HHS Public Access

Author manuscript

Obesity (Silver Spring). Author manuscript; available in PMC 2018 February 07.

Published in final edited form as:

Obesity (Silver Spring). 2017 October ; 25(10): 1660–1666. doi:10.1002/oby.21917.

Improvements in Cardiovascular Risk Factors in Young Adults in a Randomized Trial of Approaches to Weight Gain Prevention

Rena R. Wing, Ph.D.[1], **Deborah F. Tate, Ph.D.**[2], **Katelyn R. Garcia, MS**[3], **Judy Bahnson**[3], **Cora E. Lewis, MD**[4], **Mark A. Espeland, Ph.D.**[3], and **for the Study of Novel Approaches to Weight Gain Prevention (SNAP) Research Group.**

[1]Alpert Medical School of Brown University, the Miriam Hospital

[2]University of North Carolina – Chapel Hill

[3]Wake Forest School of Medicine

[4]University of Alabama at Birmingham

Abstract

Objective—Weight gain occurs commonly in young adults and increases cardiovascular (CVD) risk. We previously reported that two self-regulation interventions reduced weight gain relative to control. Here we examine whether these interventions also benefit CVD risk factors.

Methods—SNAP (Study of Novel Approaches to Weight Gain Prevention) was a randomized trial in 2 academic settings (N=599; 18–35 years; body mass index 21–30 kg/m^2) comparing two interventions (Self-Regulation with Small Changes; Self-Regulation with Large Changes) and Control. Small Changes taught participants to make daily small changes (approximately 100 calorie) in intake and activity. Large Changes taught participants to initially lose 5–10 pounds to buffer anticipated weight gains. CVD risk factors were assessed at baseline and 2 years in 471 participants.

Results—Although Large Changes was associated with more beneficial changes in glucose, insulin, and HOMA-IR than Control, these differences were not significant after adjusting for multiple comparisons or 2-year weight change. Comparison of participants grouped by percent weight change baseline to 2 years showed significant differences for several CVD risk factors, with no interaction with treatment condition.

Conclusions—Magnitude of weight change, rather than specific weight gain prevention interventions, was related to changes in CVD risk factors in young adults.

Keywords

Cardiovascular risk; prevention; risk factors; weight gain

Corresponding Author: Rena R. Wing, Ph.D., Professor of Psychiatry & Human Behavior, Warren Alpert Medical School of Brown University, Director, Weight Control & Diabetes Research Center, The Miriam Hospital, 196 Richmond Street, Providence, RI 02903, Tel: 401-793-8959, Fax: 401-793-8944. rwing@lifespan.org.

Trial registration: ClinicalTrials.gov, NCT01183689

INTRODUCTION

Young adults, ages 20–35, experience the fastest weight gain, averaging 1 to 2 pounds per year.[1–3] This weight gain is associated with worsening cardiovascular (CVD) risk factors, including lipids, blood pressure, glucose, and insulin, and increased risk of metabolic syndrome.[4–7] In Coronary Artery Risk Development in Young Adults (CARDIA) Study, for example, individuals age 18–30 gained approximately 21 kg over 25 years, with larger weight gains at younger ages.[8] The 16% who maintained their weight ± 5 pounds) over the first 15 years had no changes in CVD risk factors, whereas those who gained weight (> 5 pounds) had worsening in these risk factors and increased prevalence of metabolic syndrome.[7,9] Other epidemiological studies have shown that weight gain during young adulthood is associated with increased risk of coronary heart disease events, type 2 diabetes, hypertension and a variety of other disease.[10–14] In fact, weight gained by young adults has stronger negative associations with cancer risk and mortality than weight gained at later ages.[15,16]

There have been few large randomized trials designed to prevent weight gain in young adults.[17–19] Those which have been conducted have had limited long term success.[4,20] Moreover, we are not aware of any weight gain prevention studies in young adults that examined the effect of these interventions on change in CVD risk factors. In contrast, weight gain prevention in older individuals has had beneficial effects on CVD risk factors.[21,22]

We recently reported positive results from the Study of Novel Approaches to Weight Gain Prevention (SNAP), a randomized trial of two new approaches to weight gain prevention in 599 young adults.[23] The interventions were both based on self-regulation and stressed the importance of daily self-weighing and making changes in diet and physical activity based on those weights.[24] One intervention focused on making small, daily changes in eating and exercise behaviors, whereas the other recommended larger, periodic changes to buffer against expected weight gains. We found significant effects of both interventions, relative to the control group, on the primary outcome of weight gain over the average of 3 years of follow-up; moreover, weight in the Large Change condition was significantly reduced relative to the Small Change condition. In the present study, we hypothesized that the Large Change intervention would produce the most positive effects on CVD risk factors in association with its effect on weight change.

METHODS

Design

SNAP was an NIH funded clinical trial conducted at 2 clinical sites (UNC at Chapel Hill and The Miriam Hospital Providence RI) (Figure S1); Wake Forest University Health Sciences was the coordinating center. The study was approved by the IRBs at each of these locations. SNAP targeted 600 young adults who were randomly assigned with equal probability to 1 of 3 groups: Control, Small Changes, Large Changes. Assessments were completed at baseline, 4 months, and then annually. However, lipids, glucose and insulin were assessed only at baseline and 2 years and thus those time points are the focus of the present analyses. Change in CVD outcomes was a pre-specified secondary outcome for this trial. The design has been

described in detail and consort diagram provided in a prior publication.[25] Participants were recruited from 2010 to 2012 and data were analyzed from 2015 to 2016.

Participants

The SNAP study recruited individuals who were aged 18–35, with a BMI of 21 to 30.9. Normal weight and overweight young adults were included since both weight groups are at increased risk of weight gain relative to older individuals. Other eligibility criteria included access to Internet, English speaking, no history or current anorexia or bulimia nervosa and completion of screening and baseline assessment visits. Of the 599 participants in SNAP, 76 did not have measured weight at 2 years and an additional 52 had neither blood pressure nor blood work at this time, bringing the sample for this analysis to N= 471.

Interventions

The interventions have been described in detail.[23,25] The Control group attended one face-to-face meeting where they were introduced to both the small and large changes approaches to prevention of weight gain, but this group did not receive any further assistance with weight gain prevention. The two active interventions were both based on self-regulation; participants were encouraged to weigh themselves daily and to use the information from the scale to make changes in diet and physical activity as needed.[26–29] Both interventions began with 10 face-to-face group meetings over the first 4 months. The “Small Change” intervention group was introduced to a variety of strategies to reduce daily intake by approximately 100 calories each day (e.g. omitting a slice of cheese from sandwich; selecting wine rather than a mixed drink).[30–34] They were given pedometers and taught to increase their daily steps by 2000 steps per day (thereby expending approximately an additional 100 calories in activity) through changes such as using the stairs and parking further from their destination. The “Large Change” intervention was encouraged to lose 5 pounds if normal weight and 10 pounds if overweight to produce a buffer against the expected weight gains.[21] The weight loss was produced by prescribing an initial weight loss calorie goal and encouraging gradual increases in moderate to vigorous activity until achieving a goal of 250 minutes per week. At the end of the 4 months, participants in both groups were instructed to report their weight at least once a week via web or SMS. They received monthly e-mail feedback on their weight based on a color-coded system[24,25] and were either reinforced for their success, encouraged to use problem solving, or recommended additional strategies that were consistent with the small or large change approach to help reverse weight gain. Participants who gained above baseline were encouraged to contact us for additional guidance, but few participants used this option. In addition, after the initial 4 months, both interventions were offered two 4-week on-line refresher courses each year to help them reinstate the large or small change strategies. Approximately 50% of participants joined these optional refreshers, with comparable participation in Large and Small Changes.

Clinical measures

Assessments were completed by research staff members who were masked to the intervention assignments. Weight was assessed in kilograms in to the nearest .1 kg, in light clothes, without shoes, using a calibrated scale, and height was determined in centimeters to

the nearest .5 cm with a wall mounted stadiometer. Two measures of weight and height were taken and averaged; if the two measures differed by more than .2 kg or .5cm, a third measure was taken and all three were averaged. Blood pressure was assessed with a Dinamap Monitor Pro 100. Cuff size was determined by arm circumference. Three measures were taken with a 30-second interval between and averaged. Fasting blood samples were collected at baseline and two years for analysis of lipids (total cholesterol; HDL-C; triglycerides and calculated LDL-C) and glucose and insulin levels. These measures were analyzed at the Northwest Lipid Metabolism and Diabetes Laboratory at the University of Washington. We calculated HOMA-IR [(mg/dl) * (uU/mL)]. All participants brought their prescription and non-prescription medications to the assessment visits. Following each assessment, participants were sent written feedback on their baseline and subsequent values and the ideal ranges for each measure.[25]

Statistical analyses

All analyses were completed on the 471 participants who had weight and at least one CVD risk factor assessed at baseline and year 2. Baseline descriptive statistics were computed overall and by treatment group. Means and standard deviations were obtained for continuous variables; frequencies and percentages for categorical variables. Differences at baseline among the treatment groups were assessed using ANOVA for continuous measures, and chi-square tests or Fisher's exact tests were used for categorical measures depending on the adequacy of the sample size. Changes in weight and CVD risk factors from baseline to year 2 were compared among the three treatment groups using multiple linear regression; all models included clinic as a covariate. Additionally, models for blood pressure and lipids excluded participants who had ever taken medication for that condition (no participants had ever taken diabetes medication). Both the nominal p-values and the Bonferroni adjusted bounds (Type 1 error=0.05) required for significance are provided. In addition, an observational analysis was conducted to examine the effects of changes in weight on the risk factor changes. Participants were divided into 5 categories based on percent change in weight between baseline and year 2 (Lost ≥ 5%, Lost <5% to Lost >1%, Lost ≤1% to Gain ≤1%, Gain>1% to Gain <5%, Gain ≥5%. These categories were chosen because they corresponded to the clinically significant 5% weight change and the .45 kg weight gain used in the primary statistical analyses,[23] and because they resulted in similar sample sizes among the categories. CVD risk factor changes were compared across the 5 categories, adjusting for clinic and treatment group. Interactions between treatment group and percent change in weight categories were assessed. All analyses were performed using SAS® version 9.4 (SAS Institute Inc., Cary, NC).

RESULTS

Baseline characteristics of these participants are shown in Table 1. Few differences among the three groups were seen at baseline. On average, participants were 28.3 (4.4) years of age and had a BMI of 25.5 (2.6). Twenty-three percent were males and 25% reported minority race/ethnicity. In general, this was a very healthy group; on average, they had blood pressures of 110/71 mmHg, total cholesterol of 173 mg/dl, and fasting glucose of 90 mg/dl. The N=471 included in this analysis represented 78%, 77% and 81% of the participants who

were originally randomized to Control, Small Changes, and Large Changes, respectively, and contained a greater proportion of participants from UNC than from Providence relative to those not included; site was used as a covariate in subsequent analyses. No other differences at baseline or in weight changes during the trial were observed between those with 2-year data and those without these data.

Weight Change

The weight change data have been reported previously[23] but are presented here (Figure 1) to show results for the 471 participants included in this manuscript. Mean (SE) weight changes at 2 years (when blood work was completed) were +0.49 (0.37), −1.00 (0.37) and −1.58 (0.37) kg in Control, Small, and Large Changes, respectively. The overall p-value for the comparison of changes in weight from baseline to 2 years among groups was p<.001, and Control differed significantly from both Small (p=.005) and Large Changes (p<.001); the difference between Large and Small Changes was not significant at 2 years (p=.27). These weight losses are very similar to what was previously reported for the full sample.[23]

Changes in CVD risk factors by treatment group

Table 2 shows the changes in CVD risk factors from baseline to 2 years in each of the three groups. Changes in glucose (p=.05), insulin (p=.03), and the HOMA-IR ratio (p=.02) differed among the three groups, with greater improvements from baseline to two years in Large Changes than Control. However, after adjusting for either multiple comparisons or for weight change at 2 years, there were no significant differences in CVD risk factor changes among the treatment groups.

Changes in CVD risk factors by Weight Loss

We next examined the effect of percent change in weight over 2 years of follow-up on the changes in CVD risk factors. Collapsing across the three treatment groups, we divided participants into the following categories based on their percent weight change from baseline to 2 years: Lost ≥ 5% (N=103), Lost > 1 to < 5% (N=130); No change (Lost ≤ 1% to gain ≤ 1%) (N=62); gain > 1–< 5% (N=103); gain ≥ 5% (N=73). Intervention condition and clinic were both associated with the proportion of participants in each of the weight categories and were thus entered into the subsequent analyses (Supplemental Table S1). Table 3 and Figure 2 show the differences among the weight change categories for the CVD risk factors and provide the nominal p-values. With Bonferroni adjustments, larger weight loss at 2 years was significantly associated with more positive changes in HDL-C, triglycerides, insulin, HOMA-IR and SBP. There were no significant interactions between the weight change categories and intervention condition or clinic; thus, weight change per se, not the type of intervention, was associated with the changes in CVD risk factors.

DISCUSSION

This study shows that weight changes over 2 years in young adults are strongly related to changes in CVD risk factors and suggests that interventions to prevent this weight gain, especially if resulting in weight loss, can impact the health of this age group. Although the mean changes in CVD risk factors were not large in this sample which had normal CVD risk

factors at baseline, the worsening in CVD risk factors occurred over just 2 years, raising concern about the longer-term consequences of continued weight gain with age in young adults. However, our data also suggest that weight losses of just 5% or more lead to significantly greater improvements in lipids and glycemic control relative to remaining weight stable or gaining weight.

We also showed that the Large Change approach led to significant weight loss at 2 years relative to the Control group. Whereas on average participants in the Control group gained 0.49 kg, those in Large Changes lost −1.58 kg. The difference in weight loss at 2 years led to greater improvements in glucose, insulin and HOMA-IR in Large Changes than Control. However, after adjusting for multiple comparisons, none of the effects of intervention condition on risk factor change were statistically significant. Our findings suggest that interventions that result in greater weight loss relative to Control groups are needed to produce significant differences in CVD risk factors.

As seen in Figure 1, the Large Change approach was associated with marked weight loss at 4 months and then gradual regain from month 4 to 2 years. However, we found no evidence that the initial (4 month) weight change in the Large Change condition contributed either positively or negatively to the changes in glucose, insulin or insulin resistance at 2 years, independent of the weight changes at 2 years (data not shown). In contrast to these findings, in Look AHEAD, a study of 5,145 overweight/obese individuals with type 2 diabetes, half of whom were randomized to lifestyle intervention, we found that individuals who had the largest weight losses from baseline to one year had the most positive effects for glycemic control at 4 years, even if they regained this weight between years 1 and 4.[35] This difference between the outcomes in the two studies may relate to the characteristics of the participants (in particular Look AHEAD participants all had diabetes and were older) or to the magnitude and timing of weight loss/regain. Moreover, the current study, like others,[36–38] failed to find any adverse effects of weight loss followed by weight regain (or “weight cycling”) on the CVD risk factors. Rather, in this study, the effects of weight loss on CVD risk factors appeared to be due entirely to weight losses at the time of the assessment of the CVD risk factor and did not differ according to the pattern of earlier weight changes.

The Control group in this study gained 0 .49 kg over 2 years. This was less than anticipated, and less than seen in the CARDIA study.[7,8] It is possible that the initial education session, the feedback on weight and risk factors after each assessment, or the fact that all participants had joined a weight gain prevention study, blunted the weight gain. The smaller weight gain in the Control group relative to CARDIA may also reflect temporal trends in weight gain.[8] However, it is important to note that in the Control group, 46% gained weight and 39% lost weight over the two years. In contrast, in the intervention groups 34 – 36% gained weight and over half of the participants lost weight (54 and 56%). Thus, these interventions produced a favorable shift in the distribution of weight changes experienced over two years.

Strengths of this study include the large sample size, randomized design, and 2-year follow-up. The major limitations relate to the generalizability of the results to the broader population of young adults which may have more abnormal CVD risk factors and less interest in weight gain prevention than our participants. In addition, the supporting analyses

comparing changes in CVD risk factors across weight change categories has the potential to be affected by unmeasured confounds, such as family history of CVD and level of motivation to lose weight. Finally, we measured CVD risk factors only at baseline and 2 years; the differences in CVD risk factor changes among the 3 intervention groups may have been much greater at 4 months and 1 year, when the groups differed more dramatically in their weight changes. Given the impact of weight change on the changes in CVD risk factors in young adults, more research is needed to develop stronger weight gain approaches. These efforts could include strategies to increase the initial weight loss and/or prevent the subsequent weight regain that occurred in the Large Changes group or to produce additional periods of weight loss in future years by reinstating Large Changes. Alternatively, strategies to produce better maintenance of behavior changes and consequently better maintenance of weight with Small Changes might be effective longer-term if the Control group continues to gain weight as anticipated. The SNAP trial is continuing to provide intervention to the Large and Small Changes groups and will follow all participants through 6 years with repeated measures of CVD risk factors at Year 6. This will provide important information on the effects of the interventions on longer term weight change and on the association between weight change and change in CVD risk factors in these young adults.

In conclusion, these analyses show that weight change over 2 years is strongly related to changes in CVD risk factors over the same time period and suggests that interventions that reduce weight gain over time, especially if resulting in weight loss, will have positive impact on CVD risk in young adults.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

Funding Support: NIH/NHLBI Grant Numbers: U01HL090864 and U01HL090875

Disclosure: The authors report funding received from NIH/NHLBI for the conduct of this study. In addition, Dr. Wing reports consulting fees from Real Appeal, Dr. Tate reports grant funding and personal fees from Weight Watchers International, and Dr. Lewis reports grants from Novo Nordisk outside the submitted work.

SNAP RESEARCH GROUP

Clinical Sites

The Miriam Hospital/Brown Medical School Rena R. Wing, PhD[1]; Erica Ferguson, BS, RD[2]; Kristen Annis; Ryan Busha; Isabella Cassell; Eva Chen; Pamela Coward, M.Ed., RD; Jose DaCruz; Caitlin Egan, MS; Brittany James; Marie Kearns; Angelica McHugh; Kevin O'Leary; Kathy Palmer; Deborah Ranslow-Robles; Kathryn Story; Jennifer Trautvetter; Jessica Unick; Samantha Williams; Carolyn Wunsch; Annajane Yolken

The University of North Carolina at Chapel Hill Deborah Tate, PhD[1]; Karen E. Hatley, MPH, RD[2]; Candace Alick; Loneke Blackman, RD; Rachel Bordogna, RD; Melissa Crane,

PhD; Molly Diamond, MPH;. Kristen Polzien, PhD; Keneisha Quick; Brooke Tompkins Nezami, PhD; Carmina Valle, PhD;

Consultants

University of Alabama at Birmingham Cora E. Lewis, MD, MSPH[3]

University of Connecticut Amy Gorin, PhD[3]

Virginia Commonwealth University, School of Medicine Jessica G LaRose, PhD[3]

Coordinating Center

Wake Forest School of Medicine Mark A. Espeland, PhD[1]; Letitia H. Perdue, MS[2]; Judy L. Bahnson, BA, CCRP[3]; Wei Lang, PhD[3]; Cheryl Bentley; Patty Davis, BS; Katelyn Garcia, MS; Leah P. Griffin, MS; Lea Harvin, BS; Rebecca H. Neiberg, MS; Julia Robertson, BS

Central Resources Centers

Central Laboratory, Northwest Lipid Metabolism and Diabetes Research Laboratories Santica M. Marcovina, PhD, ScD[1]; Jessica Hurting[2]; Vinod Gaur, PhD

Federal Sponsors

National Heart, Lung, and Blood Institute S. Sonia Arteaga, PhD; Catherine Loria, PhD, MS, FAHA

[1]Principal Investigator

[2]Program Coordinator

[3]Co-Investigator

All other SNAP staff members are listed alphabetically by site.

References

1. Williamson DF, Kahn HS, Remington PL, Anda RF. The 10-year incidence of overweight and major weight gain in US adults. Archives of internal medicine. 1990; 150:665–72. [PubMed: 2310286]
2. Ball K, Crawford D, Ireland P, Hodge A. Patterns and demographic predictors of 5-year weight change in a multi-ethnic cohort of men and women in Australia. Public health nutrition. 2003; 6:269–81. [PubMed: 12740076]
3. Reas DL, Nygard JF, Svensson E, Sorensen T, Sandanger I. Changes in body mass index by age, gender, and socio-economic status among a cohort of Norwegian men and women (1990–2001). BMC public health. 2007; 7:269. [PubMed: 17903273]
4. James, WPT., Gill, TP. Prevention of Obesity. In: Bray, G., Bouchard, C., editors. Handbook of Obesity: ClinicalApplications. 3. New York, NY: Informa Healthcare USA, Inc.; 2008. p. 157-75.
5. Colditz GA, Willett WC, Rotnitzky A, Manson JE. Weight gain as a risk factor for clinical diabetes mellitus in women. Annals of internal medicine. 1995; 122:481–6. [PubMed: 7872581]
6. Stevens VL, Jacobs EJ, Patel AV, Sun J, Gapstur SM, McCullough ML. Body weight in early adulthood, adult weight gain, and risk of endometrial cancer in women not using postmenopausal hormones. Cancer causes & control : CCC. 2014; 25:321–8. [PubMed: 24381074]

7. Truesdale KP, Stevens J, Lewis CE, Schreiner PJ, Loria CM, Cai J. Changes in risk factors for cardiovascular disease by baseline weight status in young adults who maintain or gain weight over 15 years: the CARDIA study. International journal of obesity. 2006; 30:1397–407. [PubMed: 16534519]
8. Dutton GR, Kim Y, Jacobs DR Jr, et al. 25-year weight gain in a racially balanced sample of U.S. adults: The CARDIA study. Obesity (Silver Spring). 2016; 24:1962–8. [PubMed: 27569121]
9. Lloyd-Jones DM, Liu K, Colangelo LA, et al. Consistently stable or decreased body mass index in young adulthood and longitudinal changes in metabolic syndrome components: the Coronary Artery Risk Development in Young Adults Study. Circulation. 2007; 115:1004–11. [PubMed: 17283263]
10. Willett WC, Manson JE, Stampfer MJ, et al. Weight, weight change, and coronary heart disease in women. Journal of the American Medical Association. 1995; 273:461–5. [PubMed: 7654270]
11. Eliassen AH, Colditz GA, Rosner B, Willett WC, Hankinson SE. Adult weight change and risk of postmenopausal breast cancer. Jama. 2006; 296:193–201. [PubMed: 16835425]
12. Ford ES, Williamson DF, Liu S. Weight change and diabetes incidence: findings from a national cohort of US adults. American journal of epidemiology. 1997; 146:214–22. [PubMed: 9247005]
13. Yong LC, Kuller LH, Rutan G, Bunker C. Longitudinal study of blood pressure: changes and determinants from adolescence to middle age. The Dormont High School follow-up study, 1957–1963 to 1989–1990. Am J Epidemiol American Journal of Epidemiology. 1993; 138:973–83.
14. Manson, JE., Skerrett, PJ., Willett, WC. Obesity as a risk factor for major health outcomes. In: Bray, GA., Bouchard, C., editors. Handbook of Obesity: Etiology and Pathophysiology. New York: Marcel Dekker, Inc.; 2004.
15. Renehan AG, Flood A, Adams KF, et al. Body mass index at different adult ages, weight change, and colorectal cancer risk in the National Institutes of Health-AARP Cohort. American journal of epidemiology. 2012; 176:1130–40. [PubMed: 23186750]
16. Adams KF, Leitzmann MF, Ballard-Barbash R, et al. Body mass and weight change in adults in relation to mortality risk. American journal of epidemiology. 2014; 179:135–44. [PubMed: 24173550]
17. Hivert MF, Langlois MF, Berard P, Cuerrier JP, Carpentier AC. Prevention of weight gain in young adults through a seminar-based intervention program. International journal of obesity. 2007; 31:1262–9. (2005). [PubMed: 17356531]
18. Jeffery R, French S. Preventing weight gain in adults: the pound of prevention study. American Journal of Public Health. 1999; 89:747–51. [PubMed: 10224988]
19. Levine MD, Klem ML, Kalarchian MA, et al. Weight gain prevention among women. Obesity (Silver Spring). 2007; 15:1267–77. [PubMed: 17495203]
20. Hebden L, Chey T, Allman-Farinelli M. Lifestyle intervention for preventing weight gain in young adults: a systematic review and meta-analysis of RCTs. Obes Rev. 2012; 13:692–710. [PubMed: 22413804]
21. Kuller LH, Simkin-Silverman LR, Wing RR, Meilahn EN, Ives DG. Women's Healthy Lifestyle Project: A randomized clinical trial: results at 54 months. Circulation. 2001; 103:32–7. [PubMed: 11136682]
22. Brown T, Avenell A, Edmunds LD, et al. Systematic review of long-term lifestyle interventions to prevent weight gain and morbidity in adults. Obes Rev. 2009; 10:627–38. [PubMed: 19754634]
23. Wing RR, Tate DF, Espeland MA, et al. Innovative Self-Regulation Strategies to Reduce Weight Gain in Young Adults: The Study of Novel Approaches to Weight Gain Prevention (SNAP) Randomized Clinical Trial. JAMA internal medicine. 2016; 176:755–62. [PubMed: 27136493]
24. Wing RR, Tate DF, Gorin AA, Raynor HA, Fava JL. A self-regulation program for maintenance of weight loss. The New England journal of medicine. 2006; 355:1563–71. [PubMed: 17035649]
25. Wing RR, Tate D, Espeland M, et al. Weight gain prevention in young adults: design of the study of novel approaches to weight gain prevention (SNAP) randomized controlled trial. BMC public health. 2013; 13:300. [PubMed: 23556505]
26. Kanfer, FH., Gaelick-Buys, L. Self-management methods. In: Kanfer, FH., Goldstein, AP., editors. Helping People Change: A Textbook of Methods. 4. New York, NY: Allyn & Bacon; 1991. p. 305-60.

27. Carver, CS., Scheier, MF. Principles of Feedback Control. In: Carver, CS., Scheier, MF., editors. On the Self-Regulation of Behavior. Cambridge: Cambridge University Press; 1998. p. 10-28.
28. Carver CS, Scheier MF. Control theory: a useful conceptual framework for personality-social, clinical, and health psychology. Psychological bulletin. 1982; 92:111–35. [PubMed: 7134324]
29. Carver CS, Scheier MF. Origins and functions of positive and negative affect: A control-process view. Psychological Review. 1990; 97:19–35.
30. Hill JO, Wyatt HR, Reed GW, Peters JC. Obesity and the environment: Where do we go from here? Science. 2003; 299:853–5. [PubMed: 12574618]
31. Rodearmel SJ, Wyatt HR, Stroebele N, Smith SM, Ogden LG, Hill JO. Small changes in dietary sugar and physical activity as an approach to preventing excessive weight gain: the America on the Move family study. Pediatrics. 2007; 120:e869–79. [PubMed: 17908743]
32. Lutes LD, Daiss SR, Barger SD, Read M, Steinbaugh E, Winett RA. Small changes approach promotes initial and continued weight loss with a phone-based follow-up: nine-month outcomes from ASPIRES II. Am J Health Promot. 2012; 26:235–8. [PubMed: 22375574]
33. Rodearmel SJ, Wyatt HR, Barry MJ, et al. A family-based approach to preventing excessive weight gain. Obesity (Silver Spring). 2006; 14:1392–401. [PubMed: 16988082]
34. Damschroder LJ, Lutes LD, Goodrich DE, Gillon L, Lowery JC. A small-change approach delivered via telephone promotes weight loss in veterans: results from the ASPIRE-VA pilot study. Patient Educ Couns. 2010; 79:262–6. [PubMed: 19910151]
35. Jeffery RW, Wing RR, French SA. Weight cycling and cardiovascular risk factors in obese men and women. Am J Clin Nutr American Journal of Clinical Nutrition. 1992; 55:641–4.
36. Wing RR, Jeffery RW, Hellerstedt WL. A Prospective Study of Effects of Weight Cycling on Cardiovascular Risk Factors. Archives of internal medicine. 1995; 155:1416–22. [PubMed: 7794091]
37. Graci S, Izzo G, Savino S, et al. Weight cycling and cardiovascular risk factors in obesity. Int J Obes Relat Metab Disord. 2004; 28:65–71. [PubMed: 14647176]
38. Association of Weight Loss Maintenance and Weight Regain on 4-Year Changes in CVD Risk Factors: the Action for Health in Diabetes (Look AHEAD) Clinical Trial. Diabetes care. 2016; 39:1345–55. [PubMed: 27271190]

What is already known about this subject?

Young adults are the age group at greatest risk of weight gain. This weight gain is associated with worsening in cardiovascular risk factors.

We previously reported results from a randomized trial (Study of Novel Approaches to Weight Gain Prevention [SNAP]) showing that two self-regulation interventions (Self-regulation with Small Changes and Self-regulation with Large Changes) reduced weight gain in young adults relative to Control over an average follow-up of three years.

What does this study add?

We now show that relative to Control, the Large Change intervention produced beneficial changes in glucose, insulin, and insulin resistance (HOMA-IR) from baseline to 2 years, but after adjusting for multiple comparisons, these differences were not statistically significant.

When participants were categorized by magnitude of weight change baseline to 2 years, the categories differed significantly for change in several CVD risk factors; the interactions with treatment group were not significant

These findings suggest that changes in CVD risk factors are related to the magnitude of weight change rather than to a specific intervention. Thus, interventions that result in greater weight loss, relative to Controls, are needed to produce significant improvements in CVD risk factors.

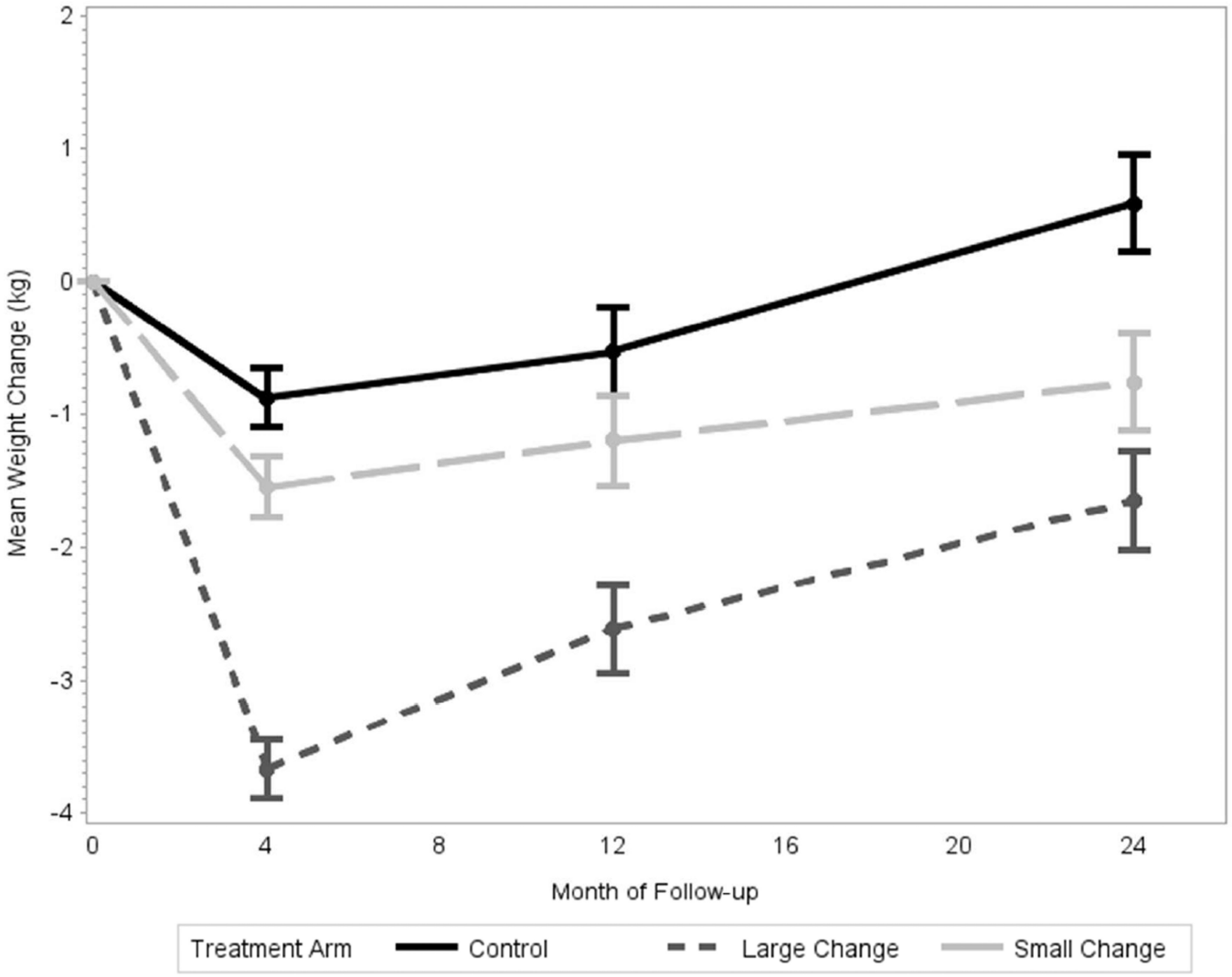

Figure 1.
Mean percent weight change from baseline to Year 2 by treatment arm

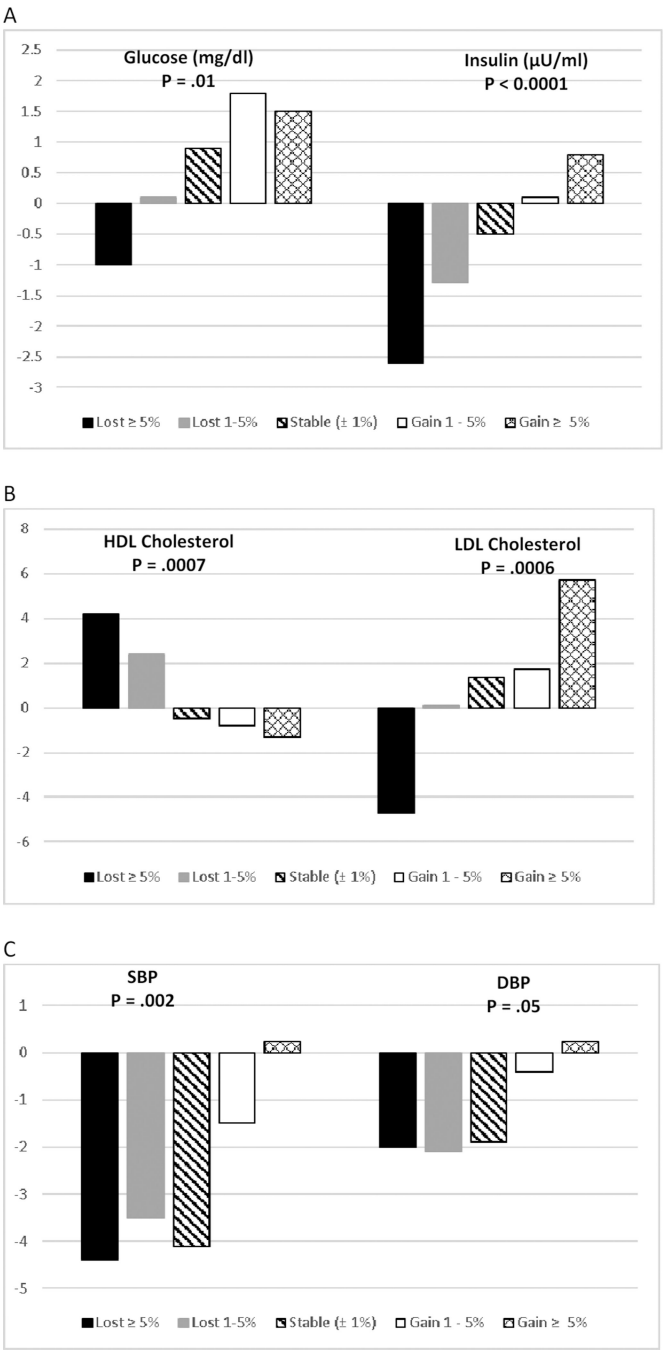

Figure 2.

Changes in cardiovascular risk factors by category of percent weight change from baseline to Year 2. Panel A show changes in glucose and insulin. Panel B shows changes in HDL-C and LDL-C. Panel C shows changes in systolic and diastolic blood pressure.

Obesity (Silver Spring). Author manuscript; available in PMC 2018 February 07.

Table 1

Baseline Data for Participants with a Year 2 Visit (N=471)

		Treatment Group			
	Overall	**Control**	**Small Changes**	**Large Changes**	**P-Value**
Number of Participants	471 (100%)	158 (33.5%)	154 (32.7%)	159 (33.8%)	
Age (yrs)	28.3 ± 4.4	28.3 ± 4.3	28.1 ± 4.6	28.5 ± 4.4	0.79
Gender: Male (%)	107 (22.7%)	37 (23.4%)	34 (22.1%)	36 (22.6%)	0.96
Clinic: Chapel Hill (%)	256 (54.4%)	85 (53.8%)	84 (54.5%)	87 (54.7%)	0.98
Brown (%)	215 (45.6%)	73 (46.2%)	70 (45.5%)	72 (45.3%)	
Race: African American (%)	52 (11.0%)	16 (10.1%)	17 (11.0%)	19 (11.9%)	0.59
White (%)	353 (74.9%)	114 (72.2%)	118 (76.6%)	121 (76.1%)	
Other/Mixed (%)	66 (14.0%)	28 (17.7%)	19 (12.3%)	19 (11.9%)	
Education: HS or More (%)	452 (96.0%)	154 (97.5%)	147 (95.5%)	151 (95.0%)	0.54
Current Smoker (%)	22 (4.7%)	10 (6.4%)	9 (6.0%)	3 (1.9%)	0.09
Weight (kg)	71.5 ± 10.8	71.5 ± 10.4	71.8 ± 10.9	71.3 ± 11.3	0.92
BMI (kg/m2)	25.5 ± 2.6	25.6 ± 2.8	25.6 ± 2.3	25.2 ± 2.6	0.24
Cholesterol (mg/dL)	173.3 ± 30.8	171.8 ± 29.2	176.9 ± 32.9	171.2 ± 30.3	0.20
HDL (mg/dL)	57.6 ± 15.6	58.4 ± 15.2	58.0 ± 16.3	56.4 ± 15.2	0.47
LDL (mg/dL)	97.5 ± 27.6	96.1 ± 27.4	100.6 ± 28.8	95.8 ± 26.4	0.23
Triglycerides (mg/dL)	91.0 ± 47.4	86.1 ± 38.5	91.7 ± 47.0	95.2 ± 54.9	0.23
Glucose (mg/dL)	89.9 ± 6.6	89.6 ± 7.0	89.9 ± 6.8	90.3 ± 5.9	0.60
Insulin (uU/mL)	8.2 ± 4.3	7.8 ± 3.8	8.3 ± 4.3	8.4 ± 4.7	0.36
HOMA ((mg/dL)*(uU/mL))	1.8 ± 1.0	1.7 ± 0.8	1.9 ± 1.0	1.9 ± 1.1	0.29
SBP (mmHg)	110.3 ± 10.9	109.6 ± 11.1	111.2 ± 10.5	110.3 ± 11.2	0.44
DBP (mmHg)	70.6 ± 8.7	70.1 ± 8.4	70.6 ± 9.4	71.1 ± 8.3	0.55
Hypertensive Meds (%)	9 (1.9%)	2 (1.3%)	4 (2.6%)	3 (1.9%)	0.65
Lipid Meds (%)	15 (3.2%)	2 (1.3%)	3 (1.9%)	10 (6.3%)	0.03

Number of Participants (%) or Mean ± Standard Deviation, Chi-Square tests were used for categorical variables, Fisher's exact tests were used for categorical variables with cell sizes less than 10, and ANOVA was used for continuous variables

Table 2

Change in Weight and CVD Risk Factors from Baseline to Year 2 By Treatment Arm

	Least Square Means (Standard Errors) By Treatment Arm				
	Control	**Small Changes**	**Large Changes**	**Nominal P-Value**	**Adjusted P-Value**
Weight change (kg)	0.49 (0.37)	−1.00 (0.37)	−1.58 (0.37)	0.001	--
Percent Weight	0.65 (0.50)	−1.26 (0.51)	−2.14 (0.50)	0.001	--
Cholesterol (mg/dl)	−0.56 (1.75)	0.74 (1.73)	1.33 (1.75)	0.73	1.00
HDL (mg/dL)	−0.09 (0.83)	1.43 (0.82)	2.12 (0.83)	0.16	1.00
LDL (mg/dL)	−0.01 (1.46)	0.16 (1.44)	1.43 (1.46)	0.75	1.00
Triglycerides (mg/dL)	−2.29 (3.77)	−4.15 (3.73)	−11.02 (3.77)	0.23	1.00
Glucose (mg/dL)	1.48 (0.49)	0.4 (0.49)	−0.18 (0.48)	0.05	0.45
Insulin (uU/mL)	−0.27 (0.33)	−0.73 (0.33)	−1.48 (0.32)	0.03	0.27
HOMA-IR ((mg/dL)*(uU/mL))	−0.03 (0.08)	−0.15 (0.08)	−0.33 (0.08)	0.02	0.18
SBP (mmHg)	−1.73 (0.69)	−3.72 (0.7)	−2.66 (0.69)	0.13	1.00
DBP (mmHg)	−0.41 (0.51)	−2.14 (0.52)	−1.33 (0.51)	0.06	0.54

Model adjusted for Treatment Arm and Clinic. With 9 CVD risk factors and Bonferroni correction for multiple comparisons (Type 1 error=0.05), a p-value of $p<0.0045$ is needed; thus, after adjustment for multiple comparisons, there were no significant differences in changes in CVD risk factors among the 3 interventions.

Obesity (Silver Spring). Author manuscript; available in PMC 2018 February 07.

Table 3

Change in CVD Risk Factors from Baseline to Two Years by Percent Weight Change

	Means (Standard Errors) By Percent Weight Loss Categories						
	Lost ≥ 5%	**Lost < 5% – Lost > 1%**	**Lost ≤ 1% – Gain ≤ 1%**	**Gain >1% – Gain <5%**	**Gain ≥ 5%**	**Nominal P-Value**	**Adjusted P-Value**
Weight change (kg)	−6.98 (0.19)	−2.1 (0.17)	−0.04 (0.24)	1.96 (0.19)	6.35 (0.23)	<0.0001	--
Percent of Weight	−9.52 (0.24)	−2.97 (0.21)	−0.04 (0.31)	2.77 (0.24)	8.93 (0.29)	<0.0001	--
Cholesterol (mg/dl)	−4.42 (2.16)	0.54 (1.91)	−0.26 (2.78)	1.16 (2.12)	6.87 (2.61)	0.03	0.27
HDL (mg/dL)	4.21 (1.02)	2.44 (0.9)	−0.53 (1.31)	−0.84 (1)	−1.24 (1.23)	0.0007	**0.0063**
LDL (mg/dL)	−4.66 (1.79)	0.14 (1.59)	1.41 (2.31)	1.79 (1.76)	5.78 (2.17)	0.006	0.054
Triglycerides (mg/dL)	−20.01 (4.60)	−10.26 (4.07)	−5.76 (5.91)	1.21 (4.52)	11.92 (5.55)	0.0002	**0.0018**
Glucose (mg/dL)	−0.99 (0.60)	0.12 (0.53)	0.93 (0.77)	1.77 (0.60)	1.56 (0.73)	0.01	0.09
Insulin (uU/mL)	−2.62 (0.39)	−1.25 (0.34)	−0.53 (0.5)	0.15 (0.39)	0.8 (0.47)	<0.0001	**0.0009**
HOMA-IR ((mg/dL)*(uU/mL))	−0.61 (0.09)	−0.28 (0.08)	−0.08 (0.12)	0.07 (0.09)	0.22 (0.11)	<0.0001	**0.0009**
SBP (mmHg)	−4.34 (0.85)	−3.45 (0.76)	−4.04 (1.08)	−1.41 (0.85)	0.31 (1.02)	0.002	**0.018**
DBP (mmHg)	−2.01 (0.64)	−2.05 (0.57)	−1.86 (0.81)	−0.35 (0.64)	0.27 (0.77)	0.05	0.45

Model adjusted for Percent Weight Change Baseline to Year 2, Treatment Arm, and Clinic. Interaction of Percent Weight Change and Treatment Arm was not significant in any of these analyses. With 9 CVD risk factors and Bonferroni adjustment for multiple comparisons, a p-value of p<0.0045 is needed; **bold font** signifies that the differences among the five weight loss categories are statistically significant after adjustment.