matched Caucasians, and also different patterns of fat distribution.[6,7] Hence the relationship between obesity and IR may be different in Chinese compared to Caucasians.

Two of the most common lipid abnormalities observed in IR are increased triglyceride (TG) and reduced high-density lipoprotein cholesterol (HDL-C).[8] IR has been shown to have a negative correlation with HDL-C and its protein moiety, apolipoprotein A-1 (Apo A1)[9,10] and a positive correlation with TG, total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and apolipoprotein B (Apo B).[10] In addition, predominance of small, dense low density lipoprotein (LDL) particles is associated with an increased risk of IR, T2DM and CVD.[11,12,13,14]

The Chinese population is one of the fastest growing in Australia.[15] Studies assessing the relationships between obesity and adverse health outcome have been mostly based in Europe and the United States[16,17,2,4], and few have addressed the relationship between IR, obesity and CVD in the Chinese population. We have found that Taiwanese females living in Australia consume more calories, carbohydrate and saturated fat than Taiwanese living in Taiwan, and that length of residence in Australia is associated with increased adiposity for Australian Taiwanese females.[18]

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There is growing evidence, particularly among Asian and female populations, that central obesity may be a more consistent predictor of glucose intolerance than overall obesity.[4,19] Chinese women have a low rate of conventional obesity but central fat may be deposited at lower BMI than in other populations[6,7], thus providing an opportunity to examine the effects of central adiposity on risk of insulin resistance among nonobese women. The purpose of this cross-sectional study was to investigate the relationships between measures of fatness and blood insulin and lipid profiles of Taiwanese females living in Taiwan or Australia and to examine the effect of length of residence in Australia on those relationships.

Methods

This cohort has been described previously.[18] Briefly, 197 Taiwanese females (100 in the Sydney metropolitan area, Australia, and 97 living in Taiwan), aged 20 to 62 years participated. The study was approved by the Human Ethics Committee of the University of Sydney and subjects gave informed consent in their native language. Anthropometric measurements were taken according to standard protocols.[18]

Overnight fasting venous blood samples were packed in ice and centrifuged within 2 hours. Aliquots were frozen at -80°C until analysis. All the study samples from Taiwan and Australia were analyzed in the same laboratory using the same methodology. TC, HDL-C, TG and serum glucose concentrations were measured using a Reflotron reflectance photometric analyzer (Boehringer Mannheim, Germany). The accuracy of the Reflotron used in this study is regularly assessed through participation in the RCPA-AACB chemical pathology quality assurance programme. LDL-C concentration was estimated using a modification of the Friedewald formula.[20] Apo A1 and Apo B were measured by immunoturbidimetric analyses using a Turbitimer System (Behring Diagnostics, Australia).

LDL peak particle diameter was determined by vertical gradient gel electrophoresis which entails the migration of charged particles through a matrix comprised of increasing concentrations of polyacrylamide gel at 4 °C, using non-denaturing polyacrylamide gradient (3-13%) gels (Alamo gels Inc., Texas, US). For particle size calibration, 2 standards were used, thyroglobulin (17.0 nm) (Amersham Pharmacia Biotech, UK) and polystyrene latex beads (33.0 nm) (Duke Scientific Corporation, US).

Insulin was measured using an AxSYM system insulin assay (Abbot, Japan) which is based on Microparticle Enzyme Immunoassay (EMIA) technology, in a major tertiary referral hospital. IR was calculated by the homeostatic model assessment of insulin resistance (HOMA-IR). HOMA-IR uses fasting plasma glucose and insulin concentrations to derive indices of insulin resistance 〔(fasting insulin (mIU/L) * fasting glucose (mg/dL) * 0.05551) / 22.5〕.[21]

Data were analyzed using SPSS Windows release 12.0. Continuous variables are presented as mean ± standard deviation (SD); significance was set at $p < 0.05$. Differences in continuous variables between groups were estimated by Students t-test. Pearson's correlation was used to assess the correlation between continuous variables, and crude and adjusted odds ratios were calculated using logistic regression. The distributions of fasting insulin,

Table 1. Blood insulin and lipid profiles of Taiwanese subjects stratified by country of residence: mean±SD

	Taiwan (n=97)	Australia (n=100)
Insulin (pmol/L)		
Mean (range)	51.7±42.2 (16-346)	45.0±29.0 (14-189)
Median	45.0	37.0
Logarithmic mean†	1.64±0.24	1.60±0.21
(Back transformed)‡	(43.7±1.7)	(39.8±1.6)
Adjusted age †	42.7±1.6	39.8±1.6
WC	45.7±1.6	38.0±1.6*
HC	45.7±1.6	37.2±1.6*
HOMA-IR		
Mean (range)	1.43±1.21 (0.39-0.66)	1.29 ±1.00 (0.43-6.08)
Median	1.20	1.04
Logarithmic mean†	0.07±0.25	0.05±0.22
(Back transformed) ‡	(1.18±1.78)	(1.12±1.66)
Adjusted age †	1.18±1.74	1.12±1.59
WC	1.26±1.59	1.13±1.59*
HC	1.26±1.59	1.13±1.59*
TC (mmol/L)	4.7±0.7	4.7±0.7
LDL-C (mmol/L)	3.0±0.7	3.0±0.7
HDL-C (mmol/L)	1.4±0.4	1.4±0.3
TG (mmol/L) †	1.00±1.32	0.94±1.35
Apo A1 (mg/dL)	160±27	172±30 **
Apo B (mg/dL)	87.2±20.3	110±31 ***
LDL diameter (nm)	26.9±0.6	27.1±0.7 **
Glucose (mmol/L)	4.3±0.5	4.5±0.4 **

*, **, *** Significantly different between Taiwan and Australia at the 0.05, 0.01, 0.001 level respectively. † Statistics and adjustments carried out using log transformed values which were back transformed for comparison with other published values. ‡ Differs from arithmetic mean because of logarithmic transformation.

HOMA-IR and TG were skewed to the right indicating a non-normal distribution (one-sample Kolmogorov: all $p<0.0001$). Non-normally distributed data were log transformed before analysis. After log transformation, insulin and HOMA-IR were normally distributed but TG was still non-normally distributed. Non-parametric tests (Mann Whitney U) were used for the groups with unequal number of subjects or non-normally distributed variables. Adjustment for confounding variables such as age, lipid measures and anthropometric measures was calculated by a general linear model.

Results

Table 1 presents blood insulin and lipid profiles of subjects stratified by country of residence. There were no differences in crude values of fasting insulin or HOMA-IR between Taiwan and Australia, or after adjustment for age. As waist circumference (WC), and hip circumference (HC) were significantly different between the groups (all $p< 0.001$)[18], values were adjusted for these variables. Both insulin and HOMA-IR were significantly higher in Taiwan than in Australia after adjustment for WC and HC ($p < 0.05$). Levels of Apo A1, Apo B, glucose, and LDL particle diameter were lower in Taiwan than in Australia; after adjustment for age the differences remained (adjusted data not shown).

Table 2 shows the correlation coefficients between different measures of fat mass and insulin, HOMA-IR and blood lipids. In general, although positive correlations exist between measures of fatness and insulin and HOMA-IR, the strength of the correlations is much greater in women living in Australia. For women living in Taiwan, there was no correlation between WHR and measures of insulin resistance and no correlation between measures of fatness and TG or HDL. In Australia, measures of fatness were negatively correlated with HDL-C and positively correlated with TG. On the other hand, strong positive correlations between all measures of fatness and TC and LDL-C were apparent in Taiwan, while TC was correlated only with HC, and LDL-C with most measures of fatness (except WHR), but less strongly so, in Australia. LDL diameter was negatively correlated with BMI, WC and %TBF in Australia but no significant correlation was shown between LDL diameter and measures of fatness in Taiwan. Furthermore, HOMA- IR was positively correlated with TG and negatively correlated with HDL in Australia, but not at all in Taiwan (0.52 $p<$ 0.01 and -0.24 $p<0.05$ in Australia, 0.19 and -0.06 both $p>0.05$ in Taiwan, respectively).

In regard to dietary measures and levels of exercise (previously reported)[18], there was no correlation between insulin and HOMA-IR and energy adjusted-nutrient intakes in either Taiwan or Australia. A negative correlation was found between insulin and HOMA-IR with exercise ($r = -0.22$ and $r = -0.21$ both $p<0.05$, respectively) only in Australia.

A fasting insulin greater than 60 pmol/L has been identified as an independent risk factor for CVD and T2DM.[22] Since none of the subjects living in Australia less than 5 years had insulin levels above this recommended cut-off, an insulin level greater than 50pmol/L was chosen to examine association of residence with disease risk. According to odds ratio analysis, subjects living in Australia greater than 5 years had 6 times the odds (95% CI, 1.33-27.9) of having a fasting insulin greater than 50 pmol/L (unchanged after adjustment for age).

We have previously reported that length of residence in Australia for Taiwanese women was associated with increase in fatness and saturated fat consumption.[18] When the effect of length of residence on biochemical profiles was examined in regard to lipids, only TG was significantly associated with length of residence in Australia. After adjustment for age, the strength of the association was reduced (to $p <0.05$; Table 3). Both crude and age adjusted values for insulin and HOMA-IR were significantly greater for subjects living in Australia greater than 5 years compared to those living in Australia less than 5 years (Table 3). Adjustment for BMI, WC, HC and %TBF reduced the strength of this association (from $p <0.01$ to $p <0.05$).

The Australian subjects were divided into obese (general obesity with BMI ≥ 25 kg/m^2 and central obesity with

Table 2. Pearson correlation coefficients of anthropometric measures, blood lipid profiles, insulin and HOMA-IR of all Chinese females stratified by country of residence

	Taiwan					Australia				
	BMI	WC	HC	WHR	%TBF	BMI	WC	HC	WHR	%TBF
Insulin (log)	0.29**	0.25*	0.25*	0.11	0.33**	0.39**	0.39**	0.34**	0.25*	0.38**
HOMA-IR (log)	0.27**	0.23*	0.22*	0.11	0.31**	0.43**	0.43**	0.38**	0.27**	0.41**
HDL	0.02	0.03	0.20	-0.13	-0.02	-0.36**	-0.36**	-0.24*	-0.30**	-0.36**
TG (log)	0.15	0.16	0.01	0.20	0.17	0.37**	0.32**	0.26**	0.20*	0.32**
TC	0.28**	0.36**	0.30**	0.22*	0.29**	0.17	0.16	0.21*	0.02	0.15
LDL	0.27**	0.35**	0.22*	0.28**	0.29**	0.23*	0.25*	0.26**	0.10	0.23*
Apo B	0.40**	0.45**	0.25*	0.37**	0.39**	0.23*	0.23*	0.13	0.22*	0.19
LDL diameter	0.14	-0.08	0.04	-0.13	-0.09	-0.29**	-0.20*	-0.13	-0.17	-0.22*
Apo A-1	0.02	0.11	0.28**	-0.10	0.02	-0.14	-0.16	-0.08	-0.16	-0.20*

* $p < 0.05$, ** $p <0.01$

Table 3. Anthropometric and biochemical risk factors of Chinese females stratified by length of residence in Australia: mean ± SD

	In Australia < 5 years (n= 28)	In Australia > 5 years (n=72)
Insulin (pmol/L)		
Mean (range)	32.4 ±10.5 (14-52)	50.0±32.3 (18-189)
Median	31.5	41.5
Logarithmic mean†	1.49±0.15	1.64±0.22 **
(Back transformed)‡	(30.9±1.4)	(43.7±1.66)
Adjusted age †	30.2±2.5	43.7±1.7**
BMI	33.1±2.4	41.7±1.7*
WC	33.1±2.4	41.7±1.7*
HC	32.4±2.4	42.7±1.7*
%TBF	32.1±2.5	42.0±1.7*
HOMA-IR		
Mean (range)	0.90±0.28 (0.43-1.41)	1.45±1.00(0.49-6.08)
Median	0.89	1.17
Logarithmic mean†	-0.07±0.15	0.09±0.23***
(Back transformed) ‡	(0.85 ±1.41)	(1.23 ±1.70)
Adjusted age †	0.85±2.51	1.24±1.78**
BMI	0.93±2.38	1.20±1.70*
WC	0.94±2.40	1.20±1.70*
HC	0.91±2.46	1.21±1.74*
TBF	0.94±2.46	1.20 ±1.74*
TC (mmol/L)	4.6±0.7	4.7±0.7
LDL-C (mmol/L)	2.7±0.7	2.9±0.7
HDL-C (mmol/L)	1.5±0.3	1.4±0.3
TG (mmol/L) †	0.84±1.17	1.00±1.38 **
Apo A1 (mg/dL)	177±31	170±30
Apo B (mg/dL)	106±26	112±33
LDL diameter (nm)	27.2±0.6	27.1±0.7
Glucose (mmol/L)	4.4±0.3	4.5±0.4

*, **, *** Significantly different between Taiwan and Australia at the 0.05, 0.01, 0.001 level respectively. † Statistics and adjustments carried out using log transformed values which were back transformed for comparison with other published values. ‡ Differs from arithmetic mean because of logarithmic transformation.

WC > 80cm) and non-obese in order to determine if the link between IR and length of residence in Australia was different among the obese. Among the generally obese (21%), increased HOMA-IR was associated with length of residence in Australia, while HOMA-IR remained unchanged among the non-obese (Figure 1-1). In subjects who were centrally obese (26%), increased HOMA-IR was associated with length of residence, but this was not so among those with WC less than 80cm (Figure 1-2). A similar pattern was observed for plasma insulin levels (data not shown). Among those living in Australia greater than 5 years, those who were generally obese or centrally obese had about 4 times the odds of having hyperinsulinemia (>60 pmol/L) compared to non-obese subjects (95% CI, 1.18-12.7 for general obesity, and 95% CI, 1.18-12.6 for central obesity).

Discussion

There were no significant differences in crude measures of plasma insulin and HOMA-IR between women living in Taiwan and Australia, but when values for insulin and HOMA-IR were adjusted for a measure of central fat (WC), measures of insulin and insulin resistance were greater in Taiwan. Despite this, Taiwanese females living in Australia greater than 5 years had significantly higher insulin and HOMA-IR compared to those with less than 5 years residence, even after adjustment for age and all measures of fatness. This is a notable finding and is independent of age. Although IR has been found to be strongly associated with aging,[5] age does not appear to be a factor affecting plasma insulin and HOMA-IR in this study. While it is acknowleledged that IR can have genetic causes,[23] and there is evidence that IR may vary among ethnic groups,[24] the Taiwanese ethnicity of all subjects has been confirmed by the Taiwanese author (WPL). Although the higher IR in those with more than 5 years Australian living is independent of measures of fatness, a strong positive correlation between fasting insulin and HOMA-IR with length of residence in Australia was observed only in obese subjects. This finding confirms the studies of Landin *et al.*,[25,26] in which intra-abdominal fat was an important determinant of IR in obese but not in lean subjects.

The findings that measures of central obesity (WC) and general obesity (BMI) were positively associated with insulin and HOMA-IR in Taiwanese females in both Taiwan and Australia are consistent with a recent Taiwanese study of women in Taiwan.[27] WC has been found to be the best anthropometric correlate of the amount of visceral adipose tissue as measured by computed tomography,[28] and BMI is used universally as a measure of general obesity.[29]

Figure 1-1

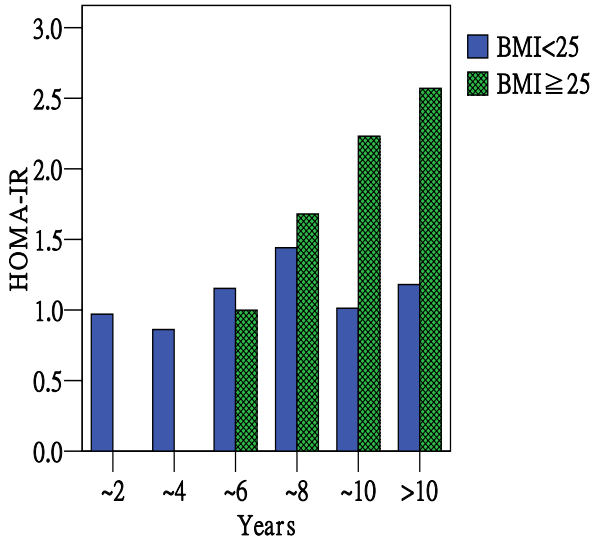

Figure 1-2

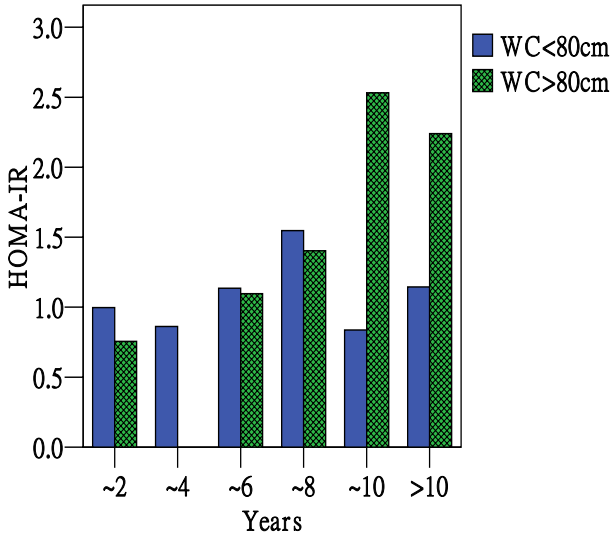

Figure 1. Mean levels of HOMA-IR by length of residence in Australia for nonobese and obese subjects; based on BMI (Figure 1-1) and WC (Figure 1-2).

Our findings that both fasting insulin and HOMA-IR were positively associated with TG and negatively correlated with HDL-C and LDL particle size in the Australian subjects are consistent with those of many other studies, but these associations were attenuated, and even absent, in the subjects living in Taiwan. The reason for the difference between Taiwan and Australia is not clear. Although the associations between IR and blood lipids have been well documented, the evidence comes largely from Caucasian studies with much less data on other ethnic groups.[8-13] There are differences in these associations even among different groups of Asian women,[30] but this cannot explain the inter-country difference in the same ethnic group. LDL particle size, a potent risk factor for T2DM[12] and CHD[13,14] also shows an inverse correlation with fasting insulin, HOMA-IR, and BMI in Australia, but not in Taiwan. There is a significant difference in prevalence of central obesity and also in intake of energy and saturated fat between subjects in the two countries. Hence the differences in relationships between insulin or HOMA-IR and lipid profiles between Taiwanese and Australian Taiwanese females might be related to these factors. Further studies are needed to elucidate these differences.

The strength of the association between body fat patterning and IR also varies among ethnic groups. Albu *et al.*,[31] found a strong positive relationship between visceral adipose tissue and IR in Caucasian women. In contrast, Tai *et al.*,[32] reported a negative relationship between IR and WHR in Chinese women in Singapore. Our results showed a stronger positive correlation between both general obesity and central obesity with insulin resistance measures in Australian Taiwanese females, which is consistent with the results of studies of Caucasian, but not of Chinese, women.

Migrant studies have reported that adopting a western lifestyle, including increased dietary fat, can cause increased prevalence of insulin resistance and T2DM.[33,34] The present study did not find a significant association between either total dietary fat or saturated fat intake and insulin or HOMA-IR among the Australian subjects. However, these subjects do have a higher intake of total energy and saturated fat than their counterparts in Taiwan and more than 5 years of Australian living is associated with higher HOMA-IR. The relationship between dietary fat intake and IR remains controversial. Several epidemiological studies have found that there is a direct relationship between total fat intake and IR among both healthy females and individuals with impaired glucose tolerance.[35,36] In contrast, controlled clinical trials have not observed an association between dietary fat intake and insulin sensitivity in healthy adults, but these studies were of comparatively short duration (1-3 weeks). [37, 38]

Van Dam and coworkers[39] found that higher fat intake was associated with lower insulin sensitivity among obese but not among non-obese subjects. This may suggest a role of obesity in mediating the association between fat intake and the degree of insulin resistance. The Kaiser Twins Study[35] has reported that a 20g/day increase in total fat intake was associated with a 9% ($p< 0.001$) and 6% ($p< 0.01$) higher fasting insulin levels among non-diabetic women before and after adjustment for BMI, respectively. Australian Taiwanese females had a mean intake of total fat 20g/day higher than their counterparts in Taiwan[18], and subjects living in Australia greater than 5 years had about 10% higher fasting insulin and HOMA-IR than subjects living in Australia less than 5 years. However, the higher measures of insulin resistance (HOMA-IR) in those with greater than 5 years Australia residence were entirely due to those subjects who were obese. Thus obesity appears to mediate the association between increased fat intake and IR among those Australian Taiwanese females.

Carbohydrate-containing foods vary systematically with respect to their effects on postprandial glucose and insulin response, and this is categorized as a glycemic index.[40]

There is growing evidence that foods with high glycemic index are associated with various metabolic diseases and obesity.[41,42] Although there was no significant difference in dietary intakes with length of residence in Australia among Australian Taiwanese females, about 57% of energy intake is carbohydrate.[18] Since increased fasting insulin levels and HOMA-IR were associated with length of residence in Australia, it will be important to further analyze the source and quality of dietary carbohydrates in relation to obesity and insulin resistance in this group.

Epidemiological studies have suggested that exercise protects against the development of IR in Chinese and Caucasian populations.[43,44] It has been postulated that the mechanism of action might be related to decreasing body fat and promoting the utilization of glucose in skeletal muscle.[45,46] In this study, the duration of exercise was found to be a significant predictor of insulin resistance in Australia, but not in Taiwan. This is important as there are few studies on the influence of exercise in healthy Chinese populations. Physical activity from occupational work has been reported to be related to decreased insulin resistance among Chinese females.[43] We have previously reported that 50% of the subjects in Taiwan were employed, compared to only 18% of the subjects in Australia.[18] The physical activity measured in the present study is focused on leisure-time physical exercise, not occupational physical activity. This may underestimate the time spent exercising among subjects in Taiwan and obscure the relationship between exercise and insulin resistance. Thus non-vigorous pursuits may be equally beneficial for women in the prevention of insulin resistance.

Our study has indicated that Taiwanese females living in Australia initially appear to have a more favorable state of IR than those in Taiwan, but insulin resistance is associated with length of residence in Australia. This association was predominantly found among those generally and centrally obese. A "healthy migrant effect", where those in good health are more likely to meet eligibility criteria and be willing to migrate, has been previously reported.[47] In Australia, this effect has been found to last some years after arrival, but lessens with increasing length of residence.[48] Our results support this finding. As this study was designed as cross-sectional, there are no data on causality. Longitudinal inter-country studies involving greater detail of dietary intake (including glycemic index) will be necessary to fully understand the relationships between IR, body fat and diet in Chinese immigrants to Australia.

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Original Article

Insulin, lipid profiles and measures of fatness in Taiwanese women in relation to duration of residence in Australia

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澳洲的台灣女性胰島素、血脂質及肥胖測量與居留時間的相關性

研究計畫為探討居住在台灣(n=97)或是澳洲(n=100)的台灣女性肥胖測量、血中胰島素與脂質之間的相關性，並評估居留在澳洲時間長短對那些相關性的影響。採用 Reflotron 測量空腹血糖與脂質，採用 Microparticle 酵素免疫分析法測量禁食胰島素濃度；胰島素阻抗(IR)則用 HOMA 確認。血漿胰島素或是 HOMA-IR 在台灣或澳洲國家間均沒有顯著差異((分別為 51.7±42.2 vs. 45.0±29.0 pmol/L 和 1.43±1.21 vs. 1.29±1.00，所有 $p > 0.05$))，但當胰島素和 HOMA-IR 經腰圍校正後，台灣則較高(分別為 45.7±1.6 vs. 38.0±1.6 pmol/L 和 1.26±1.59 vs. 1.13±1.59，所有 $p< 0.05$)。甚至在校正所有肥胖測量值之後，居留在澳洲超過五年者比居留時間少於五年者有較高的胰島素與 HOMA-IR 值(分別為 50.0±32.3 vs. 32.4±10.5 pmol/L 和 1.45±1.00 vs. 0.90±0.28，所有 $p< 0.01$)。居留在澳洲超過 5 年者，其胰島素大於 50pmol/L 的危險性增加六倍；這增加的危險性僅限於一般性或是中央型肥胖的女性。在兩個國家均顯示出，中央型肥胖與全身性肥胖的測量值與 HOMA-IR 呈現正相關(r = 0.23, 台灣女性 $p < 0.05$ 和 0.27 $p < 0.01$, 澳洲女性 0.43 和 0.43, 全部 $p< 0.01$)。台灣女性居住在澳洲開始時顯示比起居住在台灣者有較佳的 IR，但是胰島素阻抗與居留在澳洲時間長短較具有相關性，尤其是肥胖者。

關鍵字：胰島素阻抗、飲食、肥胖、血脂質、台灣人、澳洲的台灣移民。