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ORIGINAL RESEARCH

Quinoa Improves Glucose Metabolism and Fluctuation Based on β-Cell Function in Early-Stage Type 2 Diabetes Mellitus: A Randomized Study

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Video abstract

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Introduction: Clinical studies are yet to determine whether quinoa improves metabolism in patients with type 2 diabetes mellitus (T2DM).

Methods: Sixty patients with early-stage T2DM were randomly assigned to the quinoa diet (QD) or regular diet (RD) groups. Oral glucose tolerance tests (OGTTs) and dynamic blood glucose monitoring were performed before and after the intervention.

Results: Ninety-seven percent of patients adhered to the dietary intervention plan involving quinoa. In addition to postprandial blood glucose and cholesterol, fasting blood glucose, glycosylated haemoglobin, triglycerides, and insulin resistance decreased in the QD group. The reductions in postprandial blood glucose (4.57 ± 2.7 mmol/L vs 2.2 ± 2.8 mmol/L, P <0.001) and glycosylated haemoglobin (0.86 ± 0.24 vs 0.28 ± 0.23, P =0.042) as well as the increase in the time in range (94.52 ± 4.88% vs 87.83 ± 9.9%, P <0.05) were more pronounced in the QD group. The QD group achieved a higher rate of medication reduction or withdrawal (28.6% vs 7.1%, P =0.036). Multivariate analysis indicated that HOMA-β and quinoa consumption were the primary risk factors.

Conclusion: β-cell function is the key factor influencing quinoa's ability to improve metabolism and achieve diabetes remission in patients with early-stage T2DM.

Keywords: type 2 diabetes mellitus, quinoa dietary intervention, glycolipid metabolism, glycaemic fluctuations

Introduction

Medical nutrition therapy is a cornerstone of type 2 diabetes mellitus (T2DM) management.[1] A previous network meta-analysis of randomized trials indicated that ketogenic, Mediterranean, moderate-carbohydrate, and low glycaemic index (GI) diets can effectively achieve glycaemic control in patients with T2DM.[2] While dietary approaches offer clear benefits, adhering to them over time can be challenging because of factors such as cost, accessibility, and difficulty in changing long-standing dietary habits.[3] Therefore, dietary advice should be tailored to account for personal, cultural, and social factors.[4]

Among the 20 most populous countries, China has the highest mortality rates from cardiovascular diseases (57.99%) and cancer (15.32%), with unhealthy diet being a major contributing factor.[5] A previous international prospective cohort

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study revealed that Chinese food culture is characterized by a high intake of starchy foods, resulting in diets with the highest GI.[6] This dietary pattern may contribute to the extremely low achievement rate of diabetes care goals recommended by guidelines among self-reported adult diabetic patients in China.[7] However, the low-GI/low-glycaemic-load (GL) diet model is highly useful for the management of metabolic indicators in patients with T2DM and has led to important improvements in the established goals of glucose control, blood lipids, obesity, pressure, and inflammation, especially in adults with moderate control of T2DM, beyond simultaneous treatment with hyperglycaemic drugs or insulin.[8]

Quinoa is a low-GI food with unique nutritional value.[9] The bioactive compounds extracted from quinoa provide a range of health benefits, including antioxidative, anti-inflammatory, and antimicrobial effects; improvements in glucose and lipid metabolism; regulation of the gut microbiota; and potential anticancer effects.[10–13] Previous meta-analyses conducted among nondiabetic individuals have indicated that eating quinoa can reduce cardiovascular risk factors, such as weight, waist circumference, fasting insulin levels, and blood lipid levels.[14–16] While the metabolic benefits of low-GI foods are well established, their application within traditional Chinese dietary patterns remains underexplored.

This study aimed to evaluate the feasibility and efficacy of incorporating quinoa-based products into the Chinese dietary context, with a particular focus on β-cell function as a potential predictor of response. Additionally, we aimed to assess the effects of this intervention on overall metabolism and glucose fluctuations. We hypothesized that quinoa-based dietary intervention would improve glucose metabolism and reduce glycaemic fluctuations in patients with early-stage T2DM and that these benefits might be achieved by improving β-cell function.

Methods

Study Population

We recruited patients with early-stage T2DM from the Qilu Hospital of Shandong University. The inclusion criteria were as follows: (1) patients diagnosed according to the 1999 WHO diagnostic criteria for T2DM with a duration of less than 5 years; (2) HbA_{1c} levels between 6.5% and 10%; (3) no history of insulin therapy; and (4) age between 20 and 70 years. The exclusion criteria were as follows: (1) acute complications of diabetes, such as diabetic ketoacidosis; (2) severe acute or chronic conditions affecting the liver, kidneys, heart, cerebrovascular system, gastrointestinal tract, or acute infectious diseases; (3) pregnancy or plans for pregnancy soon; (4) allergies to quinoa or bean products; (5) participation in other dietary interventions or drug clinical trials within the past three months; and (6) gastrointestinal diseases within two weeks prior to enrolment.

Ultimately, 60 patients with early-stage T2DM were included in the study. The random number table method was used to allocate 30 participants to the quinoa diet (QD) group and 30 participants to the regular diet (RD) group. RD refers to the standard diabetes diet that follows the guidelines of the China Diabetes Association. The distribution of energy and macronutrients matched that of the QD group, but conventional staple foods (white rice and wheat flour products) were used. All participants provided written informed consent prior to enrolment. The procedures complied with the ethical standards established by the Committee Responsible for Human Experimentation and the 1975 Declaration of Helsinki. The Ethics Committee of Shandong University, Qilu Hospital, approved this study (KYLL-2019(ZM)-062). Trial Registration: clinicaltrials.gov Identifier: NCT06156137 (Registered November 24, 2023).

Sample Size Calculation

The sample size was calculated using the formula for comparing the means of two independent samples on the basis of the expected difference in blood glucose variation ($\delta = 1.2$ mmol/L), combined standard deviation ($\sigma = 1.5$), significance level ($\alpha = 0.05$, two-tailed), and statistical power ($1 - \beta = 80\%$). The calculations indicated that each group required at least 24 participants, resulting in a total sample size of 48. To account for potential dropout or data variability, the final sample size was set at 30 participants per group to ensure sufficient statistical power.

Research Design

After enrolment, a trained physician provided education on diet and exercise for diabetes. All participants then completed a 75-g oral glucose tolerance test (OGTT) and other baseline assessments within one week. At the beginning of the first week (W1), all patients underwent continuous glucose monitoring (cGMS; FreeStyle Libre Flash glucose monitoring system; Abbott Diabetes Care Ltd).

After one week, the daily caloric requirement from starchy foods was calculated on the basis of the standard body weight. The calculations were performed according to the Chinese Type 2 Diabetes Prevention and Treatment Guidelines (2024 edition) and the American Diabetes Association's Standards of Care in Diabetes—2024, which recommend that starchy foods (carbohydrates) should contribute 45%-60% of the total daily energy intake for adults with diabetes, with fat comprising 20%-30% and protein 15%-20%. For individuals with moderate physical activity levels, the recommended total energy intake was set at 35 kcal per kilogram of body weight per day. The patients were provided with a one-week supply of quinoa and a QD record book to document their daily quinoa intake. After two weeks (W2), all patients were followed up in person, and the cGMS data were downloaded. Following this one-week run-in period, the intervention group received a monthly supply of quinoa-based products for consumption. Follow-ups were subsequently conducted once a month, during which time food packaging was recycled, new food was distributed, and adherence to the exercise prescription was assessed. At the 13th week (W13), all the patients underwent cGMS. At the beginning of the 14th week (W14), the intervention group ceased quinoa consumption, and at the end of the 14th week, all patients were once again administered an OGTT.

All participants were provided with a fixed menu comprising 5–7 options for breakfast, lunch, and dinner, along with a selection of 10–11 snack items. They were instructed to prepare their meals at home and adhere strictly to the specific foods and weights listed on their assigned menu. The QD group consumed quinoa-based foods as substitutes for traditional staples, whereas the RD group continued with traditional staples. All participants were required to prepare meals at home and strictly abide by the specific types and quantities of food listed on the designated menu. A list of prohibited foods was supplied, which included commercial baked goods, sweets, pastries, fried foods and snacks, fatty or processed meats, and high-fat dairy products. Additionally, patients were advised to engage in at least 150 to 300 minutes of moderate-intensity aerobic exercise each week. Follow-up assessments were conducted biweekly through telephone calls or clinic visits to collect a comprehensive record of dietary intake, allowing for the calculation of the average daily consumption of protein, fat, and carbohydrates.

Study Diets

The quinoa-based food products provided to the QD group contained 15–20% pure quinoa, with the remainder consisting of other nutrient-dense ingredients (eg, whole grains, legumes), as detailed below: quinoa noodles (80 g per bag; the ingredients included quinoa powder, red bean powder, black wheat powder, black mung bean powder, and edible salt, with quinoa powder comprising 15% of the total produced by Shandong Tangren Duoyi Biotechnology Company); blended powder (15 g per bag; the ingredients included quinoa, rye wheat, black mung beans, adzuki beans, black sesame seeds, chickpeas, konjac flour, and mogroside, with quinoa flour comprising 20% of the total produced by Shandong Tangren Duoyi Biotechnology Company); and cookies (24 g per bag; quinoa, rye, adzuki beans, black sesame seeds, vegetable oil, and salt, with quinoa comprising 20% of the total produced by Shandong Tangren Duoyi Biotechnology Company). Every 100 g of noodles contained 1369 kJ of energy, 18.9 g of protein, 0 g of fat, 58.4 g of carbohydrates (including 4.0 g of sugar and 6.9 g of dietary fibre), and 639 mg of sodium. Every 100 g of blended powder contained 1681 kJ of energy, 18.5 g of protein, 5.6 g of fat, 68.2 g of carbohydrates, and 0 mg of sodium. Each 100 g cookies contained 2140 kJ of energy, 9.0 g of protein, 24.1 g of fat (including 10.7 g of saturated fatty acids and 0 g of trans fatty acids), 64.4 g of carbohydrates (0 g of sugar), and 186 mg of sodium.

Data Collection

At W0, data on the patients' sex, age, height, weight, body mass index (BMI), medical history (including hypertension (HP), hyperlipidaemia, and other conditions), and use of oral antidiabetic drugs (OADs) were collected. An OGTT was conducted to measure blood glucose and insulin levels at 0 hours (FBG, FINS), 0.5 hours (BG0.5h, INS0.5h), and

2 hours (BG2h, INS2h), as well as the area under the glucose curve (AUC). In addition, fasting total cholesterol (TC), triglyceride (TG), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), glucagon-like peptide-1 (GLP-1), haemoglobin A_{1c} (HbA_{1c}), and uric acid (UA) levels were assessed. Changes (Δ) in outcome measures from baseline to follow-up were calculated. β-cell function (HOMA-β) and insulin resistance (HOMA-IR) were assessed using the homeostasis model assessment. The hypoglycaemic medications were either reduced or discontinued. The coefficients of variation (CV), time in range (TIR), time above range (TAR), and time below range (TBR) for W1, W2, W13, and W14 were calculated on the basis of cGMS data. Furthermore, the total consumption of quinoa products in the QD group was determined using patients' quinoa food record books.

Study Endpoints

The primary endpoints were the degree of blood glucose reduction and stabilization of glucose levels. The secondary endpoints included body weight, blood lipid levels, HOMA-β, improvements in insulin resistance, the number of patients for whom hypoglycaemic medications were reduced or discontinued, and factors associated with the reduction or discontinuation of hypoglycaemic drugs.

Statistical Analysis

SPSS version 19.0 was used for statistical analysis. Descriptive statistics are presented as the mean ± standard deviation ($\bar{X} \pm s$). The Shapiro–Wilk test was used to assess normality, and Levene's test was used to evaluate the homogeneity of variance. For variables that followed a normal distribution, an independent samples *t* test was used to compare the differences. In cases of unequal variances, the Mann–Whitney *U*-test was used to compare differences between the two groups. The chi-square test and Fisher's exact test were used to assess differences in the proportions of categorical variables. Univariate logistic regression was performed to identify covariates for inclusion in the multivariate analysis. Variables with a significance level of $P < 0.05$ in the univariate analysis were subsequently entered into the multivariate analysis as covariates. Multivariate logistic regression analysis was conducted to identify factors associated with the reduction or cessation of hypoglycaemic medications.

Results

Baseline Characteristics of the Participants

Four patients from the QD and RD groups were excluded from the study. One patient in the QD group discontinued due to an inability to adhere to the diet. Additionally, three patients (one in the QD group and two in the RD group) were lost to follow-up. Ultimately, 56 patients were included in the final analysis (Figure 1). There were no significant differences in age between the two groups, nor were there significant differences in baseline or postintervention measurements of weight, BMI, complications, OADS, BG, INS, HbA_{1c}, HOMA-β, HOMA-IR, blood lipids, UA, GLP-1, or energy intake among the other variables (Table 1).

The average daily consumption of blended powder, biscuits, and noodles was 0.61 bags, 2.6 bags, and 0.67 bags, respectively, corresponding to weights of 9.15 g, 62.26 g, and 53.33 g, respectively. Based on the quinoa content, the daily intake of pure quinoa was 22.29 g. All patients followed the doctor's exercise prescription weekly, adhering closely to the recommended amount. Comparisons of the prespecified dietary targets revealed that the two groups had comparable daily energy intake and similar energy distributions from protein, fat, and carbohydrates (Table 1).

Main Outcomes

After the intervention, the FBG, BG2h, and HbA1c levels in the QD group decreased significantly from 7.70 ± 2.0 mmol/L, 15.48 ± 4.43 mmol/L and 6.88 ± 1.46% to 6.62 ± 0.88 mmol/L (P =0.007), 12.62 ± 4.42 mmol/L and 6.02 ± 0.61% (P =0.001), respectively. Only BG2h (14.46 ± 3.09 mmol/L vs 12.26 ± 2.92 mmol/L, P =0.002) was significantly reduced in the RD group, whereas FBG and HbA_{1c} levels did not change significantly. The ΔBG2h (4.57 ± 2.7 mmol/L vs 2.2 ± 2.8 mmol/L, P <0.001) and ΔHbA_{1c} (0.86 ± 0.24 vs 0.28 ± 0.23, P =0.042) in the QD group were significantly greater than those in the RD group (Table 2, Figure 2). The AUCs of the OGTT blood glucose in both the QD group

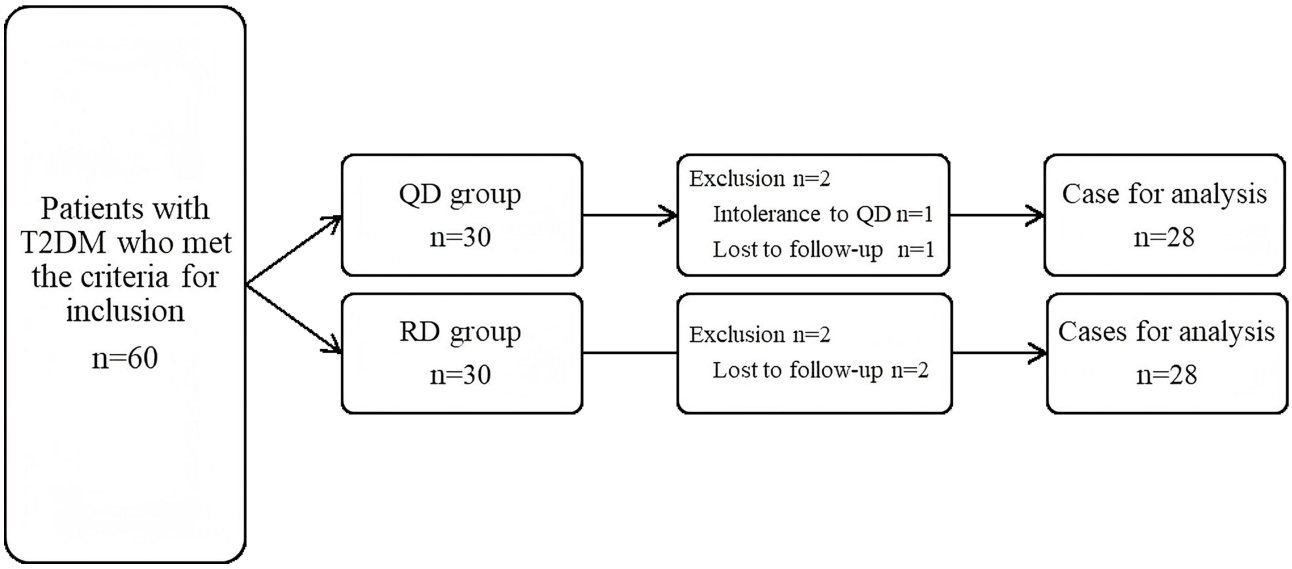

Figure 1 Flowchart of the QD and RD patient enrolment.
Abbreviations: QD, quinoa diet; RD, regular diet.

(26.35 ± 5.83 vs 22.52 ± 4.70, P =0.009) and the RD group (25.36 ± 4.32 vs 23.05 ± 4.05, P =0.044) decreased significantly after the intervention (Table 2 and Figure 3).

The TIR in the QD group was significantly greater at W13 and W14 than at W1. Moreover, TAR decreased significantly at W2, W13, and W14, whereas TBR did not change significantly. In the RD group, TIR was significantly lower at W13 and W14 than at W1. TAR was significantly greater at W2 and W13 than at W1, and TBR was significantly greater at W13 and W14 than at W1. Compared with the patients in the RD group, patients in the QD group had significantly greater TIR at W13 and W14 (Table 3 and Figure 2).

Secondary Outcomes

After adhering to the quinoa diet in the QD group, body weight decreased significantly from 72.59 ± 11.55 kg to 71.46 ± 10.28 kg (P =0.006). Additionally, the TG concentration decreased from 1.74 ± 1.02 mmol/L to 1.29 ± 0.58 mmol/L (P =0.007), and the TC concentration decreased from 4.74 ± 1.13 mmol/L to 4.33 ± 0.86 mmol/L (P = 0.012). Moreover, the HOMA-IR in QD patients decreased significantly from 0.45 ± 0.22 to 0.33 ± 0.18 (P =0.013). In the RD group, the TC concentration decreased from 4.95 ± 0.88 mmol/L to 4.39 ± 0.94 mmol/L (P =0.018), but the HOMA-IR score did not significantly improve. Eight patients in the QD group achieved drug reduction or discontinuation, which was significantly greater than that of two patients in the RD group (28.6% vs 7.1%, P =0.036) (Table 4).

Table 1 Baseline Characteristics of the Two Groups (n (%), $\bar{X}$ ± s)

Variables	QD	RD	$t/Z/\chi^2$	P
Sex (Man)	71.4%	60.7%	0.529	0.573
Age (Yeas)	53.57 ± 14.74	52.96 ± 11.29	0.173	0.863
Medical History (%)				
HP	21.4	25	0.100	1.000
Hyperlipidaemia	25	25	0.000	1.000
Others	17.9	17.9	0.000	1.000

(*Continued*)

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Table 1 (Continued).

Variables	QD	RD	$t/Z/\chi^2$	P
OADS (%)				
Metformin	53.6	75	2.800	0.162
AGI	46.4	46.4	0.000	1.000
Secretagogue	25	10.7	1.948	0.295
DPP-4 inhibitor	71.4	64.3	0.327	0.775
SGLT-2 inhibitor	3.6	3.6	0.000	1.000
Weight (kg)	72.59 ± 11.55	73.57 ± 12.40	0.037	0.76
BMI (kg/m^2)	24.69 ± 2.85	25.40 ± 3.59	0.945	0.349
FBG (mmol/L)	7.70 ± 2.0	7.20 ± 1.53	−1.048	0.299
BG0.5h (mmol/L)	12.82 ± 3.00	112.58 ± 2.36	−0.329	0.743
BG2h (mmol/L)	15.48 ± 4.43	14.46 ± 3.09	−0.998	0.328
FINS (mIU/L)	8.72 ± 4.46	9.03 ± 5.01	0.329	0.743
INS0.5h (mIU/L)	27.78 ± 18.67	35.61 ± 18.95	0.824	0.414
INS2h (mIU/L)	56.69 ± 55.07	52.96 ± 51.18	−0.308	0.759
HbA_1c (%)	6.88 ± 1.46	6.49 ± 1.19	−1.135	0.262
GLP-1 (pmol/L)	2.02 ± 0.60	1.93 ± 0.78	−0.501	0.618
HOMA-β	1.58 ± 0.34	1.63 ± 0.35	0.518	0.606
HOMA-IR	0.45 ± 0.22	0.45 ± 0.23	−0.088	0.931
TC (mmol/L)	4.74 ± 1.13	4.95 ± 0.88	0.760	0.450
TG (mmol/L)	1.74 ± 1.02	1.61 ± 0.93	−0.457	0.650
LDL-C (mmol/L)	2.55 ± 0.89	2.66 ± 0.75	0.488	0.628
HDL-C (mmol/L)	1.37 ± 0.51	1.31 ± 0.33	0.938	0.353
UA (μmol/L)	339.76 ± 65.22	344.15 ± 89.69	0.200	0.842
CV1	0.24 ± 0.06	0.23 ± 0.05	0.879	0.387
TIR1 (%)	89.69 ± 7.25	90.46 ± 9.07	0.595	0.555
TAR1 (%)	6.98 (1.13, 10.42)	2.58 (0.72, 3.91)	0.983	0.330
TBR1 (%)	0.15 (0.0, 3.27)	0.52 (0.05, 2.07)	1.279	0.204
Noodles (g/day)	53.33	–	–	–
Powder (g/day)	9.15	–	–	–
Cookies (g/day)	62.29	–	–	–
Intake				
Energy, kcal/d	2090±293.4	2107±326.7	0.202	0.841
Protein, %	16.56±2.98	17.54±3.86	1.059	0.295
Fat, %	35.08±2.93	35.65±3.14	0.725	0.472
Carbohydrate, %	48.36±4.31	46.88±4.06	1.324	0.191

Abbreviations: QD, quinoa diet; RD, regular diet; HP, hypertension; CHD, coronary heart disease; OADS, oral antidiabetic drugs; AGI, α-glucosidase inhibitor; DPP-4, dipeptidyl peptidase-4; SGLT-2, sodium–glucose cotransporter 2; BMI, body mass index; FBG, fasting blood glucose; Δ, absolute value of the difference between the numerical values before and after; INS, insulin; $HbA_{1c,}$ glycated hemoglobin A1c; TC, total cholesterol; TG, triglycerides; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; GLP-1, glucagon-like peptide-1; UA, uric acid; HOMA-β, β-cell function was assessed using the Homeostasis Model Assessment; HOMA-IR, insulin resistance was assessed using the Homeostasis Model Assessment; CV1, coefficients of variation for W1; TIR1, time in range for W1; TAR1, time above range for W1; TBR1, time below range for W1.

Correlation Analysis of Drug Consumption Reduction or Withdrawal

Univariate analysis included patient sex, height, weight before the intervention, blood glucose, INS, blood lipid, HbA_{1c}, HOMA-β, HOMA-IR, and quinoa consumption. The analysis revealed that age, male sex, disease duration, glycosylated haemoglobin levels before the intervention, HOMA-β, HOMA-IR, and quinoa consumption were associated with a reduction in or cessation of drug use. Multivariate analysis indicated that the preintervention HOMA-β (95% CI:

Table 2 Comparison of Glucose Metabolism Indices Between the Two Groups Before and After Intervention($\overline{X} \pm s$)

Variables	QD				RD				QD Post or Δ vs RD Post or Δ	
	Pre	Post or Δ	t	P	Pre	Post or Δ	t	P	t	P
FBG (mmol/L)	7.70±2.0	6.62±0.88	2.093	0.007	7.20±1.53	6.66±1.17	1.625	0.116	0.168	0.867
ΔFBG (mmol/L)	–	1.35±0.34	–	–	–	1.26±0.25	–	–	0.261	0.796
BG0.5h (mmol/L)	12.82±3.00	11.40±2.01	1.972	0.060	112.58±2.36	12.20±2.34	0.637	0.530	1.340	0.186
ΔBG0.5h (mmol/L)		2.51±0.45			2.32±0.48		–	–	0.311	0.758
BG2h (mmol/L)	15.48±4.43	12.62±4.42	3.070	0.005	14.46±3.09	12.26±2.92	3.414	0.002	0.361	0.719
ΔBG2h (mmol/L)	–	4.57±2.7	–		–	2.20±2.80	–	–	4.288	<0.001
HbA_1c (%)	6.88±1.46	6.02±0.61	3.597	0.001	6.49±1.19	6.20±0.61	1.208	0.238	1.094	0.279
ΔHbA_1c (%)	–	0.86±0.24			–	0.28±0.23	–	–	2.140	0.042
AUC	26.35±5.83	22.52±4.70	2.722	0.009	25.36±4.32	23.05±4.05	2.060	0.044	–	–
ΔAUC	–	3.98 ± 3.32	–	–		5.77 ± 4.74	–	–	1.628	0.109

Abbreviations: QD, quinoa diet; RD, regular diet; BMI, body mass index; BG, blood glucose; FBG, fasting blood glucose; Δ, absolute value of the difference between the numerical values before and after; INS, insulin; HbA1c, glycated hemoglobin A1c; AUC, area under the glucose curve.

1.013–1.132; P =0.016) and quinoa consumption (95% CI: 1.002–1.017; P =0.016) were significant factors influencing patients' ability to reduce or discontinue medication use (Figure 4).

Discussion

Our findings contribute to the literature by demonstrating the feasibility of quinoa-based dietary interventions within Chinese dietary culture. Compared with RD, QD significantly reduced BG2h and HbA1c levels and better stabilized blood glucose (reduced glycaemic variability). QD intervention was associated with improved insulin resistance, which was potentially mediated by its beneficial effects on blood lipids and body weight. QD intervention enabled a reduction, and in some cases cessation, in hypoglycaemic medication use. Multivariate analysis revealed that HOMA-β and quinoa consumption were the main factors affecting the reduction in drug use. Notably, the quinoa group received products containing 15–20% quinoa along with other ingredients, and both groups received comprehensive dietary counselling. Therefore, the observed benefits should be attributed to overall dietary intervention rather than quinoa alone. The

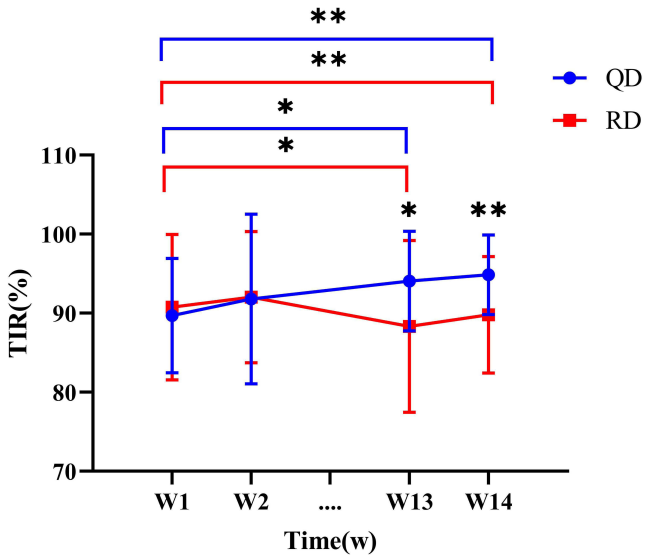

Figure 2 Trend of TIR change between the two groups. *, P <0.05; **, P <0.01.
Abbreviations: QD, quinoa diet; RD, regular diet; TIR, time in range.

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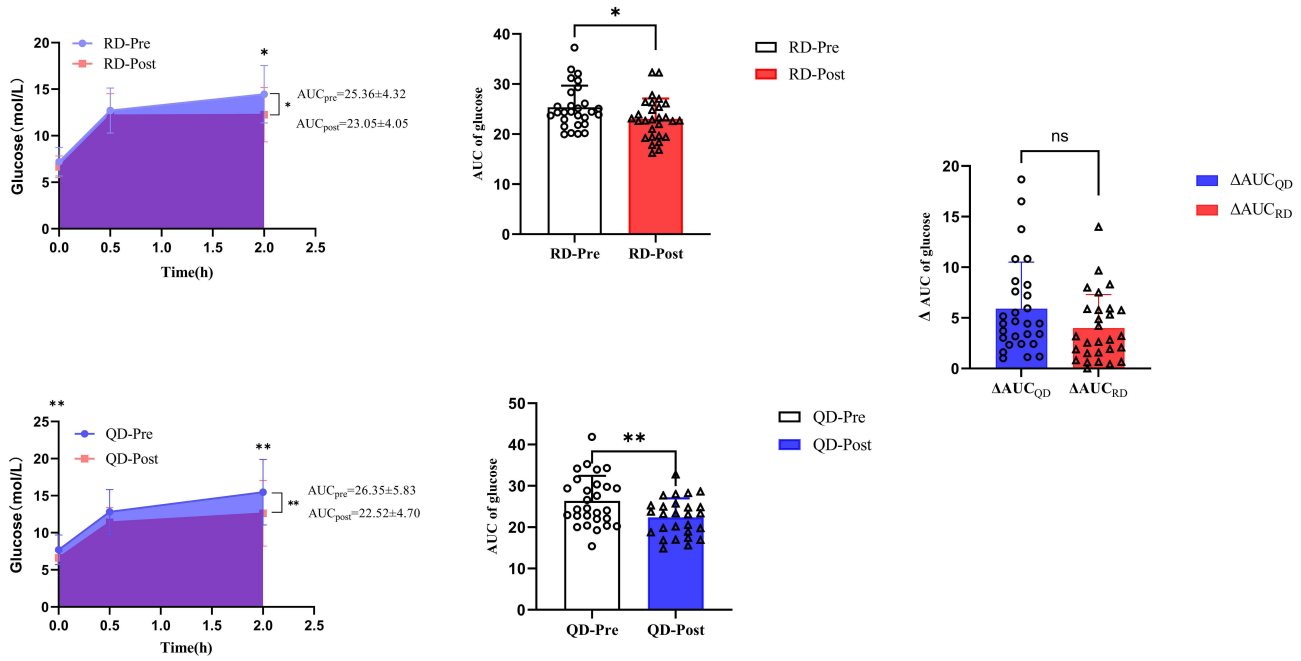

Figure 3 Comparison of OGTT blood glucose and the area under the curve between the two groups. *, *P* <0.05; **, *P* <0.01; ns, *P* >0.05.
Abbreviations: QD, quinoa diet; RD, regular diet; AUC, area under the glucose curve.

incremental benefits in the QD group likely reflect the combined effect of incorporating quinoa into a structured meal plan.

Dietary intervention plays a critical role in the remission of T2DM. Various dietary patterns—such as caloric restriction,[17,18] macronutrient-limiting diets,[19] intermittent fasting,[20] and specialized dietary regimens[21]—have

Table 3 Comparison of Blood Glucose Fluctuation Indices within the Two Groups and Between the Groups ($\overline{X} \pm s$), *Q (Q1, Q3)*)

Variables	QD (n = 28)	*t/Z*	*P*	RD (n = 28)	*t/Z*	*P*	QD vs RD	
							t/Z	*P*
CV1	0.24 ± 0.06	–	–	0.23 ± 0.05	–	–	0.879	0.387
CV2	0.22 ± 0.06	−1.587	0.124	0.21 ± 0.05	2.274	0.031	−0.411	0.684
CV13	0.24 ± 0.07	−0.199	0.844	0.25 ± 0.05	−1.307	0.202	−1.395	0.175
CV14	0.23 ± 0.07	−0.565	0.577	0.25 ± 0.06	−1.269	0.215	−1.047	0.305
TAR1 (%)	6.98 (1.13, 10.42)	–	–	2.58 (0.72, 3.91)	–	–	0.983	0.330
TAR2 (%)	1.28 (0.0, 5.41)	−3.594	<0.001	0.56 (0.0, 1.45)	−3.108	0.001	0.183	0.859
TAR13 (%)	0.36 (0.0, 2.47)	−2.248	0.024	6.03 (1.43, 11.33)	−2.781	0.004	1.520	0.130
TAR14 (%)	1.44 (0.0, 4.57)	−3.314	<0.001	4.04 (0.0, 13.19)	−1.951	0.052	1.299	0.197
TIR1 (%)	89.69 ± 7.25	–	–	90.46 ± 9.07	–	–	0.595	0.555
TIR2 (%)	91.79 ± 10.75	1.324	0.197	92.01 ± 8.30	−1.665	0.107	0.084	0.934
TIR13 (%)	94.03 ± 6.30	2.508	0.018	88.31 ± 10.87	2.485	0.019	−2.412	0.019
TIR14 (%)	94.86 ± 5.03	3.797	0.001	89.79 ± 7.38	2.965	0.006	−3.006	0.004
TBR1 (%)	0.15 (0.0, 3.27)	–	–	0.52 (0.05, 2.07)	–	–	1.279	0.204
TBR2 (%)	0.24 (0.0, 2.33)	−0.845	0.418	0.43 (0.0, 3.05)	−1.638	0.105	0.355	0.727
TBR13 (%)	0.36 (0.0, 2.47)	−0.365	0.733	0.24 (0.0, 3.15)	2.402	0.015	0.696	0.492
TBR14 (%)	0.3 (0.0, 3.49)	−0.308	0.775	0.53 (0.0, 8.17)	−2.573	0.009	0.318	0.756

Note: In the intragroup comparison, the blood glucose fluctuation indices at W2, W13, and W14 were compared to those at W1.
Abbreviations: QD, quinoa diet; RD, regular diet; CV, coefficient of variation; TIR, time in range; TAR, time above range; TBR, time below range.

Table 4 Comparison of Secondary Outcome Indices Between the Two Groups Before and After Intervention (n (%), $\bar{X} \pm s$)

Variables	QD-Post	Compared with QD-Pre		RD-Post	Compared with RD-Pre		QD Post vs RD Post	
		t	*P*		*t*	*P*	*t/Z*	*P*
Weight (kg)	71.46 ± 10.28	3.007	0.006	73.18 ± 11.84	1.462	0.155	0.579	0.565
BMI (kg/m^2)	24.32 ± 2.51	2.925	0.007	24.32 ± 2.51	1.424	0.166	1.296	0.201
FINS (mIU/L)	7.30 ± 3.89	1.569	0.129	8.53 ± 4.89	0.562	0.579	1.036	0.305
INS0.5h (mIU/L)	27.69 ± 17.81	0.025	0.980	33.37 ± 23.41	0.585	0.564	0.824	0.414
INS2h (mIU/L)	51.87 ± 32.47	0.523	0.605	45.07 ± 34.62	1.283	0.211	0.308	0.759
GLP-1 (pmol/L)	2.34 ± 0.77	1.664	0.108	2.04 ± 0.76	0.613	0.546	1.390	0.171
HOMA-β	1.62 ± 0.30	0.406	0.688	1.70 ± 0.26	1.041	0.307	0.991	0.326
HOMA-IR	0.33 ± 0.18	2.666	0.013	0.36 ± 0.21	0.440	0.663	0.655	0.515
TC (mmol/L)	4.33 ± 0.86	2.712	0.012	4.39 ± 0.94	2.529	0.018	0.760	0.450
TG (mmol/L)	1.29 ± 0.58	2.942	0.007	1.33 ± 0.66	1.573	0.129	0.219	0.828
LDL-C (mmol/L)	2.55 ± 0.89	1.547	0.133	2.66 ± 0.75	1.792	0.085	0.488	0.628
HDL-C (mmol/L)	1.37 ± 0.51	1.541	0.135	1.32 ± 0.34	0.043	0.966	0.429	0.670
UA (μmol/L)	324.24 ± 59.48	1.354	0.190	343.70 ± 100.96	0.119	0.906	0.821	0.416
Medications Red or Dis (%)	28.60	–	–	7.10	–	–	4.383	0.036

Abbreviations: QD, quinoa diet; RD, regular diet; BMI, body mass index; INS, insulin; GLP-1, glucagon-like peptide-1; HOMA-β, β-cell function was assessed using the Homeostasis Model Assessment; HOMA-IR, insulin resistance was assessed using the Homeostasis Model Assessment; TC, total cholesterol; TG, triglycerides; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; UA, uric acid.

demonstrated potential in facilitating T2DM remission. However, these approaches face challenges, including poor adherence, high cost, limited applicability across populations, difficulty in long-term maintenance, and insufficient evidence regarding their long-term effects. Functional foods are increasingly recognized as important components of dietary strategies for diabetes management, representing a paradigm shift from simple "restriction" to active "empowerment".[22,23] Unlike basic calorie reduction, the benefits of functional foods are supported by relatively well-defined physiological and molecular mechanisms—such as low GI effects and prebiotic activity—enabling more precise dietary interventions.[24] Notably, the quinoa products tested were composite foods containing 15–20% quinoa rather than

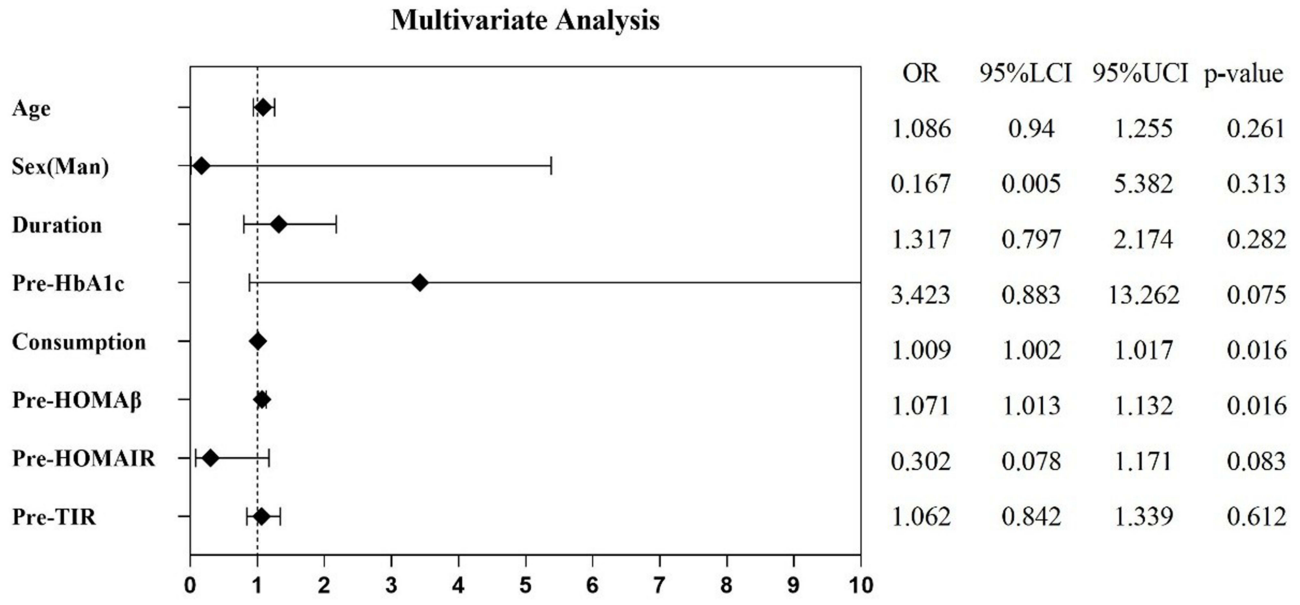

Figure 4 Correlation analysis of drug consumption reduction or withdrawal. Pre-HbA_{1c}, glycated haemoglobin A1c before QD, HOMA-β, and β-cell function were assessed using the homeostasis model assessment, and HOMA-IR and insulin resistance were assessed using the homeostasis model assessment.
Abbreviation: TIR, time in range.

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pure quinoa, and both groups received structured dietary guidance. Therefore, the observed benefits might reflect broader improvements in dietary quality and meal planning, with quinoa contributing incremental effects within this comprehensive intervention framework.

In T2DM management, although both diet and physical activity are cornerstone interventions and existing studies suggest that these behaviours are often interrelated, the effects of dietary interventions tend to be more consistent and measurable.[25] While the present study lacks detailed records on physical activity, all participants received standardized exercise guidance at baseline, and adherence was monitored during follow-up to ensure comparability between the groups. Therefore, the lack of detailed physical activity records is unlikely to substantially affect the study conclusions.

The low GI of quinoa may be a key factor underlying its ability to modulate glucose and lipid metabolism. This property is largely attributable to its high content of dietary fibre, high-quality protein, and resistant starch, which act synergistically via physical, hormonal, and microbial pathways to exert multilevel regulatory effects.

At the physical level, dietary fibre in quinoa—particularly soluble fibre—forms a viscous gel matrix in the gastrointestinal tract, which delays gastric emptying[26] and impedes contact between digestive enzymes and carbohydrates,[27,28] thereby slowing glucose digestion and absorption. This process flattens the postprandial glucose curve, mitigates sharp fluctuations in blood glucose and insulin, and reduces the sustained stimulation of pancreatic β-cells caused by glucotoxicity.

Furthermore, at the level of hormonal regulation, the aforementioned physical delaying effect, in conjunction with the short-chain fatty acids (SCFAs) produced through the gut microbial fermentation of quinoa acting as a prebiotic, significantly stimulates intestinal L-cells, leading to a marked increase in GLP-1 secretion.[29] Additionally, SCFAs directly act on L-cells by activating G protein-coupled receptors (eg, GPR43/FFAR2, GPR41/FFAR3), thus upregulating the synthesis and secretion of GLP-1.[29–31] Concurrently, the avoidance of postprandial hyperinsulinaemia contributes to reduced hepatic lipogenesis. Studies have demonstrated that a low-GI diet effectively decreases liver fat content and circulating triglyceride levels.[32]

In addition to these acute metabolic effects, quinoa-based products also mediate long-term benefits through gut microbiota modulation. Indigestible carbohydrates from quinoa-based products function as prebiotics that selectively promote the growth of beneficial gut microbiota.[33,34] This microbial modulation, in turn, regulates host metabolism by downregulating the colonic TAS1R3/TRPM5 signalling pathway and upregulating the gene expression of GLP-1.[35] Microbial fermentation of these fibres yields SCFAs such as butyrate and propionate. Butyrate strengthens the intestinal barrier, reduces systemic inflammation caused by endotoxin translocation, and directly enhances insulin sensitivity in the liver and muscle.[36] Propionate, in turn, helps suppress hepatic gluconeogenesis.[37] This cross-talk along the gut–liver and gut–peripheral tissue axes represent a crucial long-term mechanism through which quinoa improves glucose and lipid metabolism and ameliorates insulin resistance.

In summary, the low-GI property of quinoa is not an isolated feature but an initiating factor that triggers a cascade of metabolic improvements—from physical and hormonal modulation to microbiota-mediated systemic effects. Through this multitargeted, multilevel mechanism, quinoa provides comprehensive benefits to glycolipid metabolism in T2DM.

Limitations

This study had several limitations. First, the small sample size and single-centre design may have introduced bias. Second, the lack of physical activity monitoring, coupled with the relatively short 12-week intervention period, may have influenced the observed effects. However, both groups received standardized dietary and exercise guidance to minimize lifestyle confounding and better isolate the potential effect of quinoa substitution. The food provided to the participants contained other ingredients, but the effects of these ingredients remain unclear. Furthermore, changes in the intestinal flora and its metabolites were not assessed, which may represent a crucial mechanism through which a quinoa diet regulates blood glucose and lipid levels. In future research, we plan to expand the sample size, conduct long-term follow-up observations, regularly document other food intake and exercise habits of patients, analyse the intestinal flora, and clarify the role and mechanisms of a quinoa diet.

Conclusion

In conclusion, this study demonstrates that quinoa-based dietary products, when incorporated into a comprehensive dietary intervention, may provide incremental benefits for glycaemic control in patients with early-stage T2DM, particularly those with better baseline β-cell function. Notably, the products contained 15–20% quinoa along with other ingredients, and both groups received structured dietary guidance. These findings suggest that quinoa-based foods may be a feasible and beneficial component of dietary management for T2DM in Chinese dietary culture. Future studies involving larger sample sizes, longer follow-up durations, and detailed analyses of the gut microbiota and its metabolites are warranted to confirm the long-term benefits and elucidate the precise mechanisms of QD interventions in T2DM management.

Abbreviations

GI, glycaemic index; HbA_{1c}, glycated haemoglobin A_{1c}; T2DM, type 2 diabetes mellitus; QD, quinoa diet; RD, regular diet; OGTT, oral glucose tolerance test; cGMS, continuous glucose monitoring system; BMI, body mass index; HP, hypertension; OADs, oral antidiabetic drugs; BG, blood glucose; FBG, fasting blood glucose; INS, insulin; AUC, area under the glucose curve; TC, total cholesterol; TG, triglyceride; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; GLP-1, glucagon-like peptide-1; UA, uric acid; HOMA-β, β-cell function was assessed using the homeostasis model assessment; HOMA-IR, insulin resistance was assessed using the homeostasis model assessment; CV, coefficient of variation; TIR, time in range; TAR, time above range; TBR, time below range.

Data Sharing Statement

The trial protocol, datasets used, and statistical analysis plan during the current study are available from the corresponding author upon reasonable request.

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Disclosure

The authors report no conflicts of interest in this work.

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