Journal of
Kerman University of Medical Sciences

https://jkmu.kmu.ac.ir
10.34172/jkmu.2024.42
Vol. 31, No. 5, 2024, 272-277

Original Article

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Is Nutritional Status Associated with Quantitative Insulin Sensitivity Check Index in the First Trimester of Gestation?

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Abstract

Background: As pregnancy progresses, insulin sensitivity (SI) might slowly decrease to 50% of the average value, which could result in gestational diabetes. As weight gain is associated with reduced SI and vice versa, we evaluated the possible correlation between nutritional status and SI, especially in early pregnancy.

Methods: This cross-sectional study was conducted on 138 healthy primiparous women between 6 and 10 weeks of gestation. By using a researcher-made questionnaire, the characteristics of the participants were gathered. A digital scale was used to measure participants' weights. Nutrient intake was estimated based on the 72-hour dietary recall by Nutritionist 4 software. We used the pregnancy physical activity scale to estimate physical activity. In addition, fasting plasma glucose (FPG) and insulin values were included in the relevant formula to calculate the quantitative index of SI.

Results: After adjusting for significant maternal characteristics, the quantitative insulin sensitivity check index (QUICKI) in the first trimester was correlated with participants' weight and household income. Furthermore, the QUICKI index had a significant inverse relationship with saturated and polyunsaturated fatty acids (PUFAs) intake while total fat intake was positively correlated. Also, consuming vitamin C, glucose, fructose, sugar, and carbohydrates increased the QUICKI index, while vitamin E intake decreased it.

Conclusion: The results showed that weight management may prevent gestational diabetes mellitus (GDM) during the first trimester. Moreover, the significant correlation between the above-mentioned nutrients and household income with the QUICKI index can be further examined in future studies.

Keywords: Insulin sensitivity, Nutrient intake, Macronutrient intake, Micronutrient intake, Pregnancy

Citation: Atazadegan MA, Yazdi M, Biabanaki-Goortani A, Goodarzi-Khoigani M. Is nutritional status associated with quantitative insulin sensitivity check index in the first trimester of gestation? *Journal of Kerman University of Medical Sciences*. 2024;31(5):272–277. doi: 10.34172/jkmu.2024.42

Received: October 18, 2022, **Accepted**: October 7, 2023, **ePublished**: October 30, 2024

Introduction

As pregnancy progresses towards the third trimester, insulin sensitivity (SI) might slowly decrease to 50% the normal value, which is associated with resistance to insulin activity (IR) and, consequently, glucose consumption in target tissues (1). This IR causes fat to be used in the mother cells and carbohydrates remain for the developing fetus (2), but sometimes can lead to gestational diabetes, which causes complications for the mother and the fetus (3). Weight gain has a two-way relationship with the reduction of SI. Therefore, determining the possible correlation between nutritional status and SI, especially in early pregnancy, is rational and preventive, as researchers have shown that lower vitamin D in early pregnancy is related to IR in the second trimester (4). A systematic review concluded that animal protein might decrease SI while plant protein might enhance that (5). Allman et al reported that maternal body mass index (BMI) in the first trimester was more correlated with SI than with protein intake (6). Another study showed that during early pregnancy, BMI is more effective on the risk of gestational diabetes mellitus (GDM) than nutrient or food intake (7). Although nutrient metabolism is essential for the survival of all organisms (8), there are few published reports concerning nutrient intake and gestational SI development. Thus, we investigated the relationship between nutritional status and QUICKI index in the first trimester of gestation.

Methods

This cross-sectional descriptive analytical survey was conducted on 138 primiparous participants aged 18 to 40 years, while healthy women with 6 to 10 weeks of pregnancy were enrolled. By selecting primiparous women, the population under study became homogeneous, and

confounding effects of parturition on study results were eliminated.

Women with twin or multiple pregnancies and addiction were excluded. Also, diseases affecting body weight such as untreated thyroid disorders, hypertension, type 1 or 2 diabetes, following a special diet, nutritional problems, anemia, chronic disease, kidney disease, and BMI ≥ 35 kg/m^2 were other criteria (9). Using stratified sampling, five hospitals, 15 private offices, and 15 private offices were selected to introduce pregnant women to achieve a sample with different socioeconomic backgrounds. At the significance level of 0.95% ($\alpha = 0.05$), power of 0.80% ($\beta = 0.20$), and $P = 0.3$ as the least acceptable correlation in terms of performance, the minimum sample size with a 10% dropout rate was 134 (10).

$$n = \frac{(z_{1-\alpha/2} + z_{1-\beta})^2}{C(r)^2} + 3$$

A researcher-made questionnaire was used to collect the participants' characteristics. To determine the nutritional status of pregnant women, we first extracted the pregravid BMI from the prenatal files, then we measured the weight and the weight gained in the first 14 weeks of pregnancy with a digital scale. In addition, we extracted the received nutrients from the 72-hour memory with Nutritionist 4 software (First Databank Inc., Hearst Corp., San Bruno, CA - Version 3.5.2). Physical activity was estimated with the physical activity scale of pregnant women, whose validity and reliability have also been examined in Iranian women (11). QUICKI was computed using the fasting plasma glucose (FPG) and fasting serum insulin (FSI) levels based on the formula below (12):

QUICKI = 1/(log FPG in mg/dl + log FSI in μIU/ml).

FSI levels were quantified by electrochemiluminescence immunoassay at Al-Zahra Clinical Laboratory using available relevant kits. Fasting glucose values were measured by means of automatic biochemical analyzer (Mindray bs-800) using Bionik kits.

Statistical analysis

SPSS 20 software was used to analyze the obtained data. The normality of the distribution of continuous variables was appraised with the Q-Q diagram. We adjusted nutrient intake for energy by the residual method. We entered every one the demographic and nutrition-related variables in the regression analysis model as independent variables. Then, the QUICKI level was included as the dependent variable, and the association of QUICKI level and independent variables (energy-adjusted nutrient intake, maternal characteristics, participants' weights, and physical activity scores) was analyzed by the regression analysis model. All mentioned variables were entered into the independent variable box to adjust for significant maternal characteristics. At the same time, the QUICKI amount was placed in the box of the dependent variable. We added each energy-adjusted nutrient intake one by one to the group of significant maternal characteristics in the relevant box, and the most associated energy-adjusted nutrient intake was determined.

Results

Table 1 presents the participants' characteristics at 6–10 weeks of gestation with a mean maternal weight and SI index of 23.59 ± 3.91 kg and 0.36 ± 0.03, respectively. The average weight gain after 14-12 weeks was 1.31 ± 2.50 kg. Among them, 50.36% had a bachelor's degree or higher, 60.3% were homemakers, and 82.61% had medium to high income levels. The average total and occupational physical activity were 30.81 ± 11.65 and 4.20 ± 6.30 met/hour, respectively. Maternal weight, pregravid BMI, total physical activity, job activity, and household income were associated with the QUICKI index (Table 1). After adjusting for significant maternal characteristics, participants' weights and household income were determined as the most significant variables (Table 1). QUICKI index was significantly correlated with energy-adjusted intakes of carbohydrates, vitamins E, D, and C, fat, polyunsaturated fatty acids (PUFAs), alpha-linolenic acid, and percentage of energy from carbohydrates (Table 2). We also found the QUICKI index had a significant relationship with energy-adjusted intakes of monounsaturated fatty acids (MUFAs), vitamins B1 and B3, beta-carotene, and lactose. After controlling for significant maternal characteristics, there was a significant inverse relationship between PUFA and MUFA intakes and the QUICKI index while total fat intake was positively correlated. Also, vitamin C, glucose, fructose, sugar, and carbohydrate intake increased the QUICKI index while vitamin E intake decreased it (Table 2). Also, the QUICKI index had a significant relationship with energy-adjusted alpha-linolenic acid, oleic fatty acid, selenium, and beta-carotene (Table 2).

Discussion

The results showed that the QUICKI index at 6-10 weeks of pregnancy was negatively associated with participants' household income, pregravid BMI, and weight. At the same time, total and occupational physical activity scores were positively correlated. Household income and weight were the more significant variables. Similarly, Bandres-Meriz et al showed that maternal obesity was inversely related to SI throughout the first trimester of pregnancy (13). The significant negative association between household income and QUICKI score aligns with the findings of Demir et al and Yang et al (14,15) while most studies report opposite findings (16). Lower physical activity scores and higher consumption of fast and processed foods are the probable reasons (14,17). In the present article, energy-adjusted MUFA and PUFA

Table 1. Participants' characteristics at 6–10 weeks of gestation and their association with QUIKI index

Demographic and family characteristics	Mean ± SD/No. (%)	β*	*P**	95% CI	β**	*P***	95% CI
Age (y),	26.52 ± 4.06	0.238	-0.001	-0.002, 0.001	0.000	0.835	-0.002, 0.001
Pregravid BMI (kg/m^2)	23.59 ± 3.91	-0.004	<0.001	-0.005, -0.003	-0.001	0.510	-0.003, 0.002
Weight gain in the first trimester (kg)	1.31 ± 2.50	-0.001	0.219	-0.003, 0.001	-0.001	0.486	-0.003, 0.001
Participants' weight in the first trimester (kg)	62.60 ± 11.02	-0.002	<0.001	-0.002, -0.001	-0.001	0.018	-0.002, 0.000
Total physical activity (met/hour	30.81 ± 11.65	0.001	0.001	0.000, 0.001	0.000	0.295	0.000, 0.001
Homemaker activity	9.87 ± 4.17	0.000	0.639	-0.001, 0.002	0.001	0.560	-0.001, 0.002
Personal activity	6.81 ± 4.00	0.001	0.450	-0.001, 0.002	-0.001	0.171	-0.002, 0.000
Occupational activity	3.82 ± 5.25	0.001	0.012	0.000, 0.002	0.001	0.193	0.000, 0.002
Sport activity	3.53 ± 2.69	0.001	0.518	-0.001, 0.003	0.000	0.882	-0.002, 0.002
Hobby activity	5.89 ± 3.75	0.001	0.195	-0.001, 0.003	0.001	0.351	-0.001, 0.002
Education		-0.004	0.178	-0.009, 0.002	0.001	0.735	-0.004, 0.006
High-school diploma or lower	54 (39.42)	-	-	-	-	-	-
Associate degree	14 (10.22)	-	-	-	-	-	-
Bachelor's degree	65 (47.44)	-	-	-	-	-	-
Master's degree and doctorate	4 (2.92)	-	-	-	-	-	-
Job		-0.005	0.207	-0.014, 0.003	-0.007	0.125	-0.015, 0.002
Homemaker	83 (60.3	-	-	-	-	-	-
Non-governmental jobs	38 (27.2)	-	-	-	-	-	-
Government jobs	17 (12.5)	-	-	-	-	-	-
Household income (Rials		-0.004	0.044	-0.009, 0.000	-0.004	0.042	-0.008, 0.000
<4000000	6 (4.35	-	-	-	-	-	-
4000000–6000000	18 (13.04	-	-	-	-	-	-
6000000–9000000	43 (31.16	-	-	-	-	-	-
9000000–12000000	41 (29.71)	-	-	-	-	-	-
>12000000	30 (21.74)	-	-	-	-	-	-

QUICKI index: quantitative insulin sensitivity check index.
**P*: The association of maternal characteristics with QUICKI index by regression analysis.
***P*: The association of maternal characteristics with QUICKI index after adjusting for significant maternal characteristics.

were inversely correlated with the QUICKI index, while the total fat intake was positively correlated. Researchers reported that enhanced accessibility of fatty acids is related to skeletal muscle IR, but the type of fatty acid is also significant. Of course, the studied whole-body alterations are unrelated to the changed skeletal muscle role and may be related to IR in numerous tissues (18). Saturated fats are less easily oxidized and stored as ceramides and diacylglycerols *in vitro*, while unsaturated fats are stored as intramyocellular triacylglycerols or free fatty acids. Epidemiological evidence in humans and in-vitro studies shows that saturated fatty acids induce IR. At the same time, unsaturated fats protect or even increase SI through increased integration into triacylglycerol and enhanced triacylglycerol storage. Diacylglycerol and ceramides restrict insulin signaling *in vitro* through novel PKC motivation (19), but some researchers have shown that ceramides might not develop IR *in vivo* (18). However, the positive significant association between fat intake and QUICKI score aligns with the findings of Noakes and Windt, who indicated that low-carbohydrate, high-fat diets might benefit patients with IR (20). Lipids have a central role in metabolic inflexibility and the associated IR, but some surveys have shown that lipids are metabolic substrates that might correct SI by modifying hepatic glucose production (21). Lipolysis could generate glycerol, delivered to the liver and metabolized to acetyl-CoA, increasing the enzyme activity to transfer pyruvate into gluconeogenic (21).

Higher MUFA and PUFA decreased the QUICKI index in the present study. Chen et al reported that PUFA increased glutathione peroxidase activity, which might reduce SI (22). Also, researchers revealed that MUFA and PUFA gather as intramyocellular triacylglycerol or free fatty acids, which might decrease SI by disrupting glucose uptake (18). The present study found a positive trend between oleic acid (the most usual dietary MUFA) and SI, which is consistent with other studies (18). Oleic acid reduced interleukin-1β production from adipose tissue cells, which in turn reduced SI (23). Also, a significant negative relationship between linoleic fats and the QUICKI index was observed. Likewise, another

Table 2. The association of energy- adjusted nutrients intake and QUICKI index before and after adjusting for significant maternal characteristics.

Daily nutrient intake*	Mean*	Standard deviation*	RAD amounts of nutrient intake	*P***	β**	95% CI for β	*P****	β***	95% CI for β
Energy (kcal)	1773.71	524.07	2400	-	-	-	-	-	-
Protein (g)	62.34	22.71	71	0.504	0.000	-0.001, 0.000	0.815	-0.000044	0.000, 0.000
Total fat (g)	53.47	26.93	65	0.013	0.000004	-0.001, 0.000	0.010	0.000	-0.001, 0.000
Saturated fat (g)	13.59	7.16	<20	0.285	-0.001	-0.002, 0.000	0.188	-0.001	-0.002, 0.000
Cholesterol (mg)	163.00	142.07	300	0.814	0.000005	0.000, 0.000	0.752	-0.000006	0.000, 0.000
Polyunsaturated fatty acid (g)	12.89	9.04	6	0.023	-0.001	-0.001, 0.000	0.010	-0.001	-0.001, 0.000
Monounsaturated fatty acid (g)	15.62	9.77	-	0.072	-0.001	-0.001, 0.000	0.043	-0.001	-0.001, 0.000
Linoleic fatty acid (g)	9.35	8.39	13	0.122	-0.001	-0.001, 0.000	0.082	-0.001	-0.001, 0.000
Oleic acid (mg)	13.15	9.84	20	0.165	0.000	-0.001, 0.000	0.074	-0.001	-0.001, 0.000
alpha-linolenic (g)	0.14	0.11	2	0.010	-0.067	-0.118, -0.017	0.221	-0.030	-0.077, 0.018
DHA (mg)	0.41	0.12	-	0.567	-0.015	-0.067, 0.037	0.924	0.002	-0.043, 0.047
Vitamin A (mcg)	810.66	944.93	770	0.179	-0.000004	0.000, 0.000	0.219	-0.000003	0.000, 0.000
Vitamin E (mg)	3.75	3.61	15	0.002	-0.003	-0.004, -0.001	0.007	-0.002	-0.003, -0.001
Thiamine B1(mg)	1.63	0.69	1.4	0.058	0.021	0.000, 0.025	0.946	0.001	-0.012, 0.013
Vitamin B3(mg)	17.64	8.01	18	0.086	0.001	0.000, 0.002	0.973	0.000014	-0.001, 0.001
Folic acid (mcg)	204.80	121.87	600	0.239	0.000030	0.000, 0.000	0.610	0.000014	0.001, 0.000
Pantothenic acid (mg)	4.13	3.27	6	0.807	0.000	-0.001, 0.002	0.663	0.000	-0.001, 0.002
Vitamin C (mg)	99.36	66.46	85	0.019	0.000	0.000, 0.000	0.008	0.00100	0.000, 0.000
Vitamin K (mcg)	85.32	76.65	75–90	0.956	0.000002	0.000, 0.000	0.663	-0.000015	0.000, 0.000
Carbohydrate (g)	260.75	90.78	175	0.014	0.000	0.000, 0.000	0.010	0.000	0.000, 0.000
Glucose (g)	12.48	8.36	-	0.195	0.000	0.000, 0.001	0.003	0.001	0.000, 0.001
Fructose (g)	17.90	12.28	55	0.320	0.000	0.000, 0.001	0.004	0.001	0.000, 0.001
lactose (g)	5.78	5.92	<12	0.084	-0.001	-0.002, 0.000	0.678	0.000	-0.001, 0.001
Iron (mg)	16.76	6.78	27	0.541	-0.000043	0.000, 0.000	0.406	0.000051	0.001, 0.001
Magnesium (mg)	253.37	183.92	350–400	0.574	-0.000014	0.000, 0.000	0.617	0.000011	0.000, 0.000
Manganese (mg)	2.99	2.48	2	0.123	0.002	-0.001, 0.004	0.483	0.001	-0.001, 0.003
Zinc (mg)	6.23	2.90	11–12	0.929	0.000	-0.002, 0.003	0.836	0.000	-0.003, 0.002
Sodium (g)	1131.51	682.88	1.5	0.433	0.000004	0.000, 0.000	0.548	0.000003	0.000, 0.000
Potassium (mg)	2353.35	1070.27	2000	0.691	-0.000001	0.000, 0.000	0.355	0.000003	0.000, 0.000
Calcium mg	617.67	312.09	1000	0.124	-0.000016	0.000, 0.000	0.918	-0.000001	0.000, 0.000
Phosphorous (g)	849.62	403.48	700	0.188	-0.000012	0.000, 0.000	0.851	-0.000002	0.000, 0.000
Copper (mg)	1.36	1.22	1	0.754	0.001	-0.004, 0.005	0.989	0.000026	-0.004, 0.004
Selenium (mcg)	0.07	0.04	60	0.106	-0.147	-0.325, 0.032	0.073	-0.158	-0.332, 0.015
Chromium (mcg)	0.04	0.03	30	0.402	-0.099	-0.331, 0.134	0.554	0.063	-1.148, 0.274
Molybdenum (mcg)	22.80	27.27	50	0.711	-0.000038	0.000, 0.000	0.436	0.0000073	0.000, 0.000
Beta-carotene (mcg)	506.76	986.48	770	0.061	-0.000005	0.000, 0.000	0.075	-0.000004	0.000, 0.000
Alpha-tocopherol (mg)	4.19	4.91	15 mg (22.5 IU)	0.692	0.000	-0.001, 0.001	0.286	-0.001	-0.002, 0.001
VitaminB2 (mg)	1.54	1.59	1.4	0.845	0.000	-0.004, 0.003	0.420	0.001	-0.002 0.004
Vitamin B6 (mg)	1.39	0.71	1.9	0.115	-0.008	-0.018, 0.002	0.114	-0.009	-0.021, 0.002
Vitamin B12 (mcg)	3.12	4.06	2.6	0.192	-0.001	-0.002, 0.000	0.206	-0.009	-0.002, 0.000
Vitamin B8 (mcg)	18.73	14.04	15	0.364	0.000	-0.001, 0.000	0.722	0.000071	0.000, 0.000
Biotin (mcg)	19.50	16.76	30	0.626	-0.000089	0.000, 0.000	0.678	0.000064	0.000, 0.000
Vitamin D (mcg)	1.27	3.46	15	0.011	-0.005	-0.008, -0.001	0.751	0.001	-0.003, 0.004
Fiber (g)	16.32	8.96	28	0.595	0.000	-0.001, 0.001	0.565	0.000	-0.001, 0.001
Sugar (gm)	77.58	46.13	25	0.679	0.000028	0.000, 0.000	0.015	0.000	0.000, 0.000
Galactose (g)	1.02	1.54	3.1	0.309	0.002	-0.002, 0.006	0.137	0.002	-0.001, 0.006

Table 2. Continued.

Daily nutrient intake*	Mean*	Standard deviation*	RAD amounts of nutrient intake	*P***	β**	95% CI for β	*P****	β***	95% CI for β
Caffeine (mg)	33.01	31.60	400	0.928	-0.000008	0.000, 0.000	0.261	0.000	0.000, 0.000
% Energy from proteins	13.19	4.60	12–20	0.282	0.001	-0.001, 0.002	0.616	0.000	-0.001, 0.002
% Energy from carbohydrates	54.71	16.75	50–60	0.021	0.000	0.000, 0.001	0.104	0.000	0.000, 0.001
% Energy from fats	24.95	10.76	30	0.312	0.000	-0.001, 0.000	0.271	0.000	-0.001, 0.000

QUICKI index: quantitative insulin sensitivity check index.
* Mean and Std. deviation of un-adjusted nutrient intake.
***P*: The association of energy-adjusted nutrient intake with QUICKI index by regression analysis.
****P*: The association of energy-adjusted nutrient intake with QUICKI index after moderating for significant maternal characteristics.

study demonstrated that lipid emulsion infusion included linoleate, motivated ceramide production, and human insulin resistance (24).

We found that higher vitamin C intake increased the QUICKI index. In contrast, other studies reported that vitamin C, either alone or in combination with other antioxidants, could not reduce IR in diabetics (25,26). Antioxidant vitamins like vitamin C improve endothelial function and protect biomembranes as opposed to lipid peroxidation, and clinical trials with antioxidants showed better results in prediabetes. Vitamin C can reduce free radicals, decrease protein glycosylation *in vitro* and *in vivo*, and enhance SI by changing the endothelial function and reducing oxidative stress (27). Also, for every one unit increase in vitamin E, SI decreased by 0.003 unit. Although it is an antioxidant, researchers have recently reported that higher-than-normal doses of vitamin E and beta-carotene, a precursor to vitamin A, have oxidizing effects (28). This is our only explanation for the link between the increase in vitamin E and increased SI. Our results showed that fructose, glucose, sugar, and carbohydrates enhanced SI. Also, Ahmadi-Abhari et al reported that greater intakes of glucose and fructose were related to a lower risk of type 2 diabetes due to intakes of glucose and fructose by themselves or with other nutrients that may accompany them (29). Vitamin C and glucose are similar in chemical construction and could compete with each other (27). Lecoultre et al showed that short-term hypercaloric intake of simple sugars could control hepatic SI and the long-term effects should be further evaluated (30). Oral glucose induces physiological responses by increasing glucose turnover and removing dietary lipids, which causes glucose to enter the bloodstream from the hepatic pathway. Johnson et al showed that a portal infusion and not a systemic infusion improved SI, and they believed glucose could mediate the lipid-induced SI through a hepatoportal mechanism (31). This is the first survey to assess the correlations of all nutrients and physical activity scores with the SI index in the first trimester of gestation. It is also possible that our findings can be used to determine the reference range of IR, all different nutrients intake, and physical activity scores in early pregnancy, which can be used in other studies. However, this cross-sectional survey could not clarify causal relationships between the studied variables. Therefore, further prospective studies are suggested.

Conclusion

The results showed that weight management may prevent GDM during the first trimester. Moreover, the significant correlation between the abovementioned nutrients and household income with the QUICKI index can be further assessed in future studies.

Acknowledgements

We are grateful to the School of Public Health of Shahid Sadoughi University of Medical Sciences for providing financial support for this study (grant No. 4326).

Authors' Contribution

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Competing Interests

The authors declared no conflict of interest.

Ethical Approval

Ethics Committee of the School of Public Health at Shahid Sadoughi University of Yazd issued a code of ethics (IR.SSU.SPH.REC.1395.13).

Funding

This study was supported by the School of Public Health of Shahid Sadoughi University of Medical Sciences (grant No. 4326).

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