Abstract

Background: Diets with a high postprandial glycemic response may contribute to long-term development of insulin resistance and diabetes, however previous epidemiological studies are conflicting on whether glycemic index (GI) or glycemic load (GL) are dietary factors associated with the progression. Our objectives were to estimate GI and GL in a group of older women, and evaluate cross-sectional associations with insulin resistance.

Subjects and Methods: Subjects were 329 Australian women aged 42-81 years participating in year three of the Longitudinal Assessment of Ageing in Women (LAW). Dietary intakes were assessed by diet history interviews and analysed using a customised GI database. Insulin resistance was defined as a homeostasis model assessment (HOMA) value of >3.99, based on fasting blood glucose and insulin concentrations.

Results: GL was significantly higher in the 26 subjects who were classified as insulin resistant compared to subjects who were not (134±33 versus 114±24, $P<0.001$). In a logistic regression model, an increment of 15 GL units increased the odds of insulin resistance by 2.09 (95%CI 1.55, 2.80, $P<0.001$) independently of potential confounding variables. No significant associations were found when insulin resistance was assessed as a continuous variable.

Conclusions: Results of this cross-sectional study support the concept that diets with a higher GL are associated with increased risk of insulin resistance. Further studies are required to investigate whether reducing glycemic intake, by either consuming lower GI foods and/or smaller serves of carbohydrate, can contribute to a reduction in development of insulin resistance and long-term risk of type 2 diabetes.

Key words: Glycemic index, glycemic load, carbohydrate, insulin resistance, women, LAW study

Introduction

Glycemic intake, measured by glycemic index (GI) and glycemic load (GL), describes the rate of digestion of carbohydrate foods and absorption of glucose into the bloodstream. These empirically-derived measures provide a way to quantify the impact of carbohydrate on circulating blood glucose concentrations (Jenkins *et al*, 1981). The GI of a food represents the rise in blood glucose two hours after consumption of a 50 g serve of the food compared to 50 g glucose, and is given as a percentage (Jenkins *et al*, 1981). The GL of a food varies with serve size and is calculated as the food's GI multiplied by the amount of carbohydrate in the serve. Meals low in glycemic intake result in lowered postprandial increases in blood glucose and insulin concentrations (Holt *et al*, 1997), however the possible long-term impact of sustained low glycemic diets on reducing the risk of chronic disease such as type 2 diabetes mellitus is currently an area of controversy (Miles, 2008).

One early predictor of type 2 diabetes is the condition of insulin resistance (Weyer *et al*, 1999, Martin *et al*, 1992), defined as the inability of a known quantity of insulin to increase glucose uptake and utilisation in an individual as much as it does in a healthy population (Lebovitz, 2001). In a five year follow-up study of 6000 adults, people who were insulin resistant were 15 times more likely to develop diabetes than people who were not (Barr *et al*, 2006). The transition from insulin resistance to clinical diabetes is thought to be a progressive process that is associated with a decline in pancreatic islet beta-cell function and cell mass (Kahn, 2003). Dietary factors that could slow or reverse this progression have the potential to reduce the risk of developing diabetes.

In a meta-analysis of prospective observational studies, Barclay *et al* (2008) concluded that diets with a high GI or GL independently increased the risk of type 2 diabetes, although not all studies were supportive. Previous cross-sectional studies of insulin resistance have found significant positive associations with both GI and GL (McKeown *et al*, 2004) or no significant associations (Lau *et al*, 2005, Liese *et al*, 2005).

A potential limitation of these previous observational studies, noted by Barclay *et al* (2008), has been that the food frequency questionnaires (FFQ) used to measure dietary intake were not validated for GI or GL using another dietary method or against an objective standard. FFQ's have been the tool of choice for assessing dietary intake in populations, as they are feasible and economical to administer on a large scale and analysis is relatively quick using the specified food list as a template for data entry. However the food and serve size options listed could restrict the amount and type of information obtained compared to a food record or diet history interview, in which individual foods and serve sizes are directly quantified (Cameron and Van Staveren, 1988, Sempos, 1992). The impact of glycemic intake on the risk of chronic diseases could be underestimated if individual serve sizes for carbohydrate foods are not accurately quantified, contributing to some of the inconsistent findings from previous studies. The current study was conducted as part of a larger assessment of ageing in a group of older women. Our objectives were to estimate usual dietary GI and GL using a detailed diet history interview and to evaluate associations with insulin resistance.

Methods

Subjects

A total of 511 women participated in the Longitudinal Assessment of Ageing in Women (LAW study), an age-stratified multidisciplinary study conducted at the Royal Brisbane and Women's Hospital, Australia. At the beginning of the study, subjects were randomly selected from the electoral roll within four age cohorts: 40–49, 50–59, 60–69 and 70–79 years (Khoo *et al*, 2008). Data for the current study were collected during year three of the LAW study. Subjects were excluded if they were: confirmed by the study clinician to have diabetes based on self-report, use of medication, and/or fasting glucose concentrations (>6.0 mmol/L) (World Health Organisation, 1999); taking an oral hypoglycemic agent; unable to provide a fasting blood sample or participate in a diet history;

classified as under-reporters of energy intake (estimated energy intake: estimated energy expenditure <0.76) (Black, 2000); or if less than 85% of the carbohydrate in their diet was able to be allocated a GI value. Study procedures were approved by the Human Research Ethics Committees of Queensland University of Technology and Royal Women's Hospital. Subjects gave informed written consent

Assessment of dietary intake

Usual intake was assessed by a dietitian during a standardised diet history interview (Tapsell *et al*, 2000, Cameron and Van Staveren, 1988) focussing on the amount and type of carbohydrate items consumed in a typical month over the past six months. Food models and measuring displays were used to help precisely assess serve sizes and detailed information was sought on foods and beverages, including brand names and cooking procedures. An 80-item frequency checklist was included to detect possible omissions from the diet history. Data were analysed using the Foodworks dietary analysis program (Professional Version 4.00, Xyris Software, Brisbane) and the Australian Food and Nutrient database of Australian foods, combined with a customised GI database comprising published GI values (University of Sydney, 2007, Foster-Powell *et al*, 2002) and values estimated from similar foods or calculated from constituent foods where appropriate. The dietary GL was calculated as the product of the GI and carbohydrate content for each food, summed for all foods eaten and given as a daily value. The dietary GI, given as a percentage, was calculated as the product of the GI and carbohydrate content for each food, summed for all foods eaten during the day, then divided by the total daily carbohydrate intake (Jenkins *et al*, 1981).

Measurement of insulin resistance

Insulin resistance was assessed using homeostasis model assessment (HOMA), where HOMA=fasting plasma insulin (mU/L) x fasting plasma glucose (mmol/L)/22.5 (Matthews *et al*, 1985). Due to the degree of natural variation in both fasting glucose and insulin concentrations, we

used a HOMA cut-point to identify subjects with high HOMA results. For this study, subjects were categorised as insulin resistant if HOMA was >3.99 (Wahrenberg *et al*, 2005). As there is no definitive HOMA cut-point to represent insulin resistant status, associations were also investigated using values from 2.00 to 5.99.

Measurement of potential confounding variables

Clinical data were collected by interview with the study clinician. Menopausal and hormone therapy (HT) status groups were: premenopausal; HT user; post/perimenopausal and HT non-user or short-term user (<12 months). Family history of diabetes was defined as a first-degree blood relative diagnosed with type 1 or type 2 diabetes mellitus. Smoking was investigated in terms of pack years and smoking status (never/current/ex-smoker). Subjects were classified by a physiotherapist into one of six physical activity levels, based on incidental and purposeful exercise (Hirvensalo *et al*, 2000); these were subsequently collapsed into two levels: active (walk or other activity ≥2/week) or sedentary (activity <2/week) after statistical analyses showed no significant differences between using two or six levels. Anthropometric measures were obtained by a trained operator using standard methodology (Gibson, 1993). Waist and hip circumference was measured to the nearest 0.1 cm; height was assessed to the nearest 0.1 cm with a stadiometer (Holtain Ltd, Crymych, UK) and weight was measured to the nearest 0.01 kg using a Seca standing scale (B.P.S Instruments, US). Body mass index (BMI) was calculated as weight in kilograms divided by the square of height in metres. Average daily intakes of saturated fat, alcohol, dietary fibre and energy were assessed from the diet history.

Statistical analysis

Independent t-tests were used to evaluate differences between continuous measures of glycemic intake based on insulin resistant status (yes/no). Chi-square tests were used to locate significant differences in proportions with insulin resistance between pairs of tertiles. ANOVA was used to

assess differences in dietary intake across tertiles of GI and GL. Multivariate logistic regression models were used to evaluate the relationships between insulin resistance (as a dichotomous measure) and glycemic intake (GI or GL), adjusted for age, age squared, BMI, waist circumference, family history of diabetes, menopausal and HT status, physical activity level, daily intakes of energy, alcohol and dietary fibre, and percent saturated fat intake. Using log HOMA measurements, insulin resistance was also considered as a continuous response in multivariate linear regression models. Step-wise removal of the variables with the least significant association with IR status (or log HOMA) was performed until all variables were significant. Non-significant variables were re-tested in the parsimonious model before their exclusion was confirmed. Statistical significance was set to 5% and analysis was completed using the Statistical Package for Social Sciences (SPSS) for Windows (release 14.0). Values are expressed as mean±SD except where stated.

Results

Subjects

Of the 511 subjects who commenced the LAW study, 470 completed a diet history in year three. Reasons for non-completion were inability to attend the appointment (n=24), illness (n=13), death (n=2), and unwillingness to participate (n=2). A further 11 were identified as reporting unacceptably low energy intakes and 40 did not have full data on potential confounding variables, leaving 419 eligible subjects. Of these, GI and GL values were not able to be allocated to 90 subjects, with 89.7% of the total carbohydrate intake of the group allocated a GI value. The remaining 329 subjects were included in the statistical analysis; 7.9% (n=26) of these were identified as insulin resistant. Characteristics of subjects who were included were not significantly different to those who were not included (**Table 1**). Mean GI was 55.7±4.4%, with a range of 44.5-77.2% and an interquartile range (IQR) of 52.7-58.4%. Mean GL was 115±26, with a range of 47-236 and an IQR of 98-130. Intakes between GI tertiles were significantly different for dietary fibre,

protein, and fat, while intakes between GL tertiles were significantly different for energy, carbohydrate, dietary fibre, fat and protein (**Table 2**).

Associations between glycemic intake and insulin resistance status

GL was significantly higher in subjects who were insulin resistant compared to subjects who were not insulin resistant (P<0.001); there were no significant differences in GI (P=0.68) (**Table 3**). Subjects with insulin resistance were significantly older and had a higher BMI and waist to hip ratio than subjects without insulin resistance (**Table 3**). In a logistic regression model, an increment of 15 GL units increased the odds of prevalent insulin resistance by 2.09 (95%CI 1.55, 2.80) independently of potential confounding factors (P<0.001). GI was not significantly associated with insulin resistant status in the model (*P*=0.56). When analyses were re-run to include all subjects who participated in the dietary analysis (n=470), an increment of 15 GL units increased the odds of insulin resistance by 1.45 (95% CI 1.08, 1.95).

Insulin resistance was significantly higher in the third GL tertile (highest) compared to second (*P*=0.04) and first (*P*=0.002) tertiles (**Figure 1**). In a logistic regression model, adjusted for potential confounding factors, the odds of insulin resistance for subjects in the third GL tertile were 12.7 times higher compared with the first tertile (95%CI 1.6, 100, P=0.02) and 6.3 times higher compared with the second tertile (95%CI 1.4, 28.6, P=0.02). Conversely, the proportion with insulin resistant status was not statistically different between GI tertiles (*P*=0.69) or between GI tertiles after adjustment for potential confounding variables (*P*=0.25).

In logistic regression models adjusted for potential confounding variables, GL remained a significant predictor of insulin resistant status classified using HOMA cut-points from 3.25 to 4.99, in which the proportion of subjects classified as insulin resistant varied from 12% to 3%. The highest odds were observed for HOMA values between 3.75 and 4.99 (**Table 4**). Results of separate

multivariate linear regression models using HOMA as a continuous measure of insulin resistance showed no statistically significant association between GI (P=0.44) or GL (P=0.18).

Discussion

We observed an independent positive relationship between GL and insulin resistance in a group of Australian women participating in the Longitudinal Assessment of Ageing in Women (LAW). Mean GL intake was 18% higher in women with insulin resistance compared to those without insulin resistance ($P<0.001$) (**Table 3**). When adjusted for potential confounding variables in a logistic regression model, an increase of 15 GL units was associated with a two-fold increase in the odds of insulin resistance. This trend was also observed with GL tertiles, although the wide confidence intervals indicated that the categorical variable may be an imprecise measure of glycemic intake in this group (**Figure 1**). However, the magnitude and consistency of this effect supports a clinically-significant association between GL and insulin resistance in the LAW women.

In contrast to the GL findings, there was no significant association between GI and insulin resistance, either unadjusted or adjusted for potential confounding variables. Measures of GI and GL differ in quantification of glycemic intake. The GI ranks foods according to the digestion and absorption of the carbohydrate, and is a measure of the area under the 2-hour postprandial blood glucose curve after consuming the food, in relation to the same quantity of glucose (Jenkins *et al*, 1981). A low GI is achieved by consuming the majority of carbohydrate in the diet from low GI foods and is independent of total carbohydrate intake, with our results showing no significant difference in carbohydrate intake between tertiles of GI (**Table 2**). The GL represents the GI while also taking into account the carbohydrate content of the food. It is designed to capture the magnitude of the effect of dietary carbohydrates on postprandial blood glucose concentrations. A low GL can be achieved by consuming a low GI, moderate carbohydrate diet but also a moderate GI, low carbohydrate diet. Barclay *et al* (2005) proposed that for diets with the same GL, one with a

high amount of carbohydrate from low GI foods has potentially more beneficial metabolic effects than one with a low amount of carbohydrate from high GI foods. In our cross-sectional study, we were not able to distinguish between these alternatives, and further studies are required to clarify the metabolic effects of GL within differing dietary patterns.

Our study supports a proposal by Hu *et al* (2001) that GL is a more physiologically-relevant measure than GI in terms of associated risk with chronic disease. However, the absence of a significant association with GI is not conclusive, due to the limited variation of intakes in the LAW women shown by the narrow IQR of 52.7-58.4% and relatively small standard deviation of 4.4%. Other observational studies with small standard deviations have not reported significant associations with GI (Liese *et al*, 2005, Zhang *et al*, 2006). Sahyoun *et al*. (2008) also noted that homogeneity of glycemic intake values within their population could have decreased the likelihood of an association. Buyken and Liese (2005) observed that the strongest association between GI and risk of diabetes was shown in the study with the largest variation in GI intake (Hodge *et al*, 2004). The wider variation in values for GL that we observed in the LAW cohort may have contributed to the significant association we found between GL and insulin resistance.

The HOMA cut-point to define insulin resistant status has ranged from 2.0 to 4.65 in different studies. The original research showed HOMA values for subjects without diabetes were approximately 2.0 compared with 2.5 for subjects with diabetes (Matthews *et al*, 1985). In a cross-sectional study of older Swedish men and women without diabetes, Hedblad and colleagues (Stern *et al*, 2005, Hedblad *et al*, 2000) chose 2.0 as the cut-off point, which marked the 75th percentile for HOMA scores, assuming that the top 25% of subjects were insulin resistant. Stern *et al* (Stern *et al*, 2005, Hedblad *et al*, 2000) in an evaluation of European and American individuals without diabetes compared various methods with the euglycemic clamp and concluded that a HOMA score greater than 4.65 indicated insulin resistance; as this was a highly specific decision rule, authors noted that

the higher cut-off would likely misclassify some insulin-resistant subjects as non-insulin resistant. Wahrenberg *et al* (2005) used a 3.99 cut-off to classify insulin resistant status in Swedish subjects, based on the 90th percentile and previous research (Ascaso *et al*, 2001). The relationship we observed between GL and insulin resistant status remained significant for HOMA cut-off values between 2.99 and 4.99, with the strongest associations observed between values of 3.75 and 3.99, suggesting that 3.99 was an appropriate cut-off to use in our population (**Table 4**). Statistically significant results were not seen when using a continuous measure of HOMA to represent insulin resistance, suggesting that significant differences in GL are only evident in subjects with clinically high HOMA values. HOMA values can range from less than one to 2.7 based on fasting glucose and insulin concentrations considered to be normal (Dunstan *et al*, 2002, Worthley, 2003), and use of HOMA as a continuous measure may mask potential associations with dietary factors.

Although the precise cellular mechanisms of insulin resistance are not yet clear (Shulman, 2000, Petersen and Shulman, 2006), our finding is supported by proposed mechanisms to explain how high glycemic intakes could promote insulin resistance. Sustained high postprandial glucose concentrations resulting from meals with a high glycemic response can lead to increased storage of triglycerides within muscle cells (Cooney and Storlien, 1994), and skeletal muscle triglyceride concentration has been shown to be inversely related to insulin action in both animal models (Storlien *et al*, 1991) and humans (Pan *et al*, 1997, Petersen and Shulman, 2006). Postprandial hyperglycemia has also been shown to contribute to oxidative stress with production of reactive oxygen species (Stamler *et al*, 1993, Ceriello *et al*, 1998, Marfella *et al*, 2001) that could damage pancreatic beta cells and possibly affect the production of insulin (Augustin *et al*, 2002). These beta cells are particularly at risk as they have lower levels of intrinsic antioxidant defences compared to other tissues (Robertson, 2004). However, the results of our cross-sectional study cannot be used to infer a causal relationship between GL intake and insulin resistance. Insulin resistance has the

potential to create an environment of a relative glucose deficiency, which may trigger preferences for foods high in GI and carbohydrate, which in turn produce a higher glycemic response.

A strength of our study was the comprehensive assessment of glycemic intake. Individual diet history interviews were used to collect detailed data on carbohydrate serve sizes and the range of items consumed during the day. This allowed precise characterisation of GI and GL intakes for each woman and minimised measurement error, an important consideration when evaluating possible associations between glycemic intake and individual risk of chronic diseases. Imprecise estimates of GI and GL from FFQs that were not originally designed to assess GI or GL or validated for this purpose, may have partially contributed to contradictory findings in previous observational studies of insulin resistance (Barclay *et al*, 2008, McKeown *et al*, 2004, Lau *et al*, 2005, Liese *et al*, 2005).

A potential limitation of our study was that 90 women were excluded as less than 85% of their carbohydrate intake was able to be allocated a GI value. This criterion was set to ensure the estimated values were as precise as possible; a lower value would have included more subjects but decreased the precision of the values. There were no statistically significant differences between subjects allocated and not allocated GI values (**Table 1**), although there were non-significant trends towards a greater proportion of younger women and women with higher BMI in the group who were not allocated a value. Results showed that increasing the subject numbers to include the excluded subjects decreased the strength but did not change the direction or significance of the findings.

From this study of Australian women, we conclude that higher dietary GL is associated with a statistically significant increased risk of being insulin resistant, after adjustment for potential confounding variables. Intervention studies are required to investigate whether reducing glycemic

intake, by either consuming lower GI foods and/or smaller serves of carbohydrate, can contribute to a reduction in development of insulin resistance and long-term risk of type 2 diabetes.

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Table 1. Characteristics of LAW study subjects who were and were not allocated GI and GL values

Characteristic	Subjects allocated a GI/GL value[†] n=329	Subjects not allocated a GI/GL value[†] n=90	P value[‡]	Total eligible subjects n=419
Age group (%)			*0.07*	
42-51 years	23.1	35.6		25.8
52-61 years	25.5	21.1		24.6
62-71 years	28.0	18.9		26.0
72-81 years	23.4	24.4		23.6
Activity level (%)			*0.30*	
Active (walk or other activity ≥ 2/week)	66.0	71.9		67.3
Sedentary (activity less than 2/week)	34.0	28.1		32.7
Menopausal and hormone therapy (HT) status (%)			*0.29*	
Premenopausal	12.7	19.3		14.2
Using HT ≥12 months	43.8	39.8		42.9
Peri or postmenopausal, and using HT <12 months	43.5	40.9		42.9
Smoking status (%)			*0.80*	
Non-smoker	54.7	53.3		54.4
Ex-smoker	36.5	35.6		36.3
Current smoker	8.8	11.1		9.3
BMI (kg/m^2), mean±SD	26.5±4.8	27.6±5.2	*0.06*	26.8 ± 4.9
Waist to hip ratio, mean±SD	0.80±0.1	0.80 ± 0.1	*0.99*	0.80 ± 0.1
Homeostasis model assessment, mean±SD	1.9±1.3	1.9±1.9	*0.94*	1.9±1.5

[†]Dietary GI and GL values were allocated if above or equal to 85% of daily carbohydrate intake was assigned a GI value.
[‡]P value for comparison of proportions (chi-square tests) or means (independent t-tests) between the group in which GI/GL could be allocated and the group in which GI/GL could not be allocated.

Table 2. Comparison of daily dietary intake between tertiles of glycemic index (GI) and glycemic load (GL) (n=329)

Daily nutrient intake (mean±SD)	GI Tertile			*P*	GL Tertile			*P*
	0-54.0	54.0-57.4	57.4+		0-103	103-125	125+	
Energy (MJ)	8.42 ± 1.4	8.47 ± 1.2	8.41 ± 1.4	*0.94*	7.54 ± 1.1	8.46 ± 1.0	9.27 ± 1.1	*<0.01*
Carbohydrate (g)	232 ± 43	225 ± 41	225 ± 54	*0.43*	181 ± 25	226 ± 20	274 ± 23	*<0.01*
Carbohydrate (%)	47.9 ± 5.8	46.1 ± 6.5	46.0 ± 7.5	*0.06*	42.0 ± 5.7	46.7 ± 5.9	51.2 ± 4.9	*<0.01*
GI	51.1 ± 2.3	55.6 ± 1.0	60.2 ± 2.9	*<0.01*	54.7 ± 4.3	5.54 ± 4.4	56.9 ± 4.1	*<0.01*
GL	109 ± 22	113 ± 22	123 ± 30	*<0.01*	88.9 ± 11	113 ± 7	143 ± 18	*<0.01*
Dietary fibre (g)	30.0 ± 6.6	26.7 ± 7.3	25.2 ± 7.3	*<0.01*	24.4 ± 6.6	28.0 ± 7.1	29.3 ± 7.5	*<0.01*
Protein (g)	91.8 ± 17	87.5 ± 14	83.7 ± 14	*<0.01*	83.6 ± 15	87.2 ± 15	91.9 ± 16	*<0.01*
Protein (%)	19.0 ± 2.9	18.0 ± 2.5	17.4 ± 2.7	*<0.01*	19.3 ± 3.1	17.9 ± 2.5	17.2 ± 2.3	*<0.01*
Fat (g)	69.5 ± 20	72.3 ± 18	74.6 ± 19	*0.12*	69.6 ± 18	73.0 ± 20	73.9 ± 18	0.21
Fat (%)	30.8 ± 6.1	32.0 ± 5.7	33.3 ± 5.7	*0.01*	34.3 ± 5.7	32.1 ± 6.2	29.7 ± 4.8	*<0.01*
Saturated fat (g)	24.2 ± 8.7	26.8 ± 8.4	27.8 ± 9.6	*0.01*	24.8 ± 8.7	26.5 ± 9.8	27.5 ± 8.4	0.08
Saturated fat (%)	10.5 ± 2.8	11.6 ± 3.0	12.2 ± 3.8	*<0.01*	12.1 ± 3.6	11.4 ± 3.4	10.9 ± 2.7	0.03

Table 3. Comparison of glycemic intake and other characteristics between subjects who were and who were not classified as insulin resistant[†].

Characteristic (mean±SD)	Non IR[†] n=303	IR[†] n=26	P value[‡]	Total n=329
GI (%)	55.7±4.5	56.0±3.3	*0.68*	55.7±4.4
GL	114±24	134±33	*<0.01*	115±26
Energy intake (MJ)	8.4±1.4	8.8±1.5	*0.15*	8.4±1.4
Age (years)	61±10	66±9	*0.02*	62±10
BMI (kg/m^2)	26.1±4.4	31.7±6.6	*<0.01*	26.5±4.8
Waist to hip ratio	0.80±0.1	0.84±0.0	*<0.01*	0.80±0.1
Homeostasis model assessment (HOMA)	1.6±0.8	5.4±1.5	*<0.01*	1.9±1.3

[†]Subjects were classified as insulin resistant (IR) if HOMA was >3.99.
[‡]P value for comparison of means between subjects who were and were not classified as insulin resistant (t-tests).

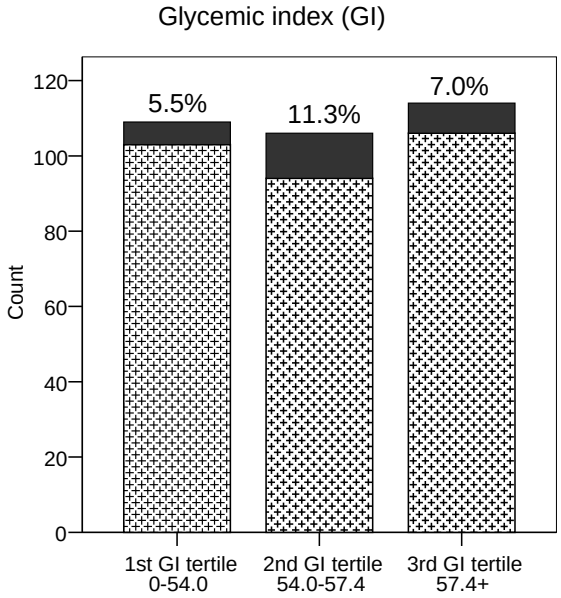
Table 4. Variation in the odds of insulin resistant status for a one unit increase in glycemic load using different homeostasis model assessment (HOMA) cut-points in multivariate logistic regression models.

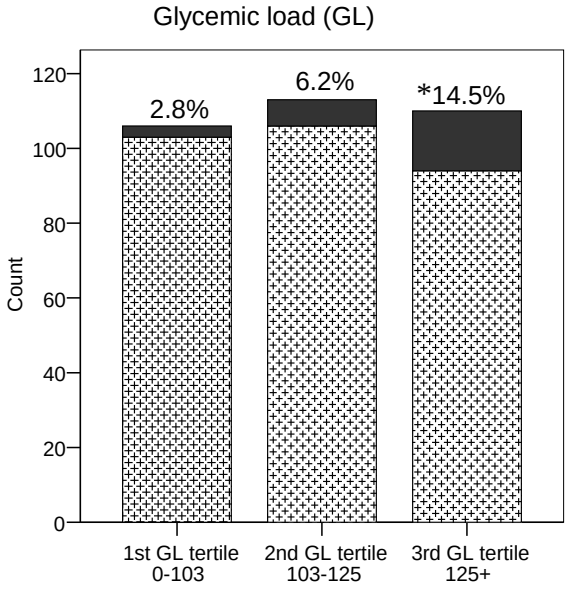
HOMA cut-point for insulin resistant status (≥)	Number (%) of subjects classified as insulin resistant	Odds of insulin resistant status (95% CI)
1.99	116 (35%)	1.00 (0.99, 1.02)
2.50	79 (24%)	1.01 (0.99, 1.03)
2.99	44 (13%)	1.02 (1.00, 1.04)
3.25	39 (12%)	1.03 (1.00, 1.05)
3.50	32 (10%)	1.04 (1.01, 1.06)
3.75	28 (9%)	1.05 (1.02, 1.08)
3.99†	26 (8%)	1.05 (1.03, 1.07)
4.25	20 (6%)	1.03 (1.00, 1.07)
4.50	15 (5%)	1.06 (1.01, 1.11)
4.75	13 (4%)	1.05 (1.00, 1.10)
4.99	11 (3%)	1.06 (1.01, 1.11)
5.50	10 (3%)	1.04 (1.00, 1.09)
5.99	8 (2%)	1.05 (1.00, 1.10)

†cut-point used in current study

Figure 1. Percentage of subjects in glycemic index (GI) and glycemic load (GL) tertiles who were insulin resistant (black) compared with subjects who were not insulin resistant (cross hatch). Percentage of insulin resistant subjects in each tertile is stated at top of columns.

* 3rd tertile (highest) significantly greater than 1st tertile (lowest) ($P<0.002$) and 2nd tertile ($P<0.04$).


Glycemic index (GI)
Count
120
100
80
60
40
20
0
5.5%
11.3%
7.0%
1st GI tertile
0-54.0
2nd GI tertile
54.0-57.4
3rd GI tertile
57.4+


Glycemic load (GL)
Count
120
100
80
60
40
20
0
2.8%
6.2%
*14.5%
1st GL tertile
0-103
2nd GL tertile
103-125
3rd GL tertile
125+