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Digestion Properties of Quinoa Starch and its Effect on Streptozotocin-induced Diabetic Mice

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Abstract

The present study aimed to investigate the digestion properties of quinoa starch and its effect on blood glucose by. The results showed that quinoa starch had a diffraction pattern between type A and type B and hence was identified as type C starch. Additionally, the diffraction peak at 20° corresponded to the V-shaped crystals, indicating the presence of an amylose and lipid complex in quinoa starch. The in vitro starch digestibility was determined by the Englyst method, and the obtained contents of readily digestible starch (RDS), slowly digestible starch (SDS), and resistant starch (RS) were 72.79%, 6.84%, and 20.67%, respectively. Furthermore, RS was classified using the comparative analysis of raw and cooked starch under the same treatment, and the proportions of raw starch granules and retrograded starch were 10.19% and 20.67%, respectively. Finally, the diabetic mouse trial was performed using various indicators, and the results showed that the customized feed obtained by replacing the original maintenance grain starch with quinoa starch had a significant relief effect on the mice with diabetes. As for diet, food utilization was high, and the body weight was maintained normal and stable, which were conducive to alleviating their symptoms of polyphagia and emaciation. During plasma glucose metabolism, quinoa starch stabilized with fasting blood glucose (FBG) was maintained in the normal range of 5 - 7 mmol/L. According to insulin tolerance and oral glucose tolerance tests, speed fluctuations of blood glucose were avoided under the stimulation of foreign hormones or glucose, making it relatively stable. Meanwhile, quinoa starch significantly improved hyperlipidemia, decreased the contents of triglycerides (TG), cholesterol (CHO), low-density lipoprotein cholesterol (LDL-C), and increased the high-density lipoprotein cholesterol (HDL-C) content.

Keywords: Quinoa; Quinoa Starch; In Vitro Digestibility; Glucose Tolerance; Insulin

Introduction

Quinoa (*Chenopodium quinoa* Willd.), also known as quinoa grain, South America quinoa, *Chenopodium quinoa*, etc. is an annual dicotyledonous plant of *Chenopodium* L., mainly distributed in Bolivia, Ecuador, Peru, and Chile of South America. Quinoa is an alkaline food with high protein (16% - 18%) and low calories, and abundant in dietary fibers, vitamins, minerals, and active substances. Furthermore, it contains all the essential fatty acids, amino acids, polyphenols, flavonoids, saponins, betaine, phytosterols, and other phytochemicals

beneficial to human health [1-4]. It is often used as a nutritional supplement to treat celiac disease or other digestive diseases due to its gluten-free proteins [5]. Therefore, it is recommended as the perfect "whole food" for mankind by the United Nations Food and Agriculture Organization and listed as one of the top 10 healthy nutrient-rich foods worldwide [6,7].

With the development of quinoa cultivation, research on its nutritional value and functionality has become more advanced, and it is found that quinoa can prevent various diseases, such as cancer, inflammation, hyperglycemia, and hyperlipidemia. Appropriate dietary management is an important means to reduce the risk of diabetes, which is considered one of the most common chronic diseases worldwide [8]. In this regard, the intake of quinoa could increase the insulin content in the blood [9], and the addition of germinated quinoa flour to noodles could effectively decrease the glycemic index [10]. Liu., *et al*. [11] discovered that the supplementation of quinoa flour and fermented quinoa to the experimental animals in a diabetic model effectively controlled the blood glucose. They established a diabetic mouse model and found that after 6 weeks, the fasting blood glucose decreased in the mice fed with quinoa flour. Compared with the mice not fed with quinoa flour, the mice fed with quinoa flour exhibited a better hypoglycemic effect during the oral glucose tolerance test, with the curve- covered area of plasma glucose declining, and the decrease was more pronounced in the high-dose group.

Studies have reported that quinoa protein hydrolysis could generate plasma glucose regulatory peptides [12-14]. The polysaccharides isolated and obtained by Tan., *et al*. [15] from quinoa could regulate blood glucose metabolism, but the effects of starch, accounting for 58% - 64.2% of the total dried quinoa substances, on blood glucose has not been fully elucidated yet.

The starch granules of quinoa are polygonal and pose a pseudoplastic non-Newtonian fluid structure. The pyran ring, -OH, -CH2, and -CHO are the typical functional groups of starch molecules, with excellent stability, emulsification, and gelation. Thus, starch-rich quinoa is a potent grain for processing various foods [16,17]. Starch, composed of starch granules and embedded in a continuous region consisting of numerous starch granules, is shaped as an oval cumulus in the quinoa endosperm under its natural state, ranging from 30 - 70 μm in size [18]. The starch granules comprising of continuous regions are ellipse-like, while the cumulus-shaped granules are polygonal in structure, which is also prevalent in other grains. However, the cumulus conformation disappears after isolation [18]. The crystal type of quinoa starch is an A-type crystal related to the alternating arrangement of the amorphous layer of amylose and the crystallizing layer of amylopectin. The particle size of the starch is generally distributed in the range of 0.2 - 0.4 μm, which is less than most plant-derived starches [19], attributing to its higher sensitivity to enzymes [20]. Quinoa starch poses the basic carbohydrate function of providing energy and exerts a certain impact on satiety/stomach emptying, blood glucose, and insulin metabolism control, protein glycosylation, and cholesterol and triglyceride metabolism [21]. According to *in vitro* digestibility, starch can be divided into readily digestible starch (RDS), slowly digestible starch (SDS), and resistant starch (RS). Of them, RS has a low glycemic index and can effectively control postprandial blood glucose levels and body weight.

Aim of the Study

The present study aimed to evaluate the effect of quinoa on plasma glucose in streptozotocin-induced diabetic mice based on the *in vitro* digestion test of quinoa starch to provide a theoretical basis for the dietary design of diabetic patients.

Materials and Methods

Quinoa starch and customization of quinoa starch maintenance feed

The quinoa grains were ground and sieved using an 80-mesh sieve. The starch was extracted according to the methods reported by N. Thoufeek Ahamed., *et al*. [22]. Accurately, 0.25% NaOH solution was added at a solid-to-liquid ratio of 1:5 under magnetic stirring till no protein was detected by the biuret reagent. Then, the solution was centrifuged at 4025g for 15 minutes followed by the removal of the supernatant, washing several times till neutral, and filtration over a 200-mesh filter cloth. After drying at 50°C in the oven, the substance

was crushed and passed through a 60-mesh sieve. Then, petroleum ether reflux was performed for 12h to defat, followed by drying and sealed storage.

Customized base maintenance feed: The corn starch in the original maintenance feed was replaced with quinoa starch, prepared by Beijing HFK Bioscience. Co., LTD. The formula of the customized quinoa starch maintenance feed is listed in table 1.

			Carbohydrates			
	Protein	**Fat**	**Quinoa starch**	**Maltodextrin**	**Sucrose**	**Cellulose**
Mass ratio /(g%)	14.20	4.00	49.57	12.50	10.00	5.00
Energy ratio /(kcal%)	14.70	9.40	51.50	12.99	10.39	0

Table 1: *Nutritional composition of customized quinoa starch maintenance feed.*

Determination of crystal type and relative crystallinity of quinoa starch and common starch

X-Ray diffraction analysis conditions: CuKa characteristic ray, graphite monochromator, a voltage of 40 kV current of 30 mA a measurement angle range of 2θ = 3°~70°, emission and anti-reflection slit of 1°, receiving slit of 0.3 mm, a scan speed of 1.5°/min, step width of 0.02° [23].

Data processing and analysis: The crystallinity of each starch was calculated by JADE software according to a previously reported method [23].

In vitro digestion test of quinoa starch

The *in vitro* digestion test was performed according to the method of Chen, YF [24] with slight modification. Reparation of digestive enzyme solution: trypsin (4 × USP specifications) was added to 100 mL of distilled water, followed by magnetic stirring for 10 minutes and centrifugation at 1500g for 10 minutes. Then, 80 mL of supernatant was collected, and amyloglucosidase (120U) and convertase (15000U) were added.

Determination of glucose content: 3, 5-dinitrosalicylic acid method

Accurately 0.5g of starch samples were taken and 20 mL of 0.1 mol/L sodium acetate buffer (pH 5.2) containing 4 mmol/L of calcium chloride was added. Then, one group of samples was immersed in boiling water for 20 minutes (starch gelatinization), while no treatment was conducted on the other group. After the samples were vibrated in the water bath at 37°C for 10 minutes, the temperature was maintained at 37°C, and 5 mL of the digestive enzyme was added. Two samples (raw and cooked) were collected at the time intervals of 0 minute, 20 minutes, and 120 minutes, and were boiled for 10 minutes followed by dilution and centrifugation at 1500g for minute. The supernatant was collected to determine the released glucose content. The remaining samples were boiled for 30 minutes, and 10 ml of 7 mol/L KOH was added to an ice-water bath. Approximately 1 mL of the mixture was added to 10 mL of 0.5 moL/L of the acetic acid solution, followed by the addition of 0.2 mL of starch glucosidase (50 AGU/mL). After reacting in the water bath at 70°C for 30 minutes, boiling water bath kills enzyme. After cooling, the supernatant was extracted. The RDS, SDS, and RS contents of different components were calculated according to the following equations in the unit of mg/mL.

$TS = (T_G - F_G) \times 0.9$ Equation (1)

$RDS = (G_{20} - F_G) \times 0.9$ Equation (2)

$SDS = (G_{120} - G_{20}) \times 0.9$ Equation (3)

$RS = TS - (RDS + SDS)$ Equation (4).

Note: TS was modified as free glucose (mg/mL); T_G was modified as glucose content (mg/mL) completely released by enzymatic hydrolysis; F_G indicates free glucose content (mg/mL) at 0 minute; G_{20} indicates glucose content (mg/mL) after enzymatic hydrolysis for 20 minutes; G_{120} indicates glucose content (mg/mL) after enzymatic hydrolysis for 120 minutes.

Diabetic mouse model establishment and their feeding trials

Molding and feeding

After adaptive feeding for one week, the mice were randomly divided into six groups according to their body weight (See table 2), with ten mice in each group and five mice per cage. The mice were allowed to fast overnight for 12h, and the other groups, except for the normal control group, were injected with streptozotocin solution at a dose of 100 mg/kg. After normal feeding for 3 days, the mice were allowed to fast for 12h, with free access to water. Then, the blood was sampled from the tail vein to detect the FBG. The mice were tested for 4 weeks until successful molding (fasting glucose > 11.1) based on table 2.

	Normal control		Model		Positive control	Test
	K	**KL**	**M**	**ME**	**MG**	**ML**
Feed type	Ordinary	Custom feed	Ordinary	Ordinary	Ordinary	Custom feed
Gastric gavage	Saline	Saline	Saline	75.83 mg/kg Metformin solution	0.3 mg/kg Glimepiride solution	Saline

Table 2: *Mice feed and gavage arrangement during the treatment cycle.*

Food intake, body weight, and FBG were recorded weekly during the test, and insulin tolerance and oral glucose tolerance tests were performed after completing the procedure.

FBG measurement in mice

The mice were allowed to fast for 12h. Then, the blood was collected from the tail to measure the blood glucose content using a blood glucose meter.

Insulin tolerance test

After 4 weeks of feeding, the mice were allowed to fast for 4h with free access to water, and the blood glucose content was measured. The 0.5 U/kg of insulin solution was subcutaneously injected to the nape, and the blood was sampled through the tail vein. The blood glucose contents after 0.5h, 1h, and 2h of insulin injection were measured by a glucometer, and the insulin tolerance curve was drawn. The percentage drop of blood glucose was calculated according to equation (5).

Percentage drop of blood glucose $= ((FBG_t - FBG_0)/FBG_0) \times 100\%$ Equation (5)

Where FBG_t refers to the blood glucose contents after insulin injection for 0.5h, 1h, and 2h; FBG_0 refers to the blood glucose content after 4h of fasting.

Oral glucose tolerance test

After 4 weeks of feeding, the mice were allowed to fast for 4h with free access to water, and the FBG was measured in the early morning. Then, 20% glucose solution was administered intragastrically at a dose of 10 mL/kg, i.e. 2.0 g/kg of glucose. The blood glucose level was measured after 0.5h, 1h, and 2h of glucose administration through tail cutting, and the change in glucose was recorded to calculate the area under the blood glucose curve.

Statistical analysis

The data were statistically analyzed using SPSS 26.0 and Excel 2020, and the curve was drawn using Origin 8.6 software.

Results and Discussion

Relative crystallinity of quinoa starch

Starch is a natural polycrystalline polymer and can be divided into three types according to its structure, such as amorphous, subcrystallized and crystallized starch. Different particle sizes present different diffraction characteristics on the X-ray diffraction curve, which can be adopted to divide the starch microcrystal structure. The region with large grain linearity, complete crystal structure, and ordered length showed obvious peak diffraction characteristics on the X-ray diffraction curves, named crystal zone, while the region with short range order and long-range disorder showed obvious dispersion diffraction characteristics on the X-ray diffraction curve, named amorphous zone (i.e. non-crystal zone). The changes in the intensity of the X-ray diffraction peak and half-peak width reflected the grain size, amorphization degree, and lattice distortion of starch granules. The crystallinity of natural starch is generally between 15% and 45%. Plant starch poses 4 types of crystals, namely type A, B, C, and V. As depicted in figure 1 and table 3, the X-ray diffraction spectra of quinoa starch exhibited strong peaks around 2θ of 15°, 17°, and 23°, corresponding to type A crystals; the diffraction peak at 26° corresponded to the characteristic diffraction peak of type B crystal, indicating that quinoa starch has a diffraction peak between type A and B. Therefore, quinoa starch was regarded as type C crystal. Furthermore, the diffraction peak at 20° corresponded to type V crystal, indicating the presence of an amylose and lipid complex in quinoa starch. The crystallization zone is mainly formed by the amylopectin molecules with a compact, double-helical structure; the amorphous zone is mainly formed by the amylose molecules with a loose structure, which is more susceptible to external forces and chemical reagents than the crystalline zone. As shown in table 4, the relative crystallinity of quinoa starch was significantly higher than that of wheat, rice, and corn, confirming its crystallization area to be relatively large. The structure of quinoa starch was similar to that of glutinous rice flour, with a higher resistance to external forces and chemical reagents, which cannot be digested or absorbed in the body easily.

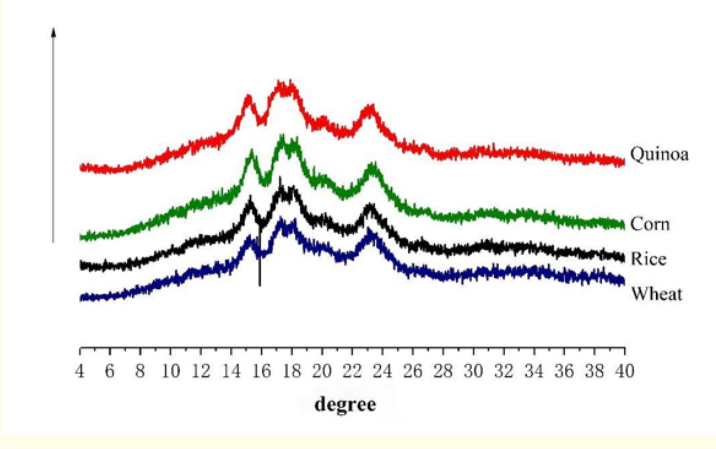

Figure 1: *X-ray diffraction pattern of each starch.*

Type of starch	Crystal form	Diffraction angle 2θ										
		5.6°	11°	15°	17°	18°	20°	22°	23°	24°	26°	34°
Quinoa	C	—	—	S	D		MW	—	MS	—	MW	—
Wheat	A	—	—	S	D		—	—	S	—	—	—
Corn	A	—	—	S	D		—	—	S	—	—	—
Rice	A	—	—	S	D		—	—	S	—	—	—

Table 3: *Shape and intensity of XRD diffraction peaks of each starch.*

Note: Letters indicate the strength of peaks: S: Strong, W: Weak, MW: Medium Weak, MS: Medium Strong, D: Double Peaks.

	Quinoa	Wheat	Corn	Rice
Relative crystallinity/(%)	45.82 ± 0.67^{c}	42.95 ± 0.88^{b}	40.91 ± 0.34^{a}	41.60 ± 1.74^{ab}

Table 4: *Relative crystallinity of each starch.*

Note: Quinoa, wheat, corn, rice crystalline region data are expressed as mean ± SD (n = 3), and the difference in different lowercase letters for the same row of data was statistically ($P < 0.05$) significant.

Composition of quinoa starch and its digestibility *in vitro*

The starch that can be digested and decomposed within 20 minutes is usually defined as RDS, and the starch digested within 20 - 120 minutes is defined as SDS. The starch that cannot be digested and decomposed in the small intestine after 120 minutes but can be used by the colon is defined as RS. RS has excellent physiological functions, such as preventing intestinal diseases, stabilizing postprandial blood glucose levels, reducing cholesterol, and inhibiting fat accumulation, with unique food processing characteristics of white color, high thermal stability, and low water retention [16]. The composition of quinoa starch was determined according to the methods described in section 2.2, and the results are shown in table 5. The RDS content of cooked starch in quinoa starch was 72.79%, which was lower than the RDS content of five cereal starches (80.4% - 85.4%), four root starches (80.2% - 86.7%), and three legume starches (78.3% - 79.7%) reported by Miu., *et al*. [25]. The SDS content of quinoa starch was 6.84%, which was within its commonly reported starch range (4.9% - 12.5%), while the RS content (20.67%) was much higher than that of the reported content in common starch (6.5% - 13.4%). The above results demonstrate that less quinoa starch is decomposed to produce glucose by enzymes after entering the small intestine of animals, and a large fraction of starch belongs to RS, which does not participate in the digestion and absorption, but actively participates in the intestinal microbial activities.

	Starch ratio (%)	
	Cooked starch	Raw starch
Fast-digesting starch	72.79 ± 4.59	21.43 ± 1.01
Slow-digesting starch	6.84 ± 0.41	47.71 ± 3.42
Resistant starch	20.67 ± 2.20	30.86 ± 2.51

Table 5: *Proportion of three types of starch in four grains.*

Note: The proportion of fast-digesting starch, slow-digesting starch and resistant .starch in cooked starch and raw starch is expressed as a mean ± SD (n = 3).

Effect of quinoa starch on diabetic mice

The *in vitro* digestion test results described in section composition of quinoa starch and its digestibility *in vitro* concluded that quinoa starch has a higher RS content compared to other common starch molecules. Since the test only simulated the digestion of starch in the small intestine, it cannot reflect the digestion and absorption of starch after entering the body. However, the digestion and absorption of carbohydrates can be reflected by the changes in blood glucose. In this study, a diabetic mouse model was established and a customized quinoa starch feed was used to evaluate the effect of quinoa starch with high RS on blood glucose control through different indexes, such as physical signs, FBG, insulin tolerance, oral glucose tolerance, and serum lipid.

Effect of quinoa starch on changes in physical signs and body weight in mice

During the four-week feeding period, the physical signs and behaviors of mice were observed daily. The signs of normal control group (K) mice changed slowly with the progress of the test cycle; after successful modeling, the diabetic mice developed polydipsia and polyuria symptoms. With the intragastric administration and supply of metformin, glimepiride, and a customized quinoa starch feed to the positive control group, the symptoms of polydipsia and polyuria improved, while in the model group, the symptoms became more intense with time, and the bedding materials were wet and smelly with severe lumps.

As shown in table 6, there was no significant difference in the body weight among the groups during gavage, and the weight growth of mice was stable. The dietary status of the mice in each group was assessed according to figure 2. The intake of mice in each group showed no difference before molding; after 4 weeks of molding, the daily average intake of mice in the blank, model, metformin, and glimepiride groups continuously increased every week without a difference. The daily average intake of mice in the blank quinoa starch group and model quinoa starch group was lower and stable. The food utilization of mice was calculated and the significance was analyzed by comparing the results of table 6 and figure 2, and the results are shown in table 7. There was no difference in food utilization in the first week. However, the food utilization of two groups fed with a customized quinoa starch feed was significantly higher than the other groups over time, suggesting that a customized quinoa starch feed could significantly relieve the symptoms of polydipsia, polyphagia, and emaciation in diabetic patients. The food intake is well controlled under the condition of maintaining a normal weight.

Effect of quinoa starch on fasting blood glucose (FBG) changes in mice

The FBG of mice was evaluated for 4 weeks after successful molding, and the results are depicted in figure 3.

The FBG changes in mice are depicted in figure 3, and after successful molding, the FBG of diabetic mice was > 11.1 mmol/L. The FBG in the blank group was lower than that in the model group, with a value above 8, indicating a possible stress response in mice. In the first

	Group					
	K	**KL**	**M**	**ML**	**ME**	**MG**
Molding	29.3 ± 1.62^{a}	29.8 ± 1.67^{a}	28.9 ± 1.65^{a}	29.67 ± 2.16^{a}	28.95 ± 1.85^{a}	29.7 ± 2.41^{a}
First week	35.2 ± 2.87^{ab}	34.55 ± 2.79^{a}	36.8 ± 2.03^{ab}	36.04 ± 2.32^{ab}	35.6 ± 2.27^{ab}	37.05 ± 2.06^{b}
Second week	36.95 ± 3.53^{ab}	36.5 ± 3.43^{a}	39.6 ± 2.55^{b}	36.96 ± 2.94^{ab}	36.2 ± 3.09^{a}	38.4 ± 2.67^{ab}
Third week	40.35 ± 4.28^{a}	39.5 ± 4.04^{a}	40.65 ± 2.05^{a}	40.29 ± 3.21^{a}	38.9 ± 3.66^{a}	41.1 ± 2.16^{a}
Fourth week	42.87 ± 4.48^{a}	41.49 ± 3.82^{a}	41.8 ± 2.54^{a}	42.3 ± 3.86^{a}	41.41 ± 3.76^{a}	42.9 ± 2.61^{a}

Table 6: *Changes in body weight during gavage.*

Note: Weight data for different groups were expressed as mean ± SD (n = 3), and the difference in different lowercase letters for the same row of data was statistically (P < 0.05) significant.

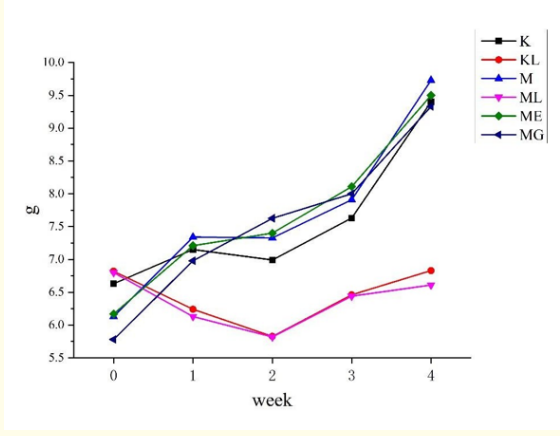

Figure 2: *Daily average food intake of mice over time.*

	Group					
	K	**KL**	**M**	**ML**	**ME**	**MG**
First week	11.79 ± 1.41[a]	10.87 ± 3.07[a]	15.38 ± 1.93[a]	14.85 ± 1.77[a]	13.18 ± 0.70[a]	15.04 ± 3.91[a]
Second week	3.58 ± 0.66[a]	4.78 ± 0.34[a]	5.46 ± 0.00[b]	2.26 ± 0.00[c]	1.16 ± 0.07[d]	2.53 ± 0.13[c]
Third week	6.37 ± 0.79[ab]	6.63 ± 1.25[a]	1.90 ± 1.25[c]	7.39 ± 0.38[a]	4.76 ± 0.00[b]	4.82 ± 1.52[b]
Fourth week	2.68 ± 0.92[ac]	4.16 ± 1.09[b]	1.69 ± 1.10[c]	4.34 ± 0.22[b]	2.77 ± 0.09[ac]	2.76 ± 0.04[c]

Table 7: *Food utilization rate.*

Note: Food utilization data of different groups of mice on a weekly basis are expressed as the mean ± SD (n = 3), and the difference between different groups of data with different lowercase letters in the same week is statistically (P < 0.05) significant.

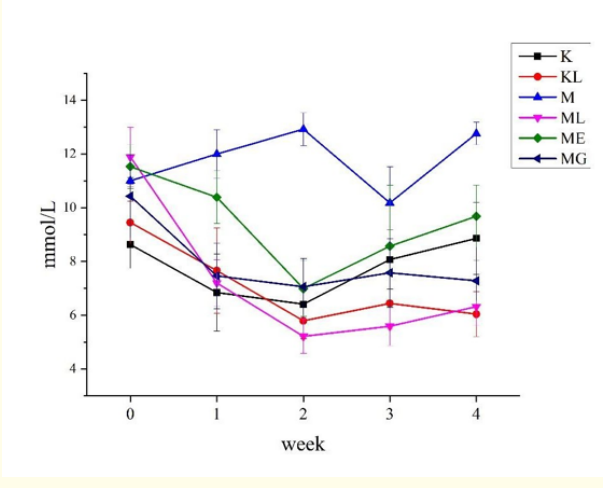

Figure 3: *Changes in fasting blood glucose in mice.*

week, except for the model group, the FBG of mice decreased in other groups. It was observed that FBG in the metformin group decreased slower compared with the glimepiride group, as metformin improves insulin resistance while glimepiride promotes insulin secretion. The pathogeny of mouse diabetes is corresponded to insulin deficiency *in vivo*, indicating that the model was type I diabetes and was successfully established. The FBG of mice fed with a customized quinoa starch feed was low and stable, which was consistent with the positive control group. This result indicated that the customized quinoa starch feed was beneficial to FBG control within a healthy range.

Effect of quinoa starch on glucose tolerance in mice

Oral glucose tolerance tests can reflect the body's ability to regulate blood glucose. Figure 4 depicts the changes in blood glucose after fasting and glucose administration in each group.

The mice were subjected to oral glucose tolerance tests after 4 weeks of feeding. The blood glucose contents were measured after fasting for 12 h and gavage of glucose for 0 minute, 30 minutes, 60 minutes, and 120 minutes, respectively. As depicted in figure 4, the blood glucose content of mice in the model group after 12h-fasting, i.e. before glucose administration, was significantly higher than those in other groups. The blank group exhibited a significant difference compared with the metformin group and the other three groups ($P < 0.05$). After 30 min of glucose gavage, the blood glucose of mice in each group significantly increased and reached the peak. Of them, the model group had the highest blood glucose content (21.08 mg/mL), and the glimepiride group had the lowest (8.82 mg/mL) content, showing a slow increase. Considering the fact that the main effect of glimepiride promotes insulin secretion, this result indicated that the glimepiride group regulated blood glucose well. However, the model quinoa starch group showed no significant difference compared with the glimepiride group ($P < 0.05$), indicating that quinoa starch also has a good effect on blood glucose regulation. Moreover, there was no significant difference in the blood glucose content among the blank, the blank quinoa starch, and metformin groups ($P < 0.05$). After intragastric glucose administration for 1h, there was no significant difference in the blood glucose content among the blank, blank quinoa starch, and model groups, while the blood glucose contents of the model quinoa starch and glimepiride groups were significantly lower than that of the model group ($P < 0.05$), which remained relatively stable. After 2h, the blood glucose contents of the blank quinoa starch, model quinoa starch, and glimepiride groups were significantly lower than that of the model group mice ($P < 0.05$).

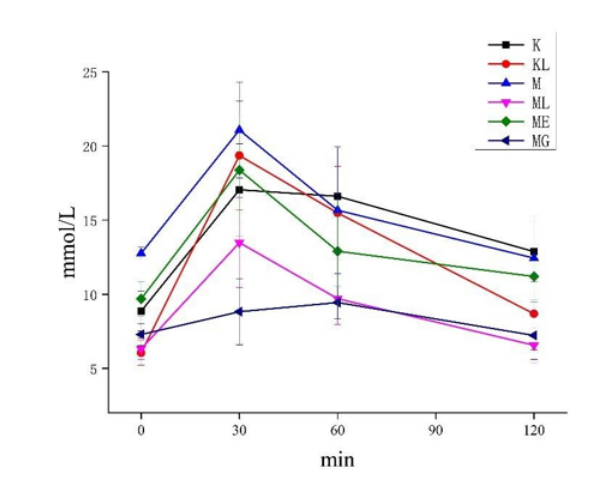

Figure 4: *Oral glucose tolerance curve.*

As depicted in figure 5, the under-curve area of the blank quinoa starch, model quinoa starch, model metformin, and model glimepiride groups was significantly lower than that of the model group ($P < 0.05$), indicating that all groups had the effect of improving the glucose tolerance level in mice. Of them, the effect of the model quinoa starch and glimepiride groups was significantly higher than those of the other two groups ($P < 0.05$), suggesting that quinoa starch could effectively inhibit the rapid increase of postprandial blood glucose.

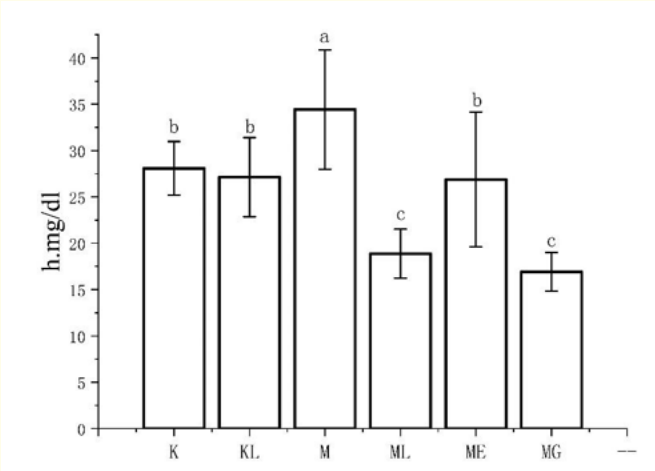

Figure 5: *Oral glucose tolerance test area under the blood glucose curve.*

Effect of quinoa starch on insulin tolerance in mice

Insulin tolerance tests can evaluate the sensitivity of the body to insulin according to the changes in blood glucose content. Figure 6 depicts the blood glucose in each group of mice after subcutaneous insulin injection.

According to the insulin tolerance curve in figure 6, after 4h of fasting, the blood glucose in the model group was significantly higher than in the other groups ($P < 0.05$), and the blood glucose content in the metformin group was higher than in the remaining 4 groups. The blood glucose contents of mice decreased in all groups after 30 minutes of subcutaneous insulin injection. Of them, the blood glucose decreased faster in the metformin group but decreased slower in the model group, which was significantly higher than in the other groups ($P < 0.05$). After 1h, the blood glucose of the model and metformin groups significantly increased than that of the other groups ($P < 0.05$). After 2h, the blood glucose of each group basically returned to the original value. The decreased rate of blood glucose can be observed more intuitively from the percentage decline curve in figure 7. After insulin injection, the blood glucose of mice in each group first decreased rapidly and then increased slowly. The decrease rate of blood glucose reflects the sensitivity of the mice to insulin. The blood glucose of the mice in the quinoa starch group remained in the normal range throughout the test, indicating that the insulin regulation ability of the mice in the two groups fed with quinoa starch was enhanced, and it was not susceptible to a sudden change of insulin dose.

Effect of quinoa starch on insulin levels and insulin resistance in mice

Insulin is a crucial hormone for regulating blood glucose. Increased blood glucose is closely related to insulin content, and type I diabetes is an absolute deficiency of insulin. Streptozotocin can cause damage to pancreatic B cells, causing a decrease in insulin content.

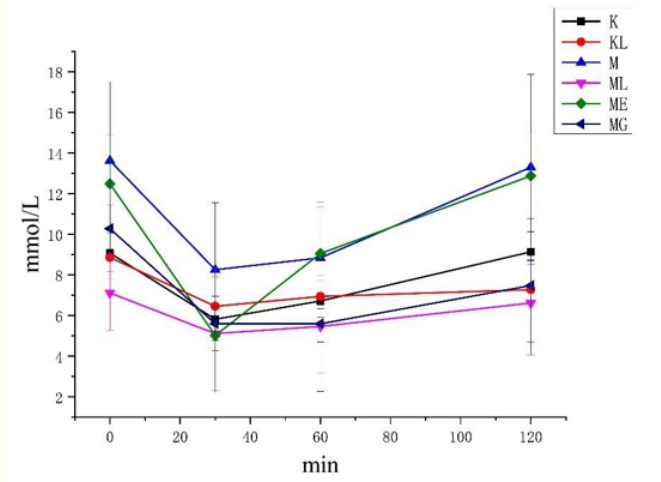

Figure 6: *Insulin tolerance curve.*

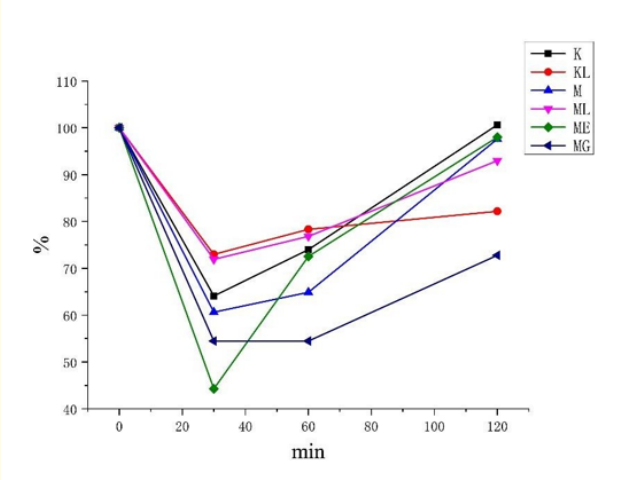

Figure 7: *Percentage drop curve of blood glucose values.*

As shown in table 8, the insulin level in the model group was significantly lower than that of the other groups, i.e. insulin deficiency, which was consistent with the cause of type I diabetes. The insulin levels of the mice in the model quinoa starch, metformin, and glimepiride groups increased to varying degrees ($P < 0.05$).

Group	Insulin /(mlU/L)	Insulin Sensitivity Index	Insulin Resistance Index
K	11.18 ± 2.12[a]	-4.50 ± 0.32[a]	4.01 ± 1.15[a]
KL	11.64 ± 2.13[a]	-4.32 ± 0.34[a]	3.33 ± 1.19[a]
M	7.84 ± 1.78[b]	-4.38 ± 0.22[a]	3.55 ± 0.80[a]
ML	9.87 ± 1.68[c]	-4.01 ± 0.43[b]	2.45 ± 0.60[b]
ME	11.12 ± 1.74[a]	-4.56 ± 0.44[a]	4.24 ± 1.95[a]
MG	11.17 ± 1.44[a]	-4.43 ± 0.36[a]	3.76 ± 1.41[a]

Table 8: *Insulin content, insulin sensitivity index and resistance index.*

Note: At the end of the experiment, the insulin content, insulin sensitivity index and resistance index of mice in different groups were expressed as mean ± SD (n = 3), and the same column of data was statistically (P < 0.05) significant in different lowercase letters.

The insulin sensitivity index of the model quinoa starch group was significantly higher than that of the other groups, while the resistance index was significantly lower than the other groups (P < 0.05), indicating that quinoa starch feed could significantly improve the action of unit insulin in breaking down glucose and promote the treating outcomes of insulin resistance.

Effect of quinoa starch on serum lipids in mice

Pre-diabetes and its pathological manifestations are usually accompanied by dyslipidemia. This test examined the serum lipid levels in mice, and the results are depicted in figure 8.

As depicted in figure 8, after 4 weeks of feeding, the serum levels of TG, CHO, and LDL-C in the model group were significantly higher than in the blank group (P < 0.05), while the HDL-C content was significantly lower than in the normal group (P < 0.05). Compared with the model group, the contents of serum TG (decreased by 23.49%, 8.58%, 17.27%, respectively), CHO (decreased by 27.12%, 18.44%, 31.01%, respectively), and LDL-C (decreased by 33.27%, 34.05%, 41.17%, respectively) significantly decreased in the model quinoa starch, metformin, and glimepiride groups. In contrast to the model group, the HDL-C content significantly increased in the model quinoa starch group (increased by 55.63%) than the metformin group (increased by 16.75%) and glimepiride group (increased by 28.42%) (P < 0.05). Additionally, there was no difference in the TG, CHO, LDL- C, and HDL-C contents between the blank quinoa starch and blank groups. This result demonstrated that quinoa starch could significantly improve hyperlipidemia.

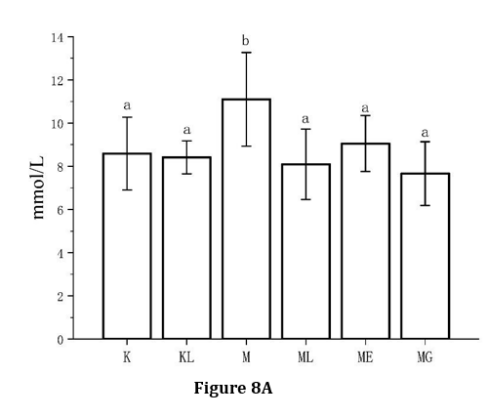

Figure 8A

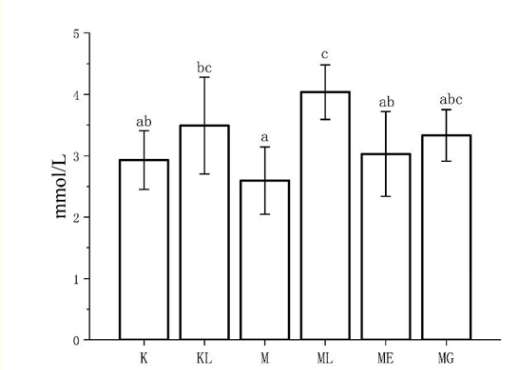

Figure 8B

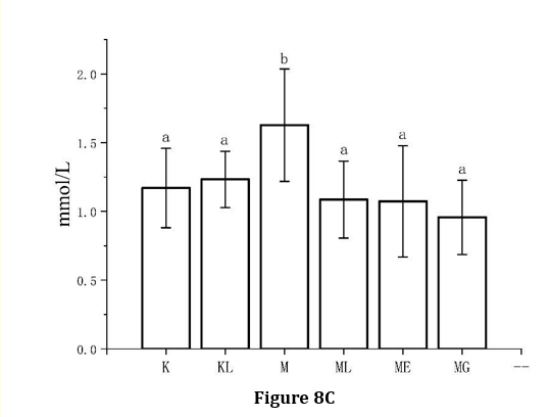

Figure 8C

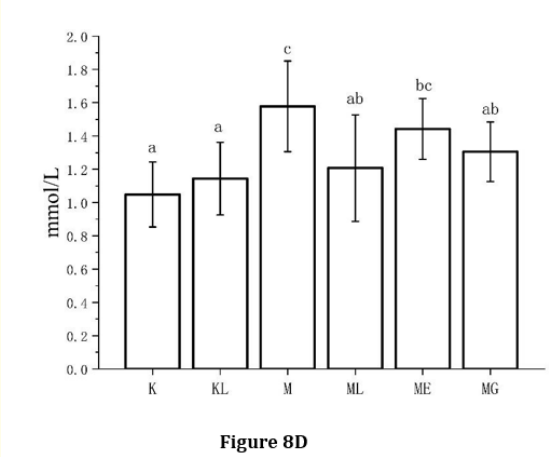

Figure 8D

Figure 8: *8A: Cholesterol (CHO); 8B: High-density lipoprotein cholesterol (HDL-C); 8C: Low-density lipoprotein cholesterol (LDL-C); 8D: Triglycerides (TG), statistically ($P < 0.05$) significant differences in different lowercase letters for different grouping data of the same index.*

Conclusion

The *in vitro* digestion and diabetic mice tests concluded that the blood glucose content of the quinoa starch group significantly decreased during rapid absorption and most of the starch did not participate in the digestion.

In vivo tests demonstrated that quinoa starch could maintain blood glucose stability, promote insulin resistance, improve hyperlipidemia, and enhance food utilization. The above results suggest that SDS is beneficial for disease stabilization during the dietary management of metabolic disorders, such as diabetes mellitus and hyperlipidemia. RS cannot be digested and absorbed in the small intestine, but it can be fermented to produce short-chain fatty acids and gases in the colon, which could relieve diabetic symptoms, protect the kidney, control body weight, and reduce the incidence of colon cancer.

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Conflict of Interest

The authors have no competing interests to declare.

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