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# Does Resistant Starch (RS) affects gut health and BMI in obese adults?

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**Abstract**

***Objective:***

Impact evaluation of RS supplementation on anthropometric, biochemical, and lifestyle factors along with establishment of gut micro flora with respect to *Bifidobacteria*, *Lactic Acid Bacteria* and *Enterobacteria* in adult grade III obese subjects.

Methods: Randomized control trial was undertaken with 60 obese adults wherein experimental group (n=30) was supplemented with 10 g RS in a sachet daily for a period 8 weeks and control group subjects (n=30) did not receive the supplement. The subjects were assessed for their gut microbiodata. Blood samples were estimated for leptin levels before and after supplementation. ***Results:*** RS supplementation in obese adults (mean BMI= 32.9 kg/$m^2$) significantly increased establishment of Bifidobacteria ($p < 0.01$) from 7.41 to 8.22 log CFU/g and lactobacilli from 4.77 to 6.96 log CFU/g and with a non significant reduction in the mean log counts of enteric pathogens. RS intake in obese adults resulted in a non significant reduction in WC, HC and systolic blood pressure by 1.2%, 1.9% and 0.5% respectively with no change in their BMI values. Subjects with higher leptin levels (>5µg/dl in males and >7µg/dl in females) showed significant reduction with 49% and 29% decrease in males and females respectively.

**Conclusions:** RS can be implicated as a promising tool for improving the gut health in the obese subjects However, no exclusive effect of RS supplementation on the weight, BMI, body fat and satiety levels of the subjects.

***Keywords:*** *Obesity, Resistant Starch, Bifidobacteria, LAB, leptin levels, gut microflora*

## Introduction

Obesity has reached its epidemic proportions globally with more than one billion adult population overweight and about 300 million of them clinically obese. It poses a major risk for serious diet-related chronic diseases, including Type 2 diabetes, cardiovascular disease, hypertension and stroke, and certain forms of cancer [1]. Modest weight reductions of 5% to 10% of body weight can decrease blood pressure, total blood cholesterol, improve glucose tolerance in diabetic patients and those

with impaired glucose tolerance prone to develop diabetes, and reduce the severity of obstructive sleep apnea. Given the rise in the prevalence of obesity, even these small changes can be important to the nation's health [2]. The link between the barrier function of colonic micro flora and susceptibility to disease is an area of great interest. The correlation of gut health and obesity is still in the infancy stage of research based on the antiquated way of assessing organisms in the GI tract (microbiota). Probiotic bacteria like *Bifidobacterium* and *Lactic Acid Bacteria (LAB)* help in improving health by improving immunity, by decreasing toxic elements and harmful bacteria in the gut and inducing hypocholestromic effect[3]. Prebiotics are defined as non-digestible food ingredient that beneficially affects the host by selectively stimulating the growth and activity of one or a limited number of bacteria in the colon [4]. The Resistant Starch (RS), a dietary component has been recently recognized as a potential prebiotic and an important factor in the bacterial ecology of human gut [5]. RS is defined as the sum of starch and products of starch degradation not absorbed in small intestine of healthy individuals [6]. RS helps in the fermentation of complex carbohydrates by the large-bowel micro flora which is important for human health. Studies are required to determine whether the stimulation of *Bifidobacteria* and LAB in adult human subjects through the ingestion of RS leads to any health benefit above and beyond that of the carbohydrate itself. Reduction of the energy content of the diet with non-digestible carbohydrates is considered a possible way to fight obesity. Adding RS to the diet also reduces the dietary energy density. However, the effect of non-digestible carbohydrates such as RS on obesity has not been thoroughly investigated and any food/food ingredient that can increase satiety may play a vital role in weight-loss diets. Some studies have examined the potential of RS as a satiety agent and that long-term consumption of RS was more satiating than glucose though the effect was small. Future studies need to objectively measure satiety using standardized test meals matched for macronutrients and fiber content but containing different levels of RS[7]. Therefore, the present study was undertaken with the objective of determining the impact of Resistant Starch supplementation on anthropometric, biochemical, and lifestyle factors along with establishment of gut micro flora with respect to *Bifidobacteria*, *Lactic Acid Bacteria* and *Enterobacteria* in adult grade III obese subjects.

## Methodology

**Selection of the subjects:** Obese adults aged 30-60 years was enrolled from free living population of the urban Vadodara. Written informed consent was obtained from the study participants. The inclusion criteria for selecting the patients were: BMI ≥25 kg/m². age group between 30 -60 years, those suffering from any chronic disease, severe hypertension, diabetes mellitus, history of renal disorder, history of gout and arthritis, subjects on weight reducing drugs, subjects on anti-thyroid drugs, or on long term antibiotics or those who have undergone surgery in last 6 months were excluded from the study.

**Intervention trials with Resistant Starch supplementation**

Based on their informed consent, 60 subjects fulfilling the inclusion and exclusion criteria were selected purposively and were randomly divided into control (N=30) and experimental groups (N=30). The experimental group was supplemented with 10 g of resistant starch daily for a period of 60 days. The investigator

supplied the packets of resistant starch every 15 days to ensure maximum compliance.

**Impact Evaluation of the Resistant Starch supplementation:**

Blood samples were collected and were estimated using standard methods after 60 days of intervention to check the parameters similar to the baseline for both the groups.

**Collection of baseline data**

Baseline information was collected with respect to anthropometric measurements, socio demographic profile, general habits, dietary intake, physical activity pattern, clinical and medical history and morbidity profile using pretested semi structured questionnaire. Dietary intake of all the subjects was gathered by using 24 hour dietary recall and food frequency method. The activity pattern of the adults was assessed using Global Physical Activity Questionnaire[8]. For the morbidity profile, subjects were asked to report various illness experienced in the past month and the recurring problems with respect to gastrointestinal problems (vomiting, diarrhoea, abdominal pain, flatulence and constipation), any dental problems like cavities, general illnesses like cough, cold, fever, skin infection and any recurrent incidences of joint pain or fracture to know the incidence of osteoporosis or osteoarthritis and any other major illness suffered by the subjects using the checklist.

Anthropometric measurements like, height was measured using flexible measuring tape and weight of subjects was measured on a pre-standardized weighing scale nearest to 100KG. BMI was calculated using the formulae weight (kg)/height (m2). Waist circumference (WC) and hip circumference (HC) were measured using flexible measuring tape. To measure waist circumference, subjects were made to stand straight and with their stomach relaxed. The tape was positioned midway between the top of the hip bone and the bottom of the rib cage. Hip circumference was measured 24 inches below navel level, tape was wrapped and circumference measured. The percent body fat of subjects was assessed using the Omron HBF 360 fat analyzer. The subject is made to grip the machine handles for about 7 seconds. The required information regarding height, weight, age and gender is fed in the machine. The body fat analyzer uses the electrical impedance to measure the body fat against the lean body weight (American council on exercise**)**. The diastolic and systolic blood pressures were assessed using a sphygmomanometer. The subject was made to sit down quietly for 5 minutes. The bare arm of the subject was supported and positioned at heart level. A cuff of suitable size was positioned over the upper arm with the bladder of the cuff positioned over the brachial artery. The cuff was inflated till the radial pulse disappears and deflated again slowly releasing the pressure. The pressure reading at which a pounding sound is heard when blood flows again in the artery is the Systolic Blood Pressure. The cuff pressure is further released until the sound can no longer be heard. This is recorded as the diastolic blood pressure.

**Collection of test samples**

Fecal and blood samples were collected at baseline and after intervention. Fecal samples were collected for determining microbial counts in terms of *Bifidobacteria, LAB* and *enteric pathogen*. Subjects were advised not to alter their usual calorie intakes and physical activity pattern and were asked to document any unusual symptoms or side effects. Blood samples were estimated for Serum leptin levels before and after supplementation of RS. The

compliance of the supplementation was monitored regularly by the research investigator.

### Test methods

### Fecal bacteria enumeration

The gut micro flora was determined with respect to the microorganisms- *Bifidobacteria, lactic acid bacteria* and *enteric pathogens*. Fecal samples were collected in an air tight sterile container kept with cold packs and within 3 hours after the collection. The serial dilutions of 1 gram of fecal samples were made up from $10^{-2}$ to $10^{-8}$ dilutions (14).The media for *bifidobacteria* and *Enteric pathogens* was prepared in laboratory using the dehydrated *bifidobacterium* agar and EMB agar respectively procured from Hi Media and was autoclaved at $121^{0}$C for 15 minutes (15). The prepared media were poured into sterile petri plates and was allowed to set (pour plate technique). The petri plates were kept inside laminar flow under UV light prior to using them for serial dilution. These plates were then kept inside the incubator along with the plates of Enterobacteria at 37°C for 48 hours. After this the colonies were counted and reported the organisms were enumerated using a colony counter and the numbers of colonies were reported as log values per gram of sample (log CFU/g).

### Blood collection and analysis

After the overnight fast, venous samples was collected in clean, sterilized vaccutainers and allowed to stand at room temperature for 15 minutes. Serum was separated and analyzed for serum hormone Leptin. The serum leptin level was estimated using the Radio Immuno Assay (RIA) method. 0.1 ml of serum sample was pipette in a test tube along with 0.1 ml of standards in standard tubes. 0.1 ml of anti mouse leptin antibody was added to all the tubes and mixed. 0.1 ml of 125 I Leptin Isotope was added to all the tubes. The solution was mixed well and incubated for 1 hour 30 minutes. 500µl precipating antibodies was added to the test tubes, mixed well and incubated for another 30 minutes. All the test tubes were incubated for 15-30 minutes at 3000 rpm. The test tubes were decanted and read on Gamma counter for one minute to obtain counts per minute (CPM). A graph was plot of standard values with CPM value to obtain the patient's leptin values.

### Statutory clearances

The Medical Ethics committee of the Foods and Nutrition Department, The M.S. University of Baroda approved the study proposal. Written consent was obtained from the participants who agreed to give baseline information through questionnaire and give sample of blood and stool for biochemical and microbiological analysis respectively

### Statistical analysis

The data was entered in a computer using an excel spreadsheet. The data was cleaned and verified and subjected to appropriate statistical analysis Data analysis was performed using the Statistical Package for the Social Sciences (SPSS19.0 version, SPSS Inc., Chicago,). Paired t test was performed to observe the effect of RS supplementation. Pearsons corelation [9] was done to find out statistical significance between the groups. The significance levels were set at 5% by two sided tests. Student t test was performed for the comparison between control and experimental group. Mean and standard deviation were calculated. Frequency distribution and percentages were calculated wherever applicable. All tests were considered significant at $P<0.05$.

## Results and Discussion

### Baseline Characteristics

The background information of the subjects revealed that almost 61% of the subjects had heredity as a causative factor for obesity, at the same time in 67% subjects the obesity was recognized in the past 10 years and family history of NCD's was prevalent in 87% of the subjects, indicating the role of hereditary and lifestyle changes as predominating factor in the etiology of obesity. Separating these lifestyle factors from genetic ones is often difficult. Still, growing evidence points to heredity as a strong determining factor of obesity. In one study of adults who were adopted as children, researchers found that the subjects' adult weights were closer to their biological parents' weights than their adoptive parents'. The environment provided by the adoptive family apparently had less influence on the development of obesity than the person's genetic makeup. Time trends in eating away from home roughly parallel the national time trends in obesity prevalence[10,11].

***Anthropometric, Biochemical and Physical activity profile***

The anthropometric data (Table 1) revealed that subjects had a mean BMI of 31.9 kg/m$^2$ with mean WC of 105.1 cm and mean WHR 0.95. According to the WHO criteria for WHR, 97% males and 80% females were at a risk of CVD's. The mean body fat percent of males and females was 18.6% and 27.7% respectively. The mean blood pressure of the subjects was 126/83 mmHg but majority of the subjects were categorized as pre hypertensive (75% according to systolic BP and 50% according to diastolic BP). In this study prevalence of hypertension was low as subjects who had severe hypertension were excluded from the study. Although the study has reported a positive linear relationship between adiposity and blood pressure is well documented [12,3], with substantial risks of hypertension among the obese being reported.

The present study also revealed that low physical activity pattern was adopted by almost 57% of the subjects with 73% of the male subjects following a sedentary lifestyle. Even in the absence of direct evidence for a causal link between physical activity and obesity prevention, there is indirect evidence to indicate that energy expenditure requirements have reduced in many facets of daily life over the past 50 years due to urbanization[15]. Several intervention studies have evaluated the impact of increased physical activity or decreased sedentary behavior as part of multifactorial interventions on prevention of weight/fatness gains or remission of overweight in clinical and community-based settings, with mixed results [16].

The leptin levels of obese subjects at baseline ranged from 2.9 to 13.6 µg/L in males and 2.8 to 15.6 µg/L in females. For both males and females the mean values were on higher side of the reference values (Table 1). Although there is greater awareness about adiposity and higher circulating leptin levels, our study has also shown a positive significant relation (r=0.56) between circulating leptin levels and enterobacteria. The possible mechanism for positive correlation can be higher BMI which is associated with increased leptin levels which can be further associated to higher establishment of enteric bacteria.

**Dietary Intake Assessment**

Dietary pattern of the subjects revealed that almost 36% of the subjects frequently (daily to 2-3 times a week) consumed outside foods (at canteen, fast food joints, and restaurants). This pattern was observed more in males (53%) than in females (20%). Hence the dual effect of having predisposition to obesity genes and

**Table 1: Mean anthropometric, biophysical, biochemical profile of the subjects under study**

| Variable | Normal Range | Total (N=60) | Male(N=30) | Female (N=30) | 't' test |
|---|---|---|---|---|---|
| | | Mean ±SD | Mean± SD | Mean ± SD | |
| Height (mt) | | 1.61±0.10 | 1.69±0.07 | 1.54±0.06 | 9.33*** |
| Weight (kg) | | 83.36±15.01 | 91.62±13.00 | 75.11±12.09 | 5.07*** |
| BMI (kg/m2) | 18-22.9 Kg/$m^2$ | 31.90±4.52 | 31.97±4.30 | 31.84±4.81 | 0.11 NS |
| WC (cm) | Female <80 cm<br>Male <90 cm | 105.08±11.24 | 110.70±10.20 | 99.47±9.04 | 4.44*** |
| HP (cm) | | 111.19±8.55 | 110.42±6.80 | 111.96±10.07 | 0.69 NS |
| WHR | Male =1<br>Female =0.8 | 0.95±0.08 | 1.00±0.05 | 0.89±0.06 | 7.92*** |
| % Body fat | Male -14-17%<br>Female -21-24% | 22.70±19.50 | 18.66±17.66 | 27.73±20.35 | 6.99*** |
| Systolic BP (mmHg) | <120mmHg | 126.62±10.83 | 129.10±11.12 | 123.62±10.83 | 2.24* |
| Diastolic BP (mmHg) | <80 mmHg | 83.50±8.25 | 85.71±7.70 | 81.14±8.29 | 2.27** |
| Leptin (mcg/L) Range | Female 3.7-11µg/L<br>Male 2.0-5.6 µg/L | - | 5.60±3.12 (2.9-13.6) | 7.91±3.64 (2.8-15.6) | 2.15* |

increase in the energy dense foods may have lead to alarming rise in the prevalence of obesity. Cross-sectional and longitudinal data on self-reported "fast food" restaurant use per se and consumption of foods frequently sold at "fast food" restaurants have been shown to be positively associated with body weight. [17,18, 19]

The nutrient intake of the obese subjects was compared with the RDA[20] (Table 2), revealed that 36% energy was contributed by fat intake which again indicates a great risk for development of hyperlipidemic and CVD's. The intake of Total Dietary Fiber (TDF) of the subjects under study was almost 52% and 64% lower than the RDA in males and females respectively. Low TDF and high fat food are known to affect the gut health in terms of higher colonization of enteric pathogens resulting in endotoxemia which is now being revealed as a causative factor for several metabolic diseases. Recent studies on rat models demonstrated that mice fed a high-fat diet for as short a term as 2 to 4 weeks, exhibited a significant increase in plasma lipopolysaccharide LPS which is a constituent of Gram negative bacteria present in the gut microbiota[21]. This can be considered as a "metabolic endotoxemia". It was demonstrated that high-fat diet feeding changed gut microbiota profile. Indeed, the population levels

**Table 2: Nutrient intake of Subjects under study.**

| Nutrients | Reference RDA | Male (N=31) Mean ± SD | % RDA Difference | Females (N=29) Mean ± SD | % RDA difference | 't' test |
|---|---|---|---|---|---|---|
| Energy (kcal/d) | Male- 2320 Female- 1900 | 2432.06±491.99 | 5 | 1992.82±349.69 | 5 | 3.98*** |
| Fat (g/d) | Visible fat – Male- 25 Female- 20 | 97.28±21.67 | 62 | 82.71±16.85 | 38 | 2.90** |
| Protein (g/d) | Male- 60 Female- 55 | 63.49±15.36 | 6 | 50.86±8.56 | 8 | 3.93*** |
| CHO (g) | | 324.29±67.38 | 14 | 259.81±48.44 | 16 | 4.25*** |
| Total dietary Fiber (g/d) | TDF-30 g/day | 19.26±7.34 | 52 | 14.54±3.47 | 64 | 3.18** |
| Soluble Dietary Fibers (g/d) | | 4.94±1.79 | - | 3.79±0.96 | - | 3.11** |
| Insoluble Dietary Fibers (g/d) | | 14.32±5.70 | - | 10.76±2.65 | - | 3.10** |
| Iron (mg/d) | Male- 17 Female- 21 | 17.98±4.73 | 4 | 13.23±4.02 | 37 | 4.18*** |
| Vitamin-C (mg/d) | Male- 40 Female- 40 | 129.88±57.52 | 224 | 99.57±50.11 | 149 | 2.17* |
| Vitamin A (µg/d) | Male- 600 Female- 600 | 295.53±95.03 | 51 | 281.12±100.50 | 53 | 0.57 NS |
| β-Carotene (µg/d) | Male- 4800 Female- 4800 | 2388.98±2258.02 | 50 | 2215.17±1899.45 | 54 | 0.32 NS |
| Calcium (mg/d) | Male- 600 Female- 600 | 882.12±225.50 | 47 | 758.79±234.57 | 27 | 2.07* |

of *Bifidobacterium* spp. and *E. rectale/ Cl. Coccoides* group were significantly reduced in high fat fed animals versus mice receiving the standard high carbohydrate diet [21].

**Subject compliance**

RS was well tolerated by all the subjects who completed the study and no intolerance or adverse events were reported. Volunteer compliance was assessed by return of used sachets and by self-reported RS intake (by means of compliance sheet), which indicated good compliance

**Impact of Resistant Starch supplementation on anthropometric, biophysical, biochemical parameters**

RS supplementation did not bring about any significant changes in the anthropometric measurements, biophysical and biochemical parameters. It can be seen that there was no significant difference in the mean values of BMI, WC and WHR of the experimental group post RS supplementation (fig 1). Also, no difference was found in the anthropometric values between the groups before and after RS consumption

**Impact of Resistant Starch supplementation on gut microflora of obese participants.**

The experimental group was supplemented with 10g RS and its effect were studied after a period of 8 weeks. RS supplementation resulted in significant increase ($p<0.001$) in the establishment of Lactic Acid Bacteria by 17% in the experimental group with no significant difference seen in the decrease of enteric pathogens. 10 gm of RS supplementation did not show reduction in the enteric bacteria because of high grade of obesity as well as high fat intake in the diet.

**Fig: 1 Impact of RS supplementation on anthropometric profile of obese subjects**

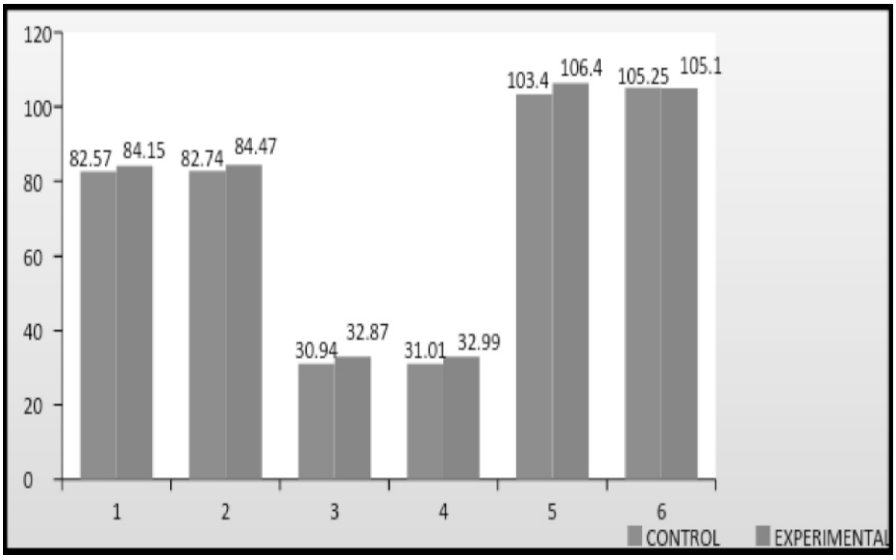


**Fig 2: Impact of Resistant Starch supplementation on gut microflora of obese participants.**

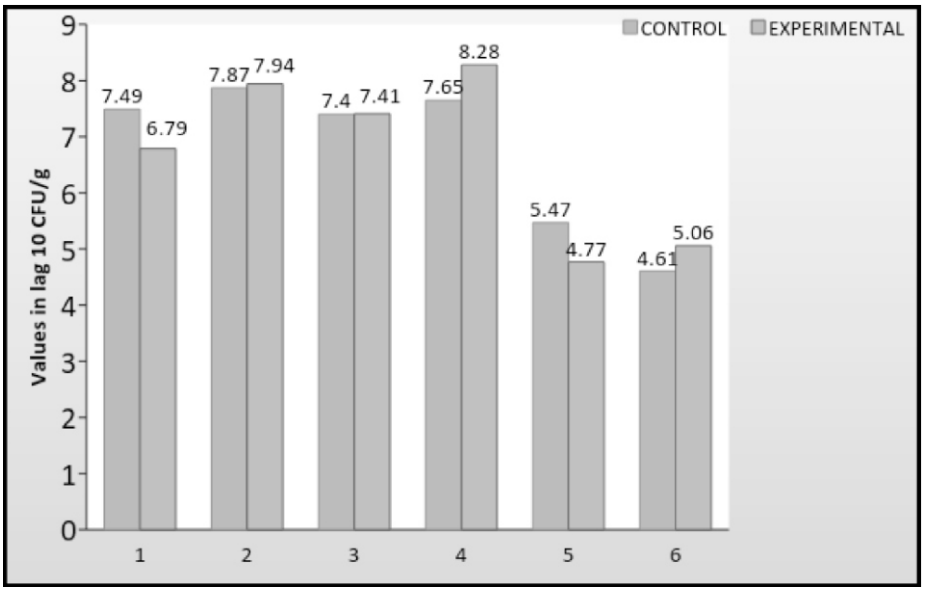
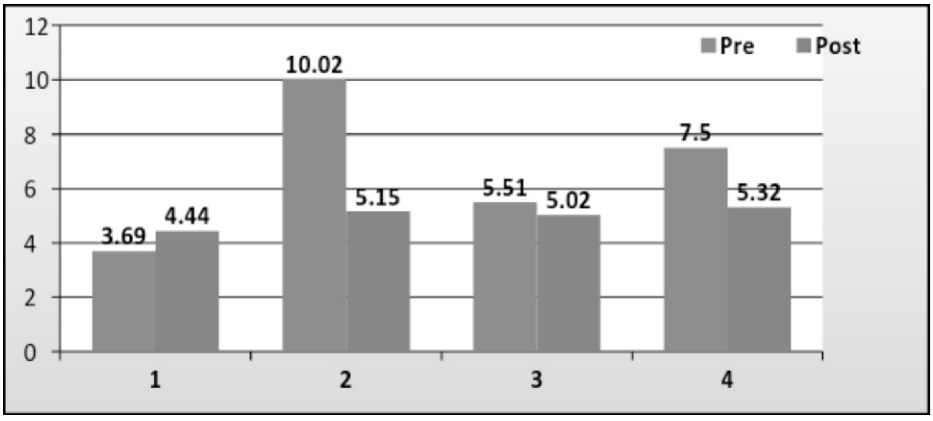



**Fig 3: Changes in leptin levels of subjects with initial higher levels post supplementation**




**Summary and Conclusion**

Thus the study has clearly demonstrated a positive impact of RS intake on the lactic acid bacteria establishment of mild and moderately obese individuals but an inverse effect in the severely obese individuals. Also 10 gm of RS supplementation failed to bring about reduction in food intake and anthropometric parameters of the studied human obese subjects, which is reported in the animal model studies.

Hence RS can be implicated as a promising tool for improving the gut health in the obese

subjects However, no exclusive effect of RS supplementation was observed on the weight, BMI, body fat and satiety levels of the subjects under study. Thus studies may be undertaken with higher amounts of RS supplementation and the effect may be observed on other gut incretins which play a role in the appetite control centres in the hypothalamus.

**Conflict of Interest**

The authors certify that they have NO affiliations with or involvement in any organization or entity with any financial or non-financial interest in the subject matter or materials discussed in this manuscript.

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