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Is there any place for resistant starch, as alimentary prebiotic, for patients with type 2 diabetes?

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ABSTRACT

Objective: The aim of the present study was to determine effects of Resistant Starch (RS2) on metabolic parameters and inflammation in women with type 2 diabetes (T2DM).
Methods: In this randomized controlled clinical trial, 60 females with T2DM were divided into intervention ($n = 28$) and placebo groups ($n = 32$). They received 10 g/d RS2 or placebo for 8 weeks, respectively. Fasting blood sugar (FBS), glycated hemoglobin (HbA1c), lipid profile, high-sensitive C-reactive protein (hs-CRP), interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α) were measured at baseline and at the end of the trial. Paired *t* test, unpaired *t*-test and ANCOVA were used to compare the quantitative variables. The data were analyzed using SPSS software version 13.0.
Results: RS2 decreased HbA1c (−0.3%, −3.6%), TNF-α (−3.4 pg/mL, −18.9%), triglyceride (−33.4 mg/dL, −15.4%), and it increased HDL-c (+9.4 mg/dL, +24.6%) significantly compared with the placebo group ($p < 0.05$). Changes in FBS, total cholesterol, low-density lipoprotein, hs-CRP and IL-6 were not significant in the RS2 group compared with the control group. RS2 can improve glycemic status, inflammatory markers and lipid profile in women with T2DM.
Conclusions: Although findings of the present study indicated positive effects of RS2 on inflammation and metabolic parameters, more studies are needed to confirm efficacy of RS2 as an adjunct therapy in diabetes.

1. Introduction

Inflammation is one of the main mechanisms in the development of diabetes mellitus (T2DM). Increased proinflammatory factors, including Interleukin-6 (IL-6), Tumor necrosis factor-alpha (TNF-α) and high-sensitivity C-reactive protein (hs-CRP), play important roles in beta cell dysfunction and insulin resistance. Moreover, inflammation is a linkage between T2DM and other metabolic dysfunctions such as lipid disorders and increased oxidative stress which trigger diabetes complications.[1] Recently, the roles of intestinal microbiota in inflammation and metabolic dysfunction have attracted a great deal of attention.[2]

Resistant Starch (RS) is a kind of starch which escapes digestion in the stomach and small intestine. RS is fermented and utilized by the microbiota in the large intestine.[3] RS is classified into five subtypes (RS1–RS5).[4] RS2, also known as high-amylose maize starch, is uncooked starch that has a B- or C-type polymorph.[5] High-amylose maize starch (Hi-Maize) is the best-characterized type of RS. It is naturally found in raw potato, green banana, some legumes and high amylose starches. RS2 passes undigested to the large intestine where it can be used as a substrate for microbial fermentation.[6] Colonic fermentation results in raising some bacterial growth including Lactobacilli bacteria and Bifidobacteriaceae family which have anti-inflammatory properties.[6,7] Short chain fatty acids (SCFAs) such as butyrate, propionate and acetate are other productsof fermentation. Butyrate is the main SCFA which is produced from RS fermentation and it acts as an anti-inflammatory factor. Moreover, RS can produce more butyrate compared with other prebiotics.[7–9] Due to its aforementioned characteristics, RS can be considered as a prebiotic.[2]

Fiber intake among Iranian subjects with diabetes mellitus is lower (16.7 g/d) than the recommended fiber intake for patients

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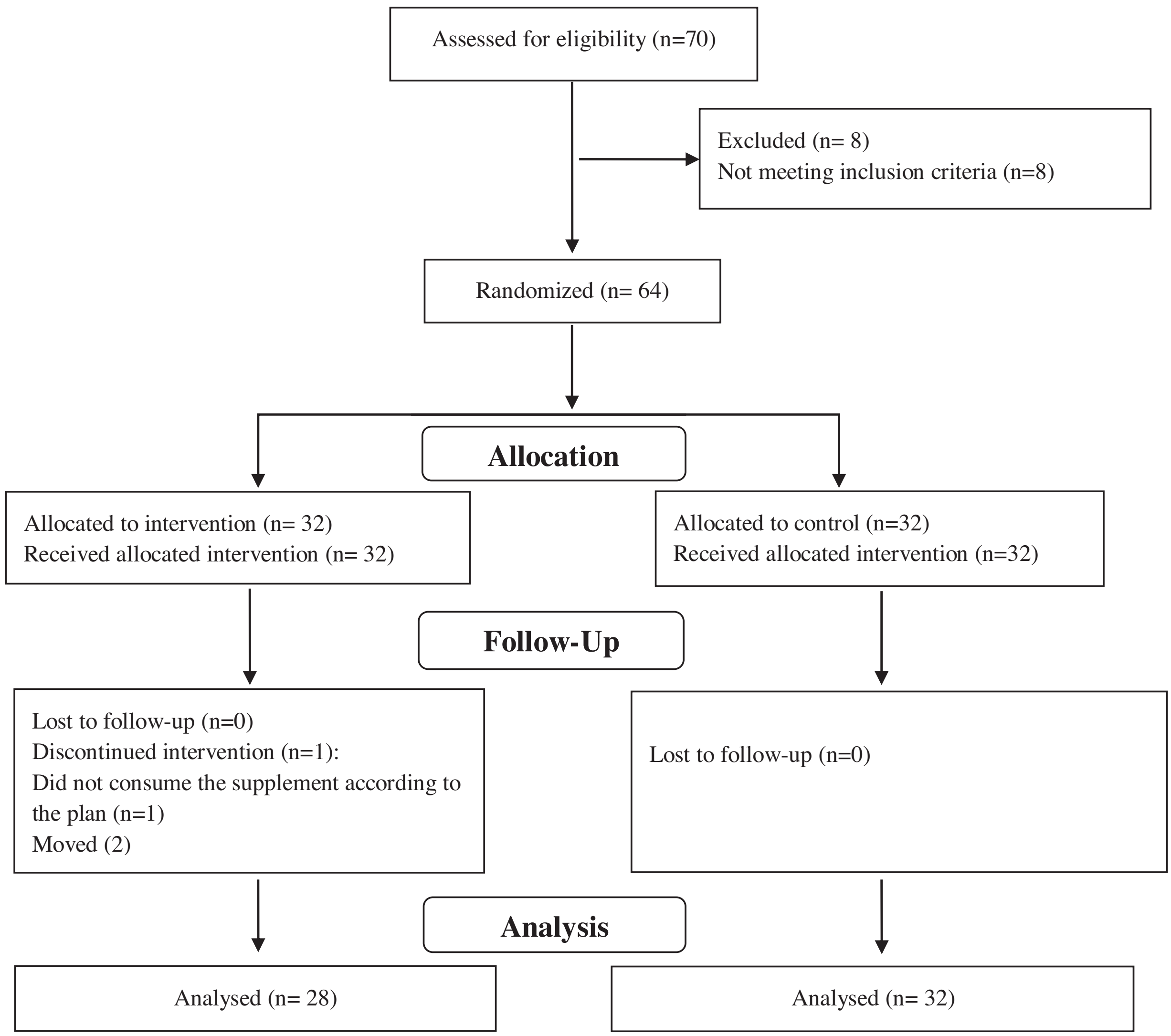

Fig. 1. Flow chart of the study.

with diabetes (20–35 g/d).[10] It seems that RS can be proposed as a dietary fiber supplement with prebiotic properties for T2DM. There are limited studies with diverse results on the effects of resistant starch on biochemical parameters.[11–14] Nilson et al. indicated that supplementation with barley-kernel bread reduced IL-6 level in healthy individuals.[15] Conversely, Johnson et al. reported that RS2 did not change inflammatory markers in patients with metabolic syndrome.[12] Mitra et al. indicated that consumption of rice containing RS2 in patients with T2DM decreased fasting glucose level after 3 months.[16] Conversely, based on Penn et al. study 12 g/d

Table 1
Baseline characteristics and dietary intakes of the study participants.

Variables	Maltodextrin group (*n* = 32)	Resistant starch group (RS2) (*n* = 28)
Age (y) (range)	49.6 (8.4) (34–60)	49.5 (8.0) (33–65)
Menopausal status (*n* (%))		
Pre-menopausal	9 (28.2)	8 (28.5)
Post-menopausal	23 (71.8)	20 (71.5)
Diabetes duration (y)	5.2(4.4)	7.5(5.9)
Weight (kg)	71.8 (3.5)	74.2 (4.3)
Height (cm)	152.6 (6.4)	153.3 (5.2)
Body mass index (kg/m^2)	30.8 (5.2)	31.5 (4.5)
Waist circumference (cm)	99.8 (4.6)	100.5 (10.5)
Hip circumference (cm)	105.9 (13.9)	108.6 (10.7)
Waist to hip circumference (cm)	0.94 (0.07)	0.92 (0.06)
Systolic blood pressure (mmHg)	131.8 (15.1)	131.9 (17.3)
Diastolic blood pressure (mmHg)	85.6 (7.8)	86.6 (11.5)
Metformin, 500 mg (tablets/d)	2.6 (1.1)	2.8 (1.1)
Glibenclamide, 5 mg (tablets/d)	1.7 (1.2)	2.3 (1.4)

Note: Data are presented as mean (SD), with the exception of menopausal status, which is presented as number (percent).