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Changes in Weight Status and the Intestinal Microbiota among Adolescent College Freshman

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Abstract

Purpose—The transition to college is a vulnerable period for weight gain and the onset of obesity. Gut microbes differ in obese compared to lean individuals, but gut microbiota in adolescent-aged college freshmen during a known period of weight gain have never been studied. This pre-post observational pilot study assessed associations between intestinal microbiota changes and weight-related outcomes in healthy adolescent college freshmen living in on-campus dormitories at Arizona State University (n=39).

Methods—We measured anthropometrics (waist circumference, height, weight, and body mass index) and collected fecal samples at the beginning and end of the 2015–2016 academic year. Fold changes in species-level microbes across time were measured by quantitative real-time PCR and used in correlation and multivariate regression analyses.

Results—A total of 24 female and 15 male adolescents (aged 18.54 ± 0.67 y) participated in this study. Over the academic year, body mass index (BMI) and waist circumference (WC) increased by 0.97±1.28 kg/m^2 and 2.64±4.90 cm, respectively. Correlation analyses indicated a significant negative association between *A. muciniphila* and both % WC change and % BMI change (r= −0.66; p<0.01 and r= −0.33; p=0.04, respectively). Multivariate regression analysis controlling for sociodemographics showed a significant association between *A. muciniphila* and % WC change, but not % BMI change (R^2=0.53; p<0.01 and R^2=0.24; p=0.15, respectively).

Conclusion—As this was the first study in a university-based adolescent population to show a relationship between *A. muciniphila* and weight-related outcomes, further research is needed to explore these findings.

Keywords

microbiota; waist circumference; obesity; students; adolescents

Introduction

Obesity is an epidemic in the United States with 78.6 million Americans classified as obese [1]. Emerging adulthood has been identified as a vulnerable period for weight gain and is a

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period marked by social (i.e. transition to college) and environmental changes. Specifically, college freshmen must adapt to changes in diet, physical activity, and peer influences [2]. Unfortunately, many college students gain weight during their freshman year and adopt unhealthy behaviors (e.g., unhealthy eating, low levels of physical activity) that persist throughout college [3,4]. These behaviors may ultimately increase the risk for cardiometabolic associated diseases (e.g. dyslipidemia, type 2 diabetes mellitus) later in life [5].

Previous research has shown that the intestinal microbiota is different in obese compared to lean individuals, and is thought to play a role in energy utilization from the host's diet [6,7]. Although the onset of obesity is complex, evidence suggests a connection between excess body weight and the intestinal microbiota. Studies of both mice and humans indicate a shift in abundance at the phyla-level, favoring Firmicutes over Bacteroidetes, with the onset of obesity or increases in fat mass resulting in differing gut microbiota profiles between obese and lean individuals [7–12]. These studies suggest that obesity-associated microbiota have an increased ability to change energy balance over time that ultimately results in weight gain [7].

Several species-level microbes have been studied in the literature for their association with health and cardiometabolic associated diseases (e.g. obesity, diabetes). *Akkermansia muciniphila, Ruminococcus gnavus* and *Faecalibacterium prausnitzii* have all been identified as microbial biomarkers with implications for obesity risk [10,13–15]. *Akkermansia muciniphila* is a mucin-degrading bacteria in the Verrucomicrobia phylum that contributes to gastrointestinal health by strengthening gut barrier function [16–18]. *A. muciniphila* has been shown in mice to be inversely associated with obesity, type 2 diabetes mellitus, and inflammation [18,19]. Similarly, A. muciniphila was significantly lower among overweight and obese children when compared to those at a healthy weight [10]. *Ruminococcus gnavus*, a gram-positive anaerobic bacteria belonging to the Firmicutes phylum [20] has been associated with inflammatory bowel diseases and is thought to contribute to a damaged mucosal epithelial barrier and chronic inflammation [21,22]. Obese rats have been shown to have greater abundance of R. gnavus [23] while examination of the adult gut microbiome has revealed that *R. gnavus* associates with low bacterial diversity, greater body mass index (BMI), and inflammation [15]. *Faecalibacterium prausnitzii*, another member of the Firmicutes phylum, is associated with health and has been shown to have a lower abundance in obese individuals [24]. *F. prausnitzii* is also negatively associated with inflammation and may be beneficial to the lining of the intestine as a producer of butyrate, a short chain fatty acid that improves gut barrier function and systemic health [9,25].

Current microbial markers of obesity risk, including those listed above, have only been studied cross-sectionally or in the context of weight loss interventions among adolescent populations [10,13,26]. While future research goals revolve around the ability to manipulate intestinal microbial communities to minimize weight gain and energy harvest, we must first understand how microbial communities change with weight gain and lead to subsequent adiposity. Therefore, we studied how these same microbes changed during a period of expected weight gain in late adolescence. To our knowledge, this is the first pre-post observational human study that has evaluated changes in the gut microbiota during a period

of increased susceptibility to weight gain, such as the first year in a college setting. The aim of this study was to assess changes in three intestinal microbiota biomarkers associated with obesity in adolescent-aged first year college students living in on-campus dormitories and to examine if these changes were associated with changing weight status and weight circumference (WC) measurements.

Methods

This pre-post observational study took place between August 2015 and May 2016 at Arizona State University. College students were recruited from floor meetings in select on-campus dormitories in the fall of 2015. After being recruited for a larger parent study, Social Impact of Physical Activity and Nutrition in College (SPARC) [27], participants were then given the option to enroll in this sub-study, called devilWASTE. Students were considered eligible for the sub-study if they were English speaking males or females and if they were enrolled in the parent study. Students were excluded from the study if they had a history of eating disorders, malabsorptive diseases, HIV infection, high blood pressure, diabetes, or were taking prebiotics, probiotics, antibiotics, or antifungal treatments fewer than 3 months prior to stool collection. All study protocols were approved by the Arizona State University Institutional Review Board and written informed consent was obtained from each participant.

Pre- and post- fecal samples were collected in August/September of 2015 and April/May of 2016, respectively. At the first collection timepoint, participants completed an initial survey with demographic information. At both study visits, participants met with researchers at the residence halls on campus for anthropometric measurements and to pick up fecal sample collection materials. At both study visit, participants were also asked to report on any medications or supplements they might have been recently prescribed so that timing of use of any antibiotics, antifungals, or probiotics could be assessed. If these products were taken within the previous 3 months at either visit, the participant was not permitted to provide a fecal sample at that study timepoint. Body mass index (BMI) was calculated as kg/m^2, with the height measured on a stadiometer (SECA, USA) and weight measured on a high-precision calibrated scale (SECA, USA). Waist circumference (WC) was measured at the umbilicus with a spring-loaded, tension measuring tape. At pre- and post- visits, height, weight, and WC were measured up to three times as replicates for each participant to obtain two measures within 0.5 cm, 0.5 kg, and 0.5 cm of each other, respectively. The two measurements within these parameter ranges were then averaged and used for data analysis. Percent changes in BMI and WC were calculated as the difference between both study time points divided by baseline values. All research staff were trained using validated measurement techniques.

Following participant collection of fecal samples, the samples were picked up as soon as possible within 30 minutes from the time that participants reported a bowel movement. Study staff were on call 24 hours a day to accommodate this protocol to ensure viability of fecal microbes. Fecal samples were then transported to the laboratory and frozen at −80°C until processing. Frozen fecal samples were thawed at 4°C and wet weight was recorded to the nearest 0.01 g after subtracting the weight of collection materials. DNA was extracted

from 200–300 mg of feces, collected from the center of the sample, using a modified version of the protocol outlined in the MoBio Power Soil DNA Isolation Kit (12888–100, MoBio, Carlsbad, CA). A heating step of 65°C for 10 minutes was added to the original protocol, per manufacturer recommendations, to reduce the influence of inhibitors commonly found in feces and increase DNA yield. DNA concentration and quality were checked and quantified using a QIAxpert System (Qiagen, Germantown, MD) according to manufacturer instructions.

Microbial targets were quantified through quantitative real-time PCR (qPCR) analysis with proprietary microbial DNA qPCR Assay kits (Qiagen, Germantown, MD for the following targets: *Ruminococcus gnavus* (BPID00299A), *Faecalibacterium prausnitzii* (BPID00154A), and *Akkermansia muciniphilia* (BPID00026A). All kits were used as intended with no modifications to the manufacturer protocols in order to achieve the reported efficiency near 100%. The PCR reactions were performed using a CFX Connect thermocycler (Bio-Rad). Each reaction was run in duplicate with a final volume of 25 μl including 12.5 μl of microbial qPCR mastermix, 1μl of microbial DNA qPCR primers, 5 ng of genomic DNA from fecal samples, and microbial DNA-free water as specified per microbial target. Each plate contained a no template control, healthy control sample, and *Pan Bacteria 3* (BPCL00362A) as a reference microbe for each participant. For all reactions, samples were activated at 95°C for 10 minutes, followed by 40 cycles of denaturation at 95°C for 15 seconds, and annealing and extension at 60°C for 2 minutes. Relative quantification of target microbes was calculated by using the $2^{(-\Delta\Delta CT)}$ method [28].

Statistical analysis was carried out using IBM SPSS Version 24 (SPSS Inc., Chicago, IL) and JMP (SAS Institute, Cary, NC). Prior to analyses, data were organized and cleaned by removing outliers >3 SD from the mean. All data were checked for normality using the Shapiro-Wilk test, and $p>0.05$ was considered normally distributed. Logarithmic transformations were performed on non-parametric microbial fold change data to meet statistical test assumptions. Spearman correlation tests were used to examine correlations between log-transformed microbial fold changes and both percent change in WC and percent change in BMI. To further assess these associations between weight status variables and microbial fold changes, multivariate regression models were used to examine associations between percent change in WC or BMI and microbial fold changes with the addition of sex and race/ethnicity as covariates. Log-transformations of microbial fold change data were not needed in the multivariate regression analyses as model residuals were normally distributed for each microbial biomarker. All data were presented as mean ± SD and $p<0.05$ was considered statistically significant.

Results

Participant Characteristics

Forty-two college freshmen living in dormitories at Arizona State University were included in this study to evaluate fecal microbiota changes in relation to college weight gain. During data analysis, 3 outliers were excluded from analyses (>3 SD from the mean), leaving 39 participants who were included in final analyses. Participant characteristics are described in Table 1. At baseline, participants had an average BMI of 24.46 ± 4.24 kg/m^2, weight of

69.76 ± 14.84 kg, and WC of 80.63 ± 11.19 cm. At the post measurement in April/May, BMI had increased by an average of 0.97 ± 1.28 kg/m^2, weight by 2.89 ± 3.74 kg, and WC by 2.64 ± 4.90 cm.

Waist Circumference, BMI, and Microbial Abundance Correlations

Spearman's correlations were used to examine the association between log-transformed microbial data and both % change in WC and % change in BMI (Figure 1). Results suggested a significant negative correlation between *A. muciniphila* fold change and % change in WC (r= −0.66, p<0.01), but correlations were not significant between *F. prausnitzii* (r=0.18, p=0.26) or *R. gnavus* (r= −0.10, p=0.54) and % change in WC. Regarding % change in BMI, a significant negative correlation was observed for *A. muciniphila* fold change (r= −0.33, p=0.04), but not *F. prausnitzii* (r= −0.15, p=0.37) or *R. gnavus* (r= −0.17, p=0.31).

Multivariate regression models were used to evaluate associations between BMI/WC and microbial fold changes while controlling for covariates known to influence the gut microbiome. For *A. muciniphila* (Table 2), the WC model was significant (R^2=0.53, p<0.01), with % change in WC having a negative influence on microbial abundance (Estimate= −0.22, p<0.01). Results were not significant for the *A. muciniphila* and % BMI change model (R^2=0.24, p=0.15) once covariates were included in the model. Neither % change in BMI nor % change in WC were signficant predictors of *F. prausnitzii* fold change (R^2=0.10, p=0.72; R^2=0.06, p=0.90, respectively; Table 3) following the inclusion of covariates. Similar models of *R. gnavus* fold change with respect to % changes in WC and BMI approached significance (R^2=0.29, p=0.07 and R^2=0.27, p=0.09, respectively; Table 4). Notably, self-reporting as Black race/ethnicity had a negative influence on the change in R. gnavus that approached significance in both BMI and WC models (Estimate= −0.69, p=0.09; Estimate= −0.78, p=0.06, respectively).

Discussion

This pilot observational study of college freshman provides insight into a period of adolescent life commonly associated with weight gain. Correlation analyses suggested that there were no associations between % change in WC and either *R. gnavus* or *F. prausnitzii* fold changes. However, *A. muciniphila* fold change was significantly negatively correlated with both % change in WC and % change in BMI. *A. muciniphila* fold change had a greater association with % change in WC than % change in BMI. Further analysis with multivariate regressions suggested that % WC change had a significant influence on *A. muciniphila* fold change after accounting for covariates, while the association with % BMI change was lost. Multivariate analysis of *R. gnavus* and *F. prausnitzii* fold changes did not yield significant associations with changes in WC or BMI.

This study was the first to show that *A. muciniphila* had a significant negative correlation with % change in WC and % change in BMI during a period of weight gain in an adolescent-aged college population, suggesting that as participants experienced increases in WC and BMI over the 9-month academic year, the abundance of *A. muciniphila* decreased. The relationship was stronger for WC, suggesting that WC may have a greater effect than

BMI on *A. muciniphila* fold change. These findings are in agreement with previous studies showing that *A. muciniphila* abundance differs significantly between overweight/obese and normal weight children [13], and negatively correlates with BMI in adults [29]. Although causality cannot be ascertained from the current pilot study, it is possible that decreases in *A. muciniphila* occur prior to the onset of obesity and related comorbid conditions as data from sub-clinical type 2 diabetes patients indicate that *A. muciniphila* abundance decreased before the onset of the disease [30]. Overall, previous studies suggest that *A. muciniphila* abundance is associated with weight-related outcomes, and data from the current study support these results.

In the present study, only % WC change remained significantly negative associated with *A. muciniphila* fold change in multivariate regressions that included covariates. These data suggest that *A.muciniphila* may be a microbial marker of reduced visceral fat storage, as WC has been shown to be a reliable measurement of central adiposity [31]. A study examining overweight/obese adults showed that the abundance of *A. muciniphila* was inversely associated with subcutaneous white adipocyte diameter and that overweight/obese participants who had a healthier metabolic status, had greater *A. munciniphila* abundance [32]. In mice fed a high-fat diet, treatment with *A. muciniphila* attenuated fat mass gain and adipose tissue inflammation via improved gut barrier integrity [18,33]. Further research is needed to explore the relationships between *A. muciniphila*, gut permeability and visceral fat in humans.

This study showed no association between *F. prausnitzii* changes and weight-related outcomes of WC and BMI. Despite our null findings, *F. prausnitzii* has been negatively associated with disease states, inflammation, increased WC, and dietary changes in a variety of populations and settings [9,11,24,34,35]. With regard to weight changes, *F. prausnitzii* abundance has been shown to increase significantly among type 2 diabetics [36] and obese individuals [9] following weight loss. In these studies, participants lost a mean of approximately 3 kg of body weight which may suggest that slightly larger changes in weight status, compared to those observed in this study, may be necessary to influence the abundance of *F. prausnitzii*.

R. gnavus fold change was not associated with changes in WC and BMI in this study. Contrary to our findings, a study showed that *R. gnavus* was positively correlated with BMI in adults [11]. Further, a weight-loss intervention in adolescents showed a decrease in *Clostridium* cluster XIVa that correlated with weight loss (~4 kg over 10 weeks) [13]. *R. gnavus* is a member of *Clostridium* cluster XIVa along with other genera such as *Coprocuccus*, *Eubacterium*, and *Lachnospira*, of which *Eubacterium* has been associated with the metabolic syndrome [13,24,37]. It is possible that the amount of weight gain observed in our study was not large enough for R. gnavus abundance to be impacted; therefore, further evaluation of the role of *R. gnavus* in weight-related outcomes is needed across a wider range of weight change.

Overall, these findings provide insight into the associations between gut microbiota and weight-related outcomes in first year college students living in on-campus housing. The strengths of this study include the pre-post observational design that allowed for the

assessment of relationships over time, and the ability to capture a period of time when participants were susceptible to weight gain. While studying weight gain is difficult due to the ethical concerns, the observational nature of this study allowed weight gain to occur in a free-living population without imposing weight gain on participants. Lastly, the focus on older adolescents during the freshman-year transition to college is an important addition to the literature as college freshmen have largely been ignored in the gut microbiome literature.

The limitations of this study include using WC as a non-invasive measure of visceral adiposity instead of using dual-energy x-ray absorptiometry. This limitation was necessary because of the large sample size in the parent study, logistics scheduling DXA scans, and limited funding. Using weight and BMI as a measure of obesity was also a limitation as weight differences over time can be due to a variety of factors. Only one fecal sample was collected at each time point, so it is unclear if results are representative of the normal gut microbial composition or if changes are due to other lifestyle factors not included in this study. Specifically, dietary and physical activity factors should be evaluated in conjunction with gut microbial changes during this time period as they are known to have an impact on weight gain during the college years [38]. Additionally, this study included a small sample size that limited our ability to also analyze these lifestyle factors. The sample size may also not have been representative of larger populations; therefore, the generalizability of results is limited and should be confirmed with further research. Lastly, two participants in this study took antibiotics within the months prior to fecal collection. While they met the study inclusion criteria (antibiotics >3 months prior to fecal collection), it is possible that the gut microbiome communities of these individuals were still altered from their treatments. However, it is important to note that previous studies have used similar cutoffs for antibiotic use [39,40].

The transition to college has been identified as a period of increased susceptibility to weight gain as students adapt to many lifestyle changes [3,4]. To our knowledge, this is the first human-based study to analyze the gut microbiota during a period of weight gain. Although the observed weight gain in this study was small (<3 kg), we found a statistically significant negative association between *A. muciniphila* fold changes and changes in WC in freshman students living in on-campus housing over a 9-month academic year. More research is needed to confirm these findings and further explore relationships between the intestinal microbiome and weight-related outcomes in college students. A larger sample size and more comprehensive sequencing of microbial communities could provide more information on potential associations and mechanisms. A specific area for further investigation is to observe how both dietary and physical activity behaviors may contribute to weight and microbial changes across the freshman year of college. Ultimately, such findings may inform future interventions aimed at preventing weight gain in this high-risk population.

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Implications and contributions:

While the transition to college is considered a vulnerable period for weight gain, no literature is available on the impacts of gut microbiota on weight status in emerging adulthood. These preliminary data may contribute to the understanding of associations of gut microbiota and weight in the onset of obesity.

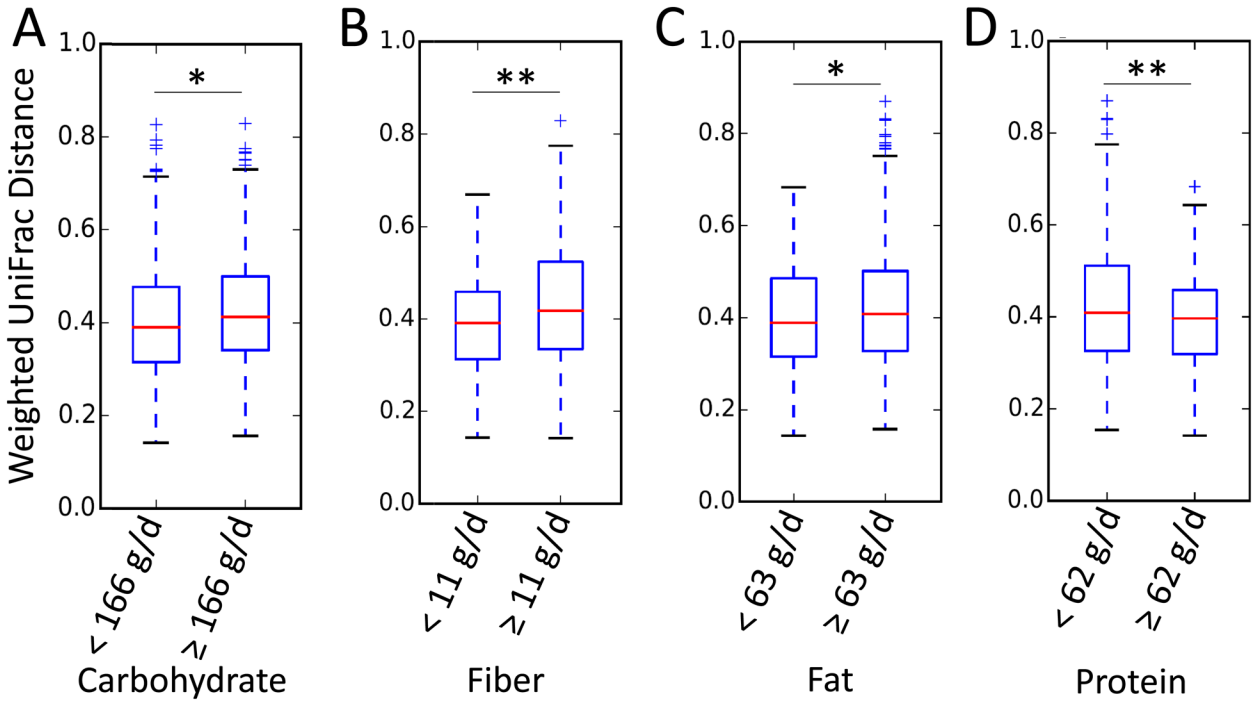

Figure 1.

Correlations between log-transformed microbial fold change and % change in WC (left) and % change in BMI (right) for adolescent-aged college freshmen.

Table 1.

Baseline characteristics of study participants (n=39)

Characteristic	Result
	% (n)
Gender	
Female	61.5 (24)
Male	38.5 (15)
Race/Ethnicity	
Native American/Mixed	12.8 (5)
Asian	5.1 (2)
Black	12.8 (5)
Hispanic	23.1 (9)
White	46.2 (18)
	Mean ± SD
Age, *y*	18.54 ± 0.67
Start of school year	
BMI, kg/m^2	24.46 ± 4.24
Weight, *kg*	69.76 ± 14.84
Waist circumference, *cm*	80.63 ± 11.19
End of school year	
BMI, kg/m^2	25.44 ± 4.68
Weight, *kg*	72.65 ± 16.10
Waist circumference, *cm*	83.27 ± 12.39

BMI, body mass index.

Table 2.

Multivariate regression for WC and BMI change and *A. muciniphila* fold change (n=39).

A. muciniphila				
Waist Circumference (R^2=0.53; p<0.01*)				
Term	**Estimate**	**Std Error**	**t Ratio**	**Prob > | t |**
Intercept	0.99	0.36	2.75	0.01*
% WC change	−0.22	0.05	−4.76	<0.01*
Sex	−0.03	0.32	−0.09	0.93
Native American/Mixed	0.06	0.65	0.09	0.93
Asian	1.60	1.02	1.57	0.13
Black	−1.04	0.70	−1.48	0.15
Hispanic	−0.07	0.53	−0.13	0.90
Body Mass Index (R^2=0.24; p=0.15)				
Term	**Estimate**	**Std Error**	**t Ratio**	**Prob > | t |**
Intercept	0.56	0.45	1.25	0.22
% BMI change	−0.11	0.07	−1.52	0.14
Sex	0.50	0.37	1.35	0.19
Native American/Mixed	−0.01	0.82	−0.01	0.99
Asian	2.03	1.38	1.48	0.15
Black	−1.65	0.87	−1.9	0.07
Hispanic	0.12	0.72	0.16	0.87

Abbreviations: BMI, Body Mass Index; WC, waist circumference.

*Significant p< 0.05

Table 3.

Multivariate regression for WC and BMI change and *F. prausnitzii* fold change (n=39).

F. prausnitzii				
Waist circumference (R^2=0.10; p=0.72)				
Term	**Estimate**	**Std Error**	**t Ratio**	**Prob > | t |**
Intercept	−0.23	0.15	−1.50	0.14
% WC change	0.02	0.02	1.20	0.24
Sex	0.08	0.13	0.61	0.55
Native American/Mixed	−0.13	0.27	−0.47	0.64
Asian	0.58	0.43	1.36	0.18
Black	−0.05	0.29	−0.18	0.85
Hispanic	−0.31	0.22	−1.41	0.17
Body Max Index (R^2=0.06; p=0.90)				
Term	**Estimate**	**Std Error**	**t Ratio**	**Prob > | t |**
Intercept	−0.15	0.15	−0.97	0.34
% BMI change	0.00	0.02	−0.12	0.91
Sex	0.02	0.12	0.13	0.90
Native American/Mixed	−0.11	0.28	−0.40	0.69
Asian	0.42	0.46	0.90	0.37
Black	0.03	0.29	0.1	0.92
Hispanic	−0.27	0.27	−1.12	0.27

Abbreviations: BMI, Body Mass Index; WC, waist circumference.

*Significant p< 0.05

Table 4.

Multivariate regression for WC and BMI change and *R. gnavus* fold change (n=39).

R. gnavus				
Waist circumference (R^2=0.29; p=0.07)				
Term	**Estimate**	**Std Error**	**t Ratio**	**Prob > | t |**
Intercept	−0.03	0.20	−0.15	0.88
% WC change	−0.05	0.03	−1.80	0.08
Sex	−0.21	0.18	−1.21	0.24
Native American/Mixed	0.17	0.37	0.46	0.65
Asian	−0.40	0.58	−0.69	0.49
Black	−0.69	0.39	−1.74	0.09
Hispanic	0.45	0.30	1.52	0.14
Body Max Index (R^2=0.27; p=0.09)				
Term	**Estimate**	**Std Error**	**t Ratio**	**Prob > | t |**
Intercept	−0.06	0.20	−0.30	0.76
% BMI change	−0.05	0.03	−1.52	0.14
Sex	−0.12	0.17	−0.71	0.48
Native American/Mixed	0.17	0.37	0.46	0.65
Asian	−0.52	0.62	−0.84	0.41
Black	−0.78	0.39	−1.99	0.06
Hispanic	0.59	0.33	1.82	0.08

Abbreviations: BMI, Body Mass Index; WC, waist circumference.

*Significant p< 0.05