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***Research Article***

# METABOLIC EFFECT OF FOS (FRUCTOOLIGOSACCHARIDE) IN TERMS OF GUT INCRETIN (GLP-1) GUT MICROFLORA AND WEIGHT REDUCTION IN OBESE ADULTS

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**ABSTRACT:** In the recent years, obesity has increased beyond imagination. Appropriate dietary strategies which have the potential for weight loss demand patience and strong determination on part of the individual, however inclusion of functional foods like FOS that modulate gut hormones have a promising role in weight management. **Methods:** A randomized double-blind placebo-controlled trial was used as the study design wherein 65 obese adults were divided into experimental group (which was given 12 g of FOS) and a placebo group (which was fed with 12 g dextrose). The subjects were given the supplements daily for 12 week period. Their plasma samples were anlaysed for GLP-1 and microbial count in fecal samples were determined in terms of *lactic acid bacteria, bifidobacteria* and enteric pathogens. Hunger scores, dietary intake, and anthropometric parameters were assessed using standard techniques. **Results:** FOS supplementation resulted in improved plasma GLP-1 level by 17.0%. Significant improvement was observed in hunger score by 3.15% ($p<0.05$) along with reduction in dietary intake of energy (kcal) by 8%, carbohydrate (g) by 8%, protein (g) by 6% and fat (g) by 2%. Further, reductions were observed in total body weight (kg), BMI, % body fat and waist circumference (cm) levels by 4%, 1.06%, 4% and 1.66% respectively ($p<0.001$, $p<0.001$, $p<0.001$, $p<0.05$). The mean log counts of beneficial gut microbiota i.e. *lactic acid bacteria* and *bifidobacteria* increased significantly by 14 % and 10 % respectively along with 20% reduction in *enteric pathogen.* **Conclusion:** Daily intake of 12 gm FOS for 12 weeks helps in improving gut health and weight loss through increased satiety in obese individuals.

**Key words:** FOS, obesity, GLP-1, *lactic acid bacteria*, *bifidobacteria*, *enteric pathogen*.

## INTRODUCTION

Obesity is considered as cluster of non-communicable diseases called 'New World Syndrome' creating an enormous socio economic and public health burden in developing countries. Recent studies from western populations have shown a plateau in the prevalence of both adult and childhood obesity during the last decade (Flegal et al, 2010, Lissner et al 2010, Ogden et al 2010). However, the problem is of a larger magnitude in developing countries like India where a significant proportion of the population belongs to younger age group (Adlakha A, 2011). In recent years obesity is known to reach pandemic proportions. WHO's projections indicated that globally in 2005, approximately 1.6 billion adults were overweight and at least 400 million adults were obese. WHO further projects that by 2015, approximately 2.3 billion adults will be overweight and more than 700 million will be obese. Animal and human data have suggested that the composition of the gut microflora may be an important mediator of the risk of obesity (Delzenne and Canni, 2011). Several studies reported that the gut microbiota differs at phylum level depending on weight status (Eckburg et al, 2005, Turnbaugh et al, 2006). Another study reported that fecal gut microbiota in 12 obese subjects participating in a weight-loss program by consuming restricted diets for a year. Following weight loss, the proportion of *Bacteroidetes* increased while the number of *Firmicutes* reciprocally decreased (Ley et al, 2006).

Inulin type fructans are well studied and clearly effective in humans and animal models to stimulate growth of health promoting species belonging to *bifidobacterium* and *lactobacillus* and modulating gut hormone GLP-1 (Macfarlane et al, 2006 and Flamm et al, 2001). A study reported that oligofructose feeding (20g/d) significantly increased plasma GLP-1 after mixed meal (Piche et al, 2003). Furthermore, a study demonstrated that in healthy humans, feeding of 16g/day FOS promoted satiety followed breakfast and dinner and reduced hunger after dinner. This was accompanied by a significant 10% lower total energy intake (Canni et al, 2006).

Many researchers have reported that prebiotic supplementation increases gut hormone signaling (GLP-1, PYY), in a study rats fed with prebiotic (resistant starch) had increased GLP-1 and PYY expression (Cani et al, 2009).
A need was felt to study the relevance of these studies in Indian context. Therefore, the study was undertaken to evaluate metabolic effect of FOS in terms of gut incretin, gut microflora and anthropometric parameters of obese adults.

## MATERIALS AND METHODS

The experimental design of the study is depicted in the Figure1

**Locale of the study:** Industry located at Vadodara, Gujarat was conveniently selected after obtaining permission obtained from the administration department.

***Study design:*** Double blind placebo control trial

**Selection of the subjects*:*** Sixty five obese (BMI> 25 kg/m$^2$<31kg/m$^2$) aged 25-55 yrs were enrolled and randomly allocated to receive either FOS supplementation or placebo daily for 12 weeks. Subjects with Presence of diabetes mellitus, cardiovascular disorder, thyroid hormone disorder, valve replacement surgery, gastric surgery or perforation, renal disorder, locomotor disorder, cancer / aids, psychological disorder, heavy physical activity were excluded from the study. Duly filled informed consent form was obtained prior to the enrollment of the subjects.

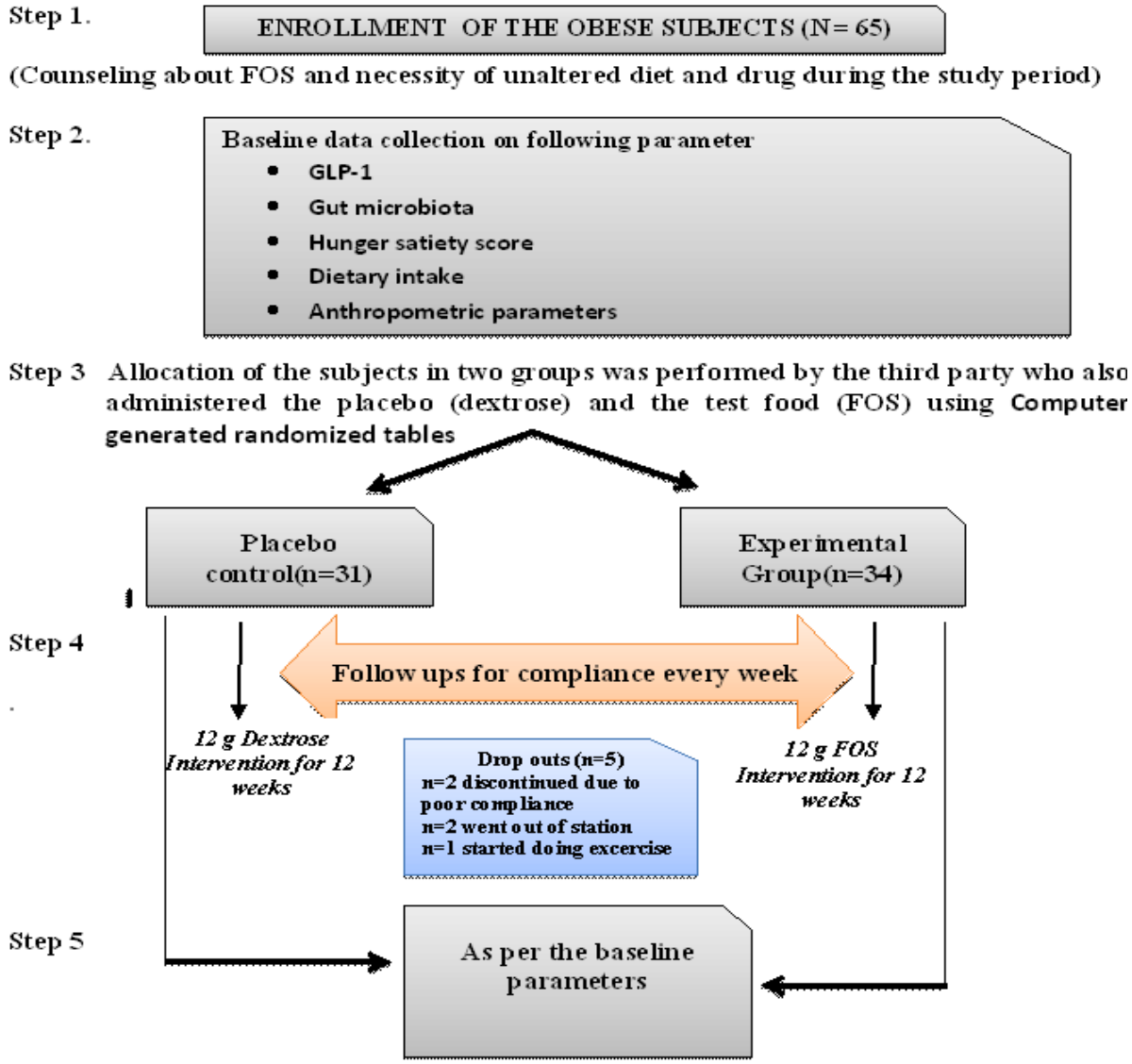


**Figure 1: Flow Chart Depicting the Experimental Design of the Study**

**Randomization of subjects and Mode of Intervention of study food and placebo**

All the subjects were randomly divided into two groups and codes were allocated to each group (1and2). Color codes were given to test food and placebo (orange, green) and then codes were given to these colors same as codes of the study group i.e. 1 and 2 (orange-1, green-2) by the third party, then test food and the placebo were distributed among subjects so that color code 1 will go to group1 and 2 will go to group 2. Twelve grams of FOS (study food) and dextrose (placebo) was given in powder form each packed in auto sealed sachets. The subjects were asked to incorporate FOS/dextrose in to water along with the breakfast/lunch for period of 12 weeks.

**Study food and placebo**

The FOS used as study food for the intervention was food grade FOS (BeneoRaftilineP95 BAG 25 kg, Orafti, Belgium) was procured in a pack of 25 kgs from Brenntag Ingredients India Pvt. Ltd, Mumbai whereas, dextrose for the placebo.

**Determination of physical, biophysical, biochemical and microbiological parameters**

Anthropometric measurements included measurement of weight, height, waist circumference, hip circumference.

**Weight:** A digital weighing scale to the nearest 100 g was used to measure weight. Scale was calibrated using standard weights.

**Height:** Height meter was used to measure the height of the subjects. Two consecutive reading were taken.

**Waist and Hip Circumference**

The circumference was recorded using the constant tension, spring loaded tape at the narrowest part of the abdomen between the ribs and iliac crest. The measurement was taken to the nearest 0.1 cm at the end of a normal expiration, without the tape compressing the skin.

**Waist- Hip Ratio (WHR):** This ratio gives an idea of central adiposity.

It was computed as-

$$WHR = \frac{waist\ circumference\ (cm)}{hip\ circumference\ (cm)}$$

Cut off used for WHR were those laid down by WHO Asia Pacific criterion for abdominal obesity (WHO 2004)

**Percent body fat:** the percent body fat of the subjects was assessed using the Omron HBF 360 fat analyzer. The required information about height, weight, age and gender is fed in the machine. The subject is made to grip the machine handles for about 7 seconds. The body fat analyzer uses electrical impedance to measure the body fat against the lean body fat.

**Cut offs for body fat**

| Category | Percent body fat (Male) | Pe5rcent body fat (Female) |
|---|---|---|
| Fitness | 14-17% | 21-24% |
| Acceptable | 18-24% | 25-31% |
| Obese | ≥25% | ≥32% |

**(American council on exercise)**

**Body Mass Index (BMI):** According to Asia pacific classification 2004

**Dietary information:** Information on food intake was collected through Food frequency and 24 hr dietary recall intake after every 15 days for 3 months. The subjects were asked to provide details of all the major meals consumed by them, throughout the previous day, along with additional fruits, beverages, snacks, pickles, sweets, etc. the amount of the cooked food was converted into the raw food and the mean nutrient intake was calculated in terms of calories, proteins, fat, iron, total dietary fibers, β-carotene and vitamin C using Diet Soft package (Gurdeep Kaur AIIMS, 2007)

**Hunger satiety index:** The hunger scores were determined using satiety and hunger scale developed by Lisa Burgoon (1998)

**Determination of GLP-1**

Venous blood sample was collected in clean, sterilized vacuum containers and allowed to stand at room temperature for 15 minutes. Plasma was separated for GLP-1 analysis using ELISA kit method.

**Determination of microbiological parameters in fecal samples**

**Collection and storage of stool sample**

The subjects were given air tight sterile containers and the stool sample was transferred in ice boxes and carried to the laboratory where it was stored at appropriate temperature (-20°C) in deep freezer.

### Enumeration of bacteria

The samples were processed for enumeration of Bifidobacteria, *Lactic Acid Bacteria* (LAB) and Enteric Bacteria using standard serial dilution techniques FAO/WHO (1979). The media used for the enumeration of *Bifidobacterium* was *Bifidobacterium agar* supplied by HiMedia. The prepared media was autoclaved at $121^0$C for 15 minutes and then poured into sterile petri plates and was allowed to set. The enumeration of *Lactic acid bacteria* and enteric pathogen was done using ready-*made HiTouch Flexi plates* supplied by HiMedia Mumbai.

The plates with *Bifidobacterium* agar placed in the anaerobic jar with the gas packs (Hi Media) were incubated at $37^0$C for 48 hours. Flexi plates of *Lactic acid bacteria* were placed in a desiccator as it is a facultative anaerobe and those of enteric pathogen were directly placed in the incubator. After 48 hours of incubation the colonies were counted on colony counter (Cintex colony counter, Dadar Mumbai) and colonies that appeared in the range of 30 – 300 were converted in to log counts after multiplying with their dilution factors (Ramona et al 2001).

### Statistical Analysis

Data were analyzed using Microsoft office excel 2007 and Epi Info 3.3.2, 2005, Statistical Package for Social Sciences (SPSS 16.0 version), SPSS Inc., Chicago, IL, USA. Paired 't' test, student 't' test. All the tests were considered significant at $p<0.05$.

## RESULTS

### Subject compliance

FOS 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 FOS intake (by means of compliance sheet), which indicated good compliance.

### Effect of FOS supplementation on gut incretin (GLP-1)

As shown in table 1 a non significant increase of 17.42% in the GLP-1 levels was observed in the obese subjects with FOS intervention.

### Effect of FOS supplementation on human fecal microbiota

Table 2 reveals gut microbiota count of the obese subjects before and after FOS supplementation. The fecal log count of *Lactic acid bacteria* and *bifidobacteria* showed significant increased by 14% and 10% respectively ($p<0.05$, $p<0.005$). There was significant reduction by 20% of fecal log counts of enteric pathogen in obese subjects after FOS supplementation. Furthermore, an attempt was made to determine the effect of FOS consumption on the fecal microflora of the subjects with weight reduction and without weight reduction after FOS supplementation (table3). Subjects with weight reduction had a higher significant increase in the fecal log counts of *Lactic acid bacteria* ($p<0.001$) and *Bifidobateria* ($p<0.01$) after intervention. Percent decrease in the fecal log count of enteric pathogen was significantly higher in the obese subjects with weight reduction (24%) as compared to with no weight reduction (14%) after FOS supplementation.

**Table 1: Mean GLP-1 levels of obese subjects before and after FOS supplementation**

| Parameters | | Placebo control Group (n=30) | Experimental Group (n=30) | Student 't' Test |
|---|---|---|---|---|
| **GLP-1** | | **Mean ± SD** | **Mean ± SD** | |
| | **Pre** | 1.76±0.94 | 1.321±0.712156 | $1.19^{NS}$ |
| | **Post** | 1.60±0.80 | 1.552±0.9298 | $0.12^{NS}$ |
| | **Paired 't' test** | $1.27^{NS}$ | $1.85^{NS}$ | |
| | **% difference** | **9.09% ↓** | **17.42% ↑** | |

NOTE: NS = non-significant, Significant from the baseline value at $p<0.05$.

### Effect of FOS supplementation on hunger scores of obese adults

The total hunger scores has increased significantly by 3.61% ($p<0.05$) for lunch hours resulting into an increased satiety as shown in table 4 after FOS supplementation. Total scores obtained by the subjects increased significantly by and 3.15% ($p<0.05$) respectively in the experimental group. A significant increase ($p<0.05$) in the hunger score (better satiety) was reported for lunch and dinner among obese subjects with weight reduction (Table 5).

### Effect of FOS supplementation on dietary intake of obese adults

Table 6 describes a composite picture of dietary analysis of obese subjects. Results revealed that there is significant reduction in the intake of carbohydrate ($p<0.005$), energy, protein and fat ($p<0.05$) in the experimental group.

**Effect of FOS supplementation on anthropometric parameters of obese adults**

As seen in table 7 FOS supplementation results in significant reduction was observed in weight ($p<0.001$), BMI ($p<0.001$), percent body fat ($p<0.001$) WC ($p<0.05$) and WHR ($p<0.05$).

**Table 2: Microbial profile of obese subjects before and after FOS supplementation**

| Parameters | Placebo Control (n = 30) | Experimental (n = 30) | *Student t* test |
|---|---|---|---|
| ***Lactic acid bacteria*** | | | |
| **Pre** | 6.66 ± 0.95 | 7.08 ± 1.10 | **1.58** NS |
| **Post** | 6.51 ± 0.96 | 8.06 ± 1.40 | **5.02*** ** |
| **Paired t test** | 0.77 NS | **5.70*** ** | |
| **% difference** | 2.25% ↓ | 13.84% ↑ | |
| ***Bifidobacteria*** | | | |
| **Pre** | 6.93 ± 0.99 | 7.28 ± 1.05 | **1.32** NS |
| **Post** | 7.02 ± .93 | 8.03 ± 1.21 | **3.6**** |
| **Paired t test** | 0.39 NS | **3.80**** | |
| **% difference** | 1.30% ↑ | 10.30% ↑ | |
| **Enteric Pathogen** | | | |
| **Pre** | 5.09 ± 0.94 | 4.99 ± 1.17 | |
| **Post** | 5.19 ± 1.07 | 3.98 ± 0.99 | 0.41 NS |
| **Paired t test** | 0.35 NS | **6.20*** ** | **4.50*** ** |
| **% difference** | 1.96% ↑ | 20.24% ↓ | |

Note: ** Significant from the baseline value at $p<0.01$, *** Significant from the baseline value at $p<0.001$, NS - Non Significant

**Table 3: Gut microbial count of obese subjects with weight reduction and no weight reduction of obese subjects before and after FOS supplementation**

| Parameters | Wt reduction (n=18) | No wt reduction (n=12) | Student 't' test |
|---|---|---|---|
| ***Lactic acid bacteria*** | | | |
| **Pre** | 7.04 ± 1.15 | 7.13 ± 1.06 | |
| **Post** | 8.37 ± 1.28 | 7.59 ± 1.50 | 0.21 NS |
| **Paired t test** | **6.94*** ** | 1.75 NS | 0.51 NS |
| **% difference** | 18.89% ↑ | 6.45% ↑ | |
| ***Bifidobacteria*** | | | |
| **Pre** | 7.33 ± 1.02 | 7.21 ± 1.14 | |
| **Post** | 8.50 ± 1.04 | 7.34 ± 1.15 | 0.29 NS |
| **Paired t test** | **4.65**** | 0.62 NS | **2.85**** |
| **% difference** | 15.96% ↑ | 1.80% ↑ | |
| **Enteric Pathogen** | | | |
| **Pre** | 4.99 ± 1.13 | 4.97 ± 1.28 | |
| **Post** | 3.79 ± 0.78 | 4.28 ± 1.22 | 0.07 NS |
| **Paired t test** | **6.42*** ** | **2.56*** | 1.32 NS |
| **% difference** | 24.05% ↓ | 13.88% ↓ | |

*Significant from the baseline value at $p<0.05$, ** Significant from the baseline value at $p<0.01$, *** Significant from the baseline value at $p<0.001$, NS - Non Significant

## DISCUSSION

Very little information is available on effects of FOS supplementation on weight reduction in obese adults. We tried to address this issue with an aim study the metabolic and gut microbial compositional changes in FOS supplemented obese adults. In the present study FOS supplementation brought noteworthy improvements in gut incretin (GLP-1), gut microbial composition (*LAB, bifidobacteria* and *enteric pathogen*), hunger score, dietary intake and anthropometric parameters.

**Table 4: Mean hunger scores of obese subjects before and after FOS supplementation**

| Meal Time | | Placebo control Group (n=30) | Experimental Group (n=30) | Student 't' Test |
|---|---|---|---|---|
| | | Mean ± SD | Mean ± SD | |
| Breakfast | Pre (mean ± SD) | 4.0 ± 0.74 | 3.8 ± 1.03 | 0.86 NS |
| | Post (mean ± SD) | 3.97 ± 0.61 | 3.9 ± 1.18 | 0.27 NS |
| | Paired t | 0.44 NS | 1.79 NS | |
| | % difference | 0.75% ↓ | 2.63% ↑ | |
| Lunch | Pre (mean ± SD) | 4.0 ± 0.83 | 3.6 ± 0.77 | 1.90 NS |
| | Post (mean ± SD) | 3.97 ± 0.76 | 3.73 ± 0.91 | 1.07 NS |
| | Paired t | 0.55 NS | **2.11 *** | |
| | % difference | 0.75% ↓ | 3.61% ↑ | |
| Evening | Pre (mean ± SD) | 4.23 ± 0.81 | 4.07 ± 0.64 | 0.88 NS |
| | Post (mean ± SD) | 4.2 ± 0.76 | 4.19 ± 0.79 | 0.16 NS |
| | Paired t | 0.57 NS | 1.36 NS | |
| | % difference | 0.71% ↓ | 2.95% ↑ | |
| Dinner | Pre (mean ± SD) | 3.93 ± 0.74 | 3.77 ± 0.86 | 0.80 NS |
| | Post (mean ± SD) | 3.93 ± 0.74 | 3.9 ± 0.96 | 0.15 NS |
| | Paired t | 0.00 NS | 1.68 NS | |
| | % difference | 0.00% ↓ | 3.45% ↑ | |
| Total scores | Pre (mean ± SD) | 4.04 ± 0.65 | 3.80 ± 0.75 | 1.28 NS |
| | Post (mean ± SD) | 4.01 ± 0.57 | 3.92 ± 0.88 | 0.47 NS |
| | Paired t | 0.72 NS | **2.13*** | |
| | % difference | 0.74% ↓ | 3.15% ↑ | |

NOTE: NS = non-significant, p < 0.05*: Hunger scores 1 – 5, where 1= Famished, starving 2= Headache, weak, cranky, low energy, 3= Want to eat now, stomach growls and feels empty, 4= Hungry - but could wait to eat, starting to feel empty but not there yet, 5= Not hungry, not full

**Table 5: Mean hunger scores of obese subjects with weight reduction and no weight reduction before and after FOS supplementation**

| Meal Time | | Wt reduction (n=18) | No wt reduction (n=12) | Student 't' Test |
|---|---|---|---|---|
| | | Mean ± SD | Mean ± SD | |
| Breakfast | Pre (mean ± SD) | 4.00 ± 1.03 | 3.50 ± 1.00 | 1.31 NS |
| | Post (mean ± SD) | 4.17 ± 1.25 | 3.50 ± 1.00 | 1.54 NS |
| | Paired t | 1.84 NS | 0.00 NS | |
| | % difference | 4.25% ↑ | 0.00% | |
| Lunch | Pre (mean ± SD) | 3.83 ± 0.62 | 3.25 ± 0.87 | **2.15*** |
| | Post (mean ± SD) | 4.06 ± 0.80 | 3.25 ± 0.87 | **2.61*** |
| | Paired t | **2.20*** | 0.00 NS | |
| | % difference | 6.01% ↑ | 0.00% | |
| Evening | Pre (mean ± SD) | 4.17 ± 0.62 | 3.92 ± 0.67 | 1.05 NS |
| | Post (mean ± SD) | 4.39 ± 0.78 | 3.83 ± 0.72 | 1.97 NS |
| | Paired t | **2.20*** | 1.00 NS | |
| | % difference | 5.28% ↑ | 2.30%↓ | |
| Dinner | Pre (mean ± SD) | 3.89 ± 0.83 | 3.58 ± 0.90 | 0.95 NS |
| | Post (mean ± SD) | 4.17 ± 0.86 | 3.50 ± 1.00 | 1.95 NS |
| | Paired t | **2.55*** | 1.00 NS | |
| | % difference | 7.20% ↑ | 2.23% ↓ | |
| Total scores | Pre (mean ± SD) | 3.97 ± 0.69 | 3.56 ± 0.79 | 1.93 NS |
| | Post (mean ± SD) | 4.19 ± 0.84 | 3.52 ± 0.82 | 1.64 NS |
| | Paired t | 1.45 NS | 1.00 NS | |
| | % difference | 5.54%↑ | 1.12%↓ | |

NOTE: NS = non-significant, p < 0.05: *, Hunger scores 1 – 5, where 1= Famished, starving 2= Headache, weak, cranky, low energy, 3= Want to eat now, stomach growls and feels empty, 4= Hungry - but could wait to eat, starting to feel empty but not there yet, 5= Not hungry, not full

**Table 6: Mean dietary intakes of obese subjects before and after FOS supplementation**

| Nutrient | | Placebo control (n=30) | Experimental (n=30) | Student 't' Test |
|---|---|---|---|---|
| | | Mean ± SD | Mean ± SD | |
| Energy (Kcal) | Pre (mean ± SD) | 2345.95±351.48 | 2431.56±368.09 | 0.92 NS |
| | Post (mean ± SD) | 2353.46±325.45 | 2236.86±338.12 | 1.36 NS |
| | Paired t | 0.08 NS | **2.98*** | |
| | % difference | 0.32% ↑ | 8.01% ↓ | |
| Carbohydrate (g) | Pre (mean ± SD) | 350.73 ± 44.21 | 356.28 ± 43.62 | 0.48 NS |
| | Post (mean ± SD) | 353.68 ± 56.23 | 328.33 ± 35.02 | **2.09*** |
| | Paired t | 0.24 NS | **3.51**** | |
| | % difference | 0.84% ↑ | 7.84% ↓ | |
| Protein (g) | Pre (mean ± SD) | 52.66 ± 7.25 | 54.90 ±10.19 | 0.97 NS |
| | Post (mean ± SD) | 53.02 ± 9.55 | 51.79 ± 9.63 | 0.49 NS |
| | Paired t | 0.15 NS | **2.14*** | |
| | % difference | 0.68% ↑ | 5.66% ↓ | |
| Fat (g) | Pre (mean ± SD) | 80.21 ± 13.42 | 87.11 ± 14.25 | 1.90 NS |
| | Post (mean ± SD) | 81.32 ± 18.19 | 80.81 ± 16.06 | 0.11 NS |
| | Paired t | 0.27 NS | **2.05*** | |
| | % difference | 1.38% ↑ | 7.23% ↓ | |
| Soluble Dietary Fibre (g) | Pre (mean ± SD) | 3.80 ± 1.68 | 3.81 ± 1.19 | 0.03 NS |
| | Post (mean ± SD) | 3.85±2.62 | 3.78 ±1.70 | 0.20 NS |
| | Paired t | 1.01 NS | 0.39 NS | |
| | % difference | 1.31↑ | 0.78↓ | |
| Insoluble Dietary Fibre (g) | Pre (mean ± SD) | 11.20 ±4.59 | 12.18 ± 4.16 | 0.86 NS |
| | Post (mean ± SD) | 11.31 ±3.47 | 12.09 ± 3.67 | 0.70 NS |
| | Paired t | 1.04 NS | 0.86 | |
| | % difference | 0.98↑ | 0.73↓ | |
| Crude Fibre (g) | Pre (mean ± SD) | 6.47 ± 2.13 | 7.25 ±2.20 | 1.39 NS |
| | Post (mean ± SD) | 6.63 ±2.24 | 7.19 ±2.42 | 0.98 NS |
| | Paired t | 2.01 NS | 0.76 NS | |
| | % difference | 2.47↑ | 0.82↓ | |
| Total Fibre (g) | Pre (mean ± SD) | 15.03 ± 6.07 | 16.06 ± 5.18 | 0.71 NS |
| | Post (mean ± SD) | 15.16 ± 5.84 | 15.87 ±5.42 | 0.39 NS |
| | Paired t | 1.22 NS | 1.13 NS | |
| | % difference | 0.86↑ | 1.18↓ | |

NOTE: NS = non-significant, $p < 0.05$: *, $p < 0.01$: **

In the present study almost 17% increase in GLP-1 values was observed following FOS supplementation. In a similar study where FOS fed to the animals (mice) was associated with increased portal GLP-1 levels, prebiotic feeding promotes GLP-1 synthesis (mRNA and peptide content) in the proximal colon by a mechanism linked to the differentiation of precursor cells into enteroendocrine cells. Moreover, in another set of experiments performed in high-fat diet induced obesity and type 2 diabetes, the modulation of gut microbiota using prebiotic protects against body weight gain, fat mass development (visceral, epidydimal and subcutaneous), glucose intolerance, and hepatic insulin resistance (Cani et al, 2006).

Present study revealed 14% and 10% increment in LAB and bifidobacteria counts respectively and 20% reduction in enteric pathogen counts as a result of 12 g FOS supplementation. A similar study conducted in mice showed that FOS increased the counts of *bifidobacteria* (Koket al, 1998). *Lactic acid bacteria* and *bifidobacteria* were screened for their ability to ferment FOS showed that of 28 strains of *LAB* and *bifidobacteria* examined, 12 of 16 LAB strains and 7 of 8 *bifidobacteria* strains fermented FOS (Handan K and Robert WH, 2000).

Present study is also supported by a study where, in the small intestine, the viable counts of *bifidobacterium* and *lactobacillus* significantly increased in broilers, fed diet with 4g/kg FOS (ZR XU et al, 2003).Moreover, human trials also elicited that oligosaccharides that are fermented by colonic microflora enhanced that growth of beneficial commensal organisms like *bifidobacteria* and *lactobacillus* (Giovanni et al, 2010).

**Table 7: Mean anthropometric values of obese subjects before and after FOS supplementation**

| Parameters | | Placebo control (n=30) Mean ± SD | Experimental (n=30) Mean ± SD | Student 't' Test |
|---|---|---|---|---|
| **Height (cm)** | **Pre** | 1.70 ± 0.040 | 1.71 ± 0.06 | 0.25 $^{NS}$ |
| | **Post** | 1.70 ± 0.040 | 1.71 ± 0.06 | 0.25 $^{NS}$ |
| **Weight(kg)** | **Pre** | 79.15 ± 4.8 | 79.41 ± 6.60 | 0.17 $^{NS}$ |
| | **Post** | 79.27 ± 5.05 | 78.57± 6.42 | 0.47 $^{NS}$ |
| | **Paired 't' Test** | 0.73 $^{NS}$ | **4.05*** ** | |
| | **% difference** | 0.15% ↑ | 1.06% ↓ | |
| **BMI(kg/m2)** | **Pre** | 27.34 ± 1.56 | 27.29 ± 1.43 | 0.14 $^{NS}$ |
| | **Post** | 27.38 ± 1.62 | 27.00 ± 1.44 | 0.95 $^{NS}$ |
| | **Paired 't' Test** | 0.73 $^{NS}$ | **4.03*** ** | |
| | **% difference** | 0.15% ↑ | 1.06% ↓ | |
| **WC (cm)** | **Pre** | 98.1 ± 3.1 | 98.67 ± 4.99 | 0.53 $^{NS}$ |
| | **Post** | 98.3 ± 3.2 | 97.03 ± 4.99 | 1.28 $^{NS}$ |
| | **Paired 't' Test** | 1.71 $^{NS}$ | **2.52*** | |
| | **% difference** | 0.20% ↑ | 1.66% ↓ | |
| **HC (cm)** | **Pre** | 103.86 ± 2.6 | 104.77 ± 3.22 | 1.18 $^{NS}$ |
| | **Post** | 104.25 ± 2.4 | 104.67 ± 3.18 | 0.56 $^{NS}$ |
| | **Paired 't' Test** | **2.12*** | 1.70 $^{NS}$ | |
| | **% difference** | 0.38% ↑ | 0.10 % ↓ | |
| **WHR** | **Pre** | 0.94 ± 0.02 | 0.94 ± 0.03 | 0.50 $^{NS}$ |
| | **Post** | 0 .94 ± 0 .02 | 0.92± 0.02 | **2.45*** |
| | **Paired 't' Test** | 0.74 $^{NS}$ | **2.41*** | |
| | **% difference** | 0.00% ↓ | 2.13% ↓ | |
| **% Body Fat** | **Pre** | 28.34 ± 1.52 | 28.40 ± 2.14 | 0.12 $^{NS}$ |
| | **Post** | 28.41 ± 1.52 | 27.20 ± 2.02 | **2.53*** |
| | **Paired 't' Test** | 1.20 $^{NS}$ | **3.53*** ** | |
| | **% difference** | 0.25% ↑ | 4.23% ↓ | |
| NOTE: NS = non-significant, $p < 0.05$: *, $p < 0.01$: **, $p < 0.001$: *** | | | | |

In our study the total hunger scores of the obese subjects improves significantly ($p<0.05$) by 3.15% resulting in their decreased food intake (Table 6). Similar findings are also reported by several investigators that prebiotic treatment increased breath hydrogen excretion (a marker of gut microbiotia fermentation) by ≈3 folds and lowered hunger rates. Prebiotic supplementation was associated with increased in plasma gut peptide concentration (GLP-1, PYY) which may contribute in part to changes in appetite sensation and glucose excretion response after a meal in healthy subjects (Cani et al, 2009, Cani et al, 2006, Whelan et al, 2006, Parnell and Reimer 2009, Genta et al, 2009)

Total dietary intake of subjects reduced significantly in our study which was also supported by Cani et al, 2009 who reported that in prebiotic treated subjects total dietary intake (energy, protein, carbohydrate, fat, dietary fiber) was lowered by ≈ 6%.

Together these results suggests role of fermentable FOS in explaining the reduced weight and the underlying mechanism behind it.

## CONCLUSION

Thus, it can be concluded that FOS is an encouraging therapy for management of obesity in terms of increasing satiety, increasing beneficial gut microbiota and reducing harmful pathogens in the colon and stimulating production of GLP-1. Furthermore, longitudinal studies are needed to study the sustainability of the effects of FOS consumption on the weight reduction on a long term basis.

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