Published in final edited form as:

Nutr Metab Cardiovasc Dis. 2020 September 24; 30(10): 1785–1794. doi:10.1016/j.numecd.2020.06.005.

Reducing metabolic syndrome through a community-based lifestyle intervention in African American women

Abdullah Mamun^{a,*}, Heather Kitzman^{a,b,c}, Leilani Dodgen^{a,b}

^aBaylor Scott and White Health and Wellness Center, Dallas, Texas, USA

^bUniversity of North Texas Health Science Center, Fort Worth, Texas, USA

^cBaylor University, Waco, Texas, USA

Abstract

Background and Aims—Metabolic syndrome (MetS) increases the risk of cardiovascular disease and type 2 diabetes. Despite a higher prevalence of MetS in African American (AA) women, little is known about the effectiveness of lifestyle interventions in improving metabolic markers in this high-risk group. This study investigated the effectiveness of a community-based lifestyle intervention delivered by lay health coaches in reducing MetS among AA women.

Methods and Results—A cluster-randomized diabetes prevention program (DPP) was implemented in 11 churches utilizing a community-based participatory research (CBPR) approach to develop and deliver the interventions. A total of 221 adults, AA women who were overweight or obese, and did not have diabetes were included in this study. The prevalence of MetS was 42.08% before receiving the DPP intervention and 31.22% after the intervention that represented a 10.86% absolute reduction and a 25.81% relative reduction from baseline. The adjusted odds ratio (OR) of being free from MetS at post-intervention in contrast to baseline was 2.14 ($p = 0.02$). Factors that increased the odds of being free from MetS were younger age, reduction in intake of total calories, total fat, saturated and trans-fat, and dietary sodium.

Conclusion—A faith adapted lifestyle intervention held in church settings and delivered by minimally trained lay health coaches reduced the prevalence of MetS in AA women who were overweight or obese. Findings from this study can be used to translate evidence into public health programs at the community level for the prevention of type 2 diabetes and cardiovascular disease.

Keywords

Metabolic Syndrome; Lifestyle; CBPR; African American; Diabetes; Cardiovascular disease; Diet

*Corresponding author. 4500 Spring Ave, Dallas, Texas-75210, USA. Tel: +1 214 865 3153; Fax: +1 214 865 3070. abdullah.mamun@bswhealth.org (Abdullah Mamun).

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

Disclosure

Abdullah Mamun, Heather Kitzman, and Leilani Dodgen report no conflict of interests in this work.

Introduction

Metabolic syndrome (MetS) increases the risk of cardiovascular disease and type 2 diabetes mellitus [1]. As the syndrome constitutes of interrelated risk markers of metabolic origin, people with MetS also have an increased risk of developing chronic kidney disease, fatty liver disease, and have a higher rate of all-cause mortality [2–4]. The prevalence of MetS is increasing to epidemic proportions in the US. In 2012, 34.2% of US adults had MetS; that rose from 25.3% in 1994 [5]. Although the crude prevalence of MetS was similar in African-American (AA) women and non-Hispanic White women, the age and socioeconomic status adjusted odds of MetS was 20% higher in AA women than non-Hispanic White women [5]. Consequently, the prevalence of diabetes, coronary heart disease, hypertension, and stroke were higher in AA women than non-Hispanic White women [6, 7].

As a major predictor of chronic diseases, both prevention and management of MetS are emphasized in the literature. Excess abdominal adiposity and insulin resistance are the leading markers of MetS; therefore, the first-line of therapeutic choice to preventing or treating MetS is lifestyle modification through diet and physical activity [8]. Both lifestyle modification intervention and pharmacological therapy were successful in reducing the incidence of MetS. For example, the landmark Diabetes Prevention Program (DPP) reported that the incidence of MetS was reduced by 41% in the lifestyle modification group and by 17% in the metformin group compared with the placebo group for individuals with impaired glucose tolerance but without diabetes [9]. The American Heart Association and the National Heart, Lung, and Blood Institute recommend that, in addition to lifestyle modification, high-risk individuals with atherogenic dyslipidemia, arterial hypertension, glucose intolerance, and obesity should undergo clinical management for individual factors of MetS [10]. A recent meta-analysis found that a wide array of lifestyle change interventions that include high, moderate, and low-intensity interventions related to physical activity and diet help to clinically improve the majority of MetS markers [11]. Of the 15 studies included in the meta-analysis, dietary interventions were operationalized with different intensity, duration, frequency, and degree of supervision such as moderate reduction of calorie intake, very low or low-calorie diet, and/or evidence-based diet (e.g., modified DASH diet, food-based dietary guidelines) while the exercise interventions were mostly supervised and delivered with a low, moderate, or high intensity [11].

The majority of lifestyle interventions on reducing MetS through intensive physical activity utilized very restrictive dietary approaches, and/or were delivered by trained professionals [9, 12–14]. Though such interventions are effective in improving metabolic markers, adoption at the community level is a challenge. Feasibility and cost are the primary challenges in translating evidence into practice at the community level as the intervention delivery format is stringent and rigorously supervised, individualized rather than involving group-settings, and delivered by skilled professionals (registered dietitians and exercise trainers, for instance) [15]. Community-based participatory research (CBPR) offers a unique opportunity to implement lifestyle change programs since the focus is to partner with communities in an empowering process that develops solutions to community-identified needs. CBPR incorporates cultural components, community strengths, and the ecological context of communities to promote meaningful behavior change [16]. Several CBPR studies

have been effective in the prevention and management of chronic diseases through lifestyle change interventions [17]. However, there is sparse evidence of CBPR studies in reducing MetS through lifestyle change interventions in AA women who are a high-risk group. Though subgroup analyses on AA women are often reported in conjunction with overall outcome assessment, most subgroup analyses suffer from inadequate power in drawing a definitive inference [8]. To address this gap in the literature, this report presents findings in lowering the prevalence of MetS in AA women from a CBPR study, the Better Me Within (BMW) trial, that implemented a faith enhanced DPP compared to standard DPP intervention.

Methods

Study design and settings:

The BMW trial was a cluster-randomized community-based diabetes prevention study conducted in predominantly African American, Christian churches. Clusters were defined as churches and selected from the North Texas region based on recommendations from the Community Advisory Board. Eligible churches were those with parishioners who were primarily African American and agreed to provide one or two peers to facilitate the intervention. Six churches were randomized to a faith-enhanced diabetes prevention program (F-DPP) while the remaining five churches were randomized to the standard diabetes prevention program (S-DPP). A comprehensive description of the study design and implementation of the intervention is previously reported [18]. The experimental protocol and the process for obtaining informed consent from the study participants were approved by the Institutional Review Board (IRB) at the University of North Texas Health Science Center (2011–164). Informed consent was collected from all participants before completing baseline measurements.

Study Population:

A total of 221 adult AA women who were overweight or obese ($\text{BMI} > 25 \text{ kg/m}^2$) and did not have diabetes were included in this study. To exclude participants with diabetes, research staff with a clinical background reviewed medications, answers to self-report questions regarding physician diagnosis, and baseline hemoglobin A1c or fasting glucose. All enrolled participants completed baseline measurements and received the intervention, and 83% of the total sample completed follow-up measurements in all five markers of MetS.

Intervention:

The standard diabetes prevention program and the faith-enhanced diabetes prevention program were delivered by 1 or 2 trained peers (lay Health Coaches) from each church. A CBPR approach was utilized in designing the research study, as well as in recruiting churches, designing intervention components, and training Health Coaches to deliver the interventions. Health Coaches exclusively delivered the DPP intervention at the S-DPP churches, whereas, in the F-DPP churches pastors or church leaders in addition to Health Coaches delivered the intervention. Faith-enhanced adaptations developed by the Community Advisory Board in partnership with the research team. The faith-enhanced curriculum included five faith elements: 1) a mini-sermon delivered by a pastor or church

leader, 2) memory verse, 3) faith activity to connect the participant's faith with their health, 4) promises to remember, 5) prayer and key Bible scriptures integrated into each DPP session [18]. All Health Coaches were instructed to adjust examples and suggestions in the curriculum to their group based on what the Health Coach knew about food preferences, social norms, environmental barriers, socioeconomic circumstances and cultural elements of the women in their congregation to make them more relevant to their participant's everyday life. Research staff monitored the sessions monthly to assess dose, fidelity, and adherence to the curriculum of the intervention. The dosage was measured using a binary questionnaire regarding the completion of pre-determined intervention components such as weigh-in, distribution of intervention materials, checking for food and physical activity tracking, and reviewing learning objectives. Fidelity was measured using a Likert scale to assess the extent to which the intervention components were delivered. Adherence was determined by the number of intervention sessions attendance and from turning in food and physical activity tracking logs. Further details on the process evaluation were previously described [18]. Participants from each church received approximately 10 months of intervention that consisted of 16 weeks of group-based weekly sessions and six bi-monthly or monthly maintenance sessions.

Measurements:

In this study, measurements were utilized from the baseline and the end of 16-weeks of group-based sessions. Trained and blinded research staff collected the measurements. Demographic and socio-economic data were collected using a survey questionnaire. Body weight and height were measured without shoes and in light clothing using calibrated machines (Doran Digital Scale DS6100 for weight and Seca 213 Stadiometer for height). Both weight and height were measured twice and averaged. Following the NIH guideline, a pre-tested measuring tape was used to measure waist circumference twice at the top of the pelvis [19]. Fasting glucose, glycated hemoglobin A1c (HbA1c), triglycerides, and high-density lipoprotein (HDL) cholesterol were measured using point-of-care machines (Cholestech LDX system for glucose and lipids, Bayer A1cNow + Multi-Test A1c System for HbA1c) from blood samples collected by finger stick. An automated blood pressure machine (Omron Digital Blood Pressure Monitor (HEM-907XL)) was used to measure systolic and diastolic blood pressure two times in a quiet area. Current medication use was collected for hypertension, high glucose level, elevated triglycerides, and low HDL cholesterol. A food frequency questionnaire (Delta NIRI) was used to measure the dietary patterns of the participants [20]. Data were analyzed to estimate dietary intake including total energy (Kcals), fat (gram), sugar (gram), carbohydrates (gram), and sodium (milligram) [20]. The Past Week Modifiable Physical Activity Questionnaire [21] was used to collect self-reported physical activity of any type and duration over the last 7 days.

Metabolic syndrome was defined using the criteria provided by the American Heart Association and the National Heart, Lung, and Blood Institute in a scientific statement on diagnosis and management of metabolic syndrome [10]. Precisely, women with three or more of the following five risk factors were identified as having metabolic syndrome: 1) waist circumference ≥ 35 inches, 2) triglycerides ≥ 150 mg/dL or on drug treatment for elevated triglycerides, 3) HDL cholesterol < 50 mg/dL or on drug treatment for reduced

HDL, 4) systolic blood pressure ≥ 130 mmHg or diastolic blood pressure ≥ 85 mmHg or on antihypertensive drug treatment, and 5) fasting glucose ≥ 100 mg/dL or on drug treatment for elevated glucose. The 10-year-risk for cardiovascular disease was estimated using the algorithm provided by the American College of Cardiology and American Heart Association Task Force in 2013 [22].

Statistical Analyses

Data analyses were conducted in SAS 9.4. The primary outcome of this study was metabolic syndrome defined as a dichotomous variable and longitudinally measured at baseline (pre-intervention) and 16-weeks (post-intervention). Univariate summary statistics were presented to observe a percent change in outcome variables at the two measurement time points. Due to repeated measures (two times) from the same subjects and cluster randomization of the intervention conditions, a mixed-effect logistic regression model was used to estimate the intervention effects on the outcome variable from baseline to post-intervention after adjusting for cluster effect as a random effect term. The model also included a set of fixed effect variables which were intervention conditions, socio-demographic variables, physical activity, and dietary change. A parsimonious model was selected using the likelihood ratio test and Akaike Information Criterion (AIC) when the competing models were nested and not nested, respectively. As the purpose of this study was to evaluate the effect of the DPP intervention, unadjusted analyses were conducted combining both S-DPP and F-DPP groups. The adjusted model included the group assignment as a covariate to account for the effect of these two DPP interventions. The estimated intracluster correlation coefficient (ICC) was 0.49.

The power analysis for the original study was conducted to test the additional benefits of faith components in the standard DPP intervention in comparison to the standard DPP alone in weight loss [18]. As the objective of the current study was to evaluate the effect of a DPP intervention in reducing metabolic syndrome, we conducted a post-hoc power analysis to determine if this study had enough power to assess the significance of the observed effect from baseline to post-intervention. We used the **simr** package in R language to estimate the post-hoc power based on simulated data that used the observed prevalence of MetS at baseline and post-intervention for 11 clusters in two measurement time points [23]. In 100 simulations, the observed power was above 0.80 in more than 75% of the situations to find a significant effect of the intervention.

A total of 37 participants were lost to follow up. No differential characteristics were found between the lost to follow-up and continuing participants. We further utilized Little's test to ensure that the data were missing completely at random (MCAR) [24]. To avoid bias from case-wise deletion, a set of 20 multiple imputed data sets were estimated using a fully conditional specification (FCS) method to conduct intent-to-treat (ITT) analyses. Results were reported from the ITT analyses as no drastic differences were observed between the findings from ITT analyses and complete case analyses.

To adhere to both the traditional approach and recent suggestions in p-value reporting, we presented the actual p-value of null hypothesis significance testing, 95% confidence interval,

and, for some key results, the upper bound of the Bays Factors (UBF) that describes the data-based odds of the alternative hypothesis being true compared to the null hypothesis being true [25].

Results

A total of 221 participants were eligible for this study. The CONSORT diagram (Figure 1) illustrates the recruitment of churches and participants, randomization, loss to follow-up, and participation in measurement time points.

Table 1 presents the participants' characteristics in the whole sample and by MetS status at baseline and post-intervention. On average, participants classified as having MetS at baseline were significantly older (48.40 vs. 45.41, $p = 0.01$) and mostly post-menopausal (62.70% vs. 49.30%, $p = 0.03$). There were no significant differences in educational status, smoking status, baseline body weight, physical activity, and dietary pattern between participants with or without MetS at baseline.

The prevalence of MetS was 42.08% before receiving the DPP intervention (baseline) and 31.22% after the intervention that represented a 10.86% absolute reduction and a 25.81% relative reduction from baseline. Among the participants who had MetS at baseline ($n = 93$), 42.54% of those were free from the syndrome at post-intervention. In contrast, 12.72% were newly classified as having MetS at post-intervention from those who did not have MetS at baseline ($n = 128$). The odds ratio (OR) (adjusted for cluster effect and repeated measures) of being free from MetS at post-intervention in contrast to baseline was 2.41 (95% CI: 1.29 – 4.48, p -value = 0.0057, upper bound of Bays factor (UBF) = 12.49).

Table 2 presents the summary data of the five metabolic markers, 10-year CVD risk score, and changes from baseline to post-intervention. On average, participants' waist circumference (mean reduction (SD): -1.64 (2.33) inches, $p < 0.0001$), systolic blood pressure (-3.86 (13.11) mmHg, $p = 0.01$), diastolic blood pressure (-2.10 (7.97) mmHg, $p = 0.01$), triglyceride (-16.29 (42.27) mg/dL, $p = 0.01$), and 10-year CVD risk score (-0.47 (2.52), $p = 0.02$) were significantly reduced. Fasting glucose was similar to baseline while HDL cholesterol had a significant reduction; however, the subset of participants with glucose intolerance (≥ 100 mg/dL) ($n = 47$) and/or low HDL (< 50 mg/dL) ($n = 81$) had an improvement in both markers. For these subgroups, the average (SD) glucose level was reduced by 5.07 mg/dL (13.31) ($p = 0.01$) and HDL increased by 1.00 mg/dL (9.10) ($p = 0.33$) from baseline to post-intervention.

Figure 2 presents the comparative distribution of the number of MetS risk factors at baseline and post-intervention. In comparison to baseline, percentages of individuals with 1 or 2 MetS risk factors were increased whereas 3 or 4 MetS risk factors were decreased at post-intervention. Overall, the average (SD) metabolic syndrome score was reduced by 0.21 (0.75) ($p = 0.01$).

Table 3 presents the results from adjusted analysis using a mixed-effect logistic regression model. The adjusted odds of being free from MetS was 2.14 times higher at post-intervention than the odds at baseline ($p = 0.02$, UBF = 4.35). Older age was a risk factor for

MetS that significantly decreased the odds of being free from MetS (OR = 0.94, $p = 0.01$). Individuals randomized to the F-DPP intervention had a slightly higher odds of being free from MetS at post-intervention than those randomized in S-DPP intervention; however, the evidence was not statistically significant (OR = 1.43, $p = 0.48$). Participants who reduced their intake of fat (OR = 2.33, $p = 0.03$), saturated and trans-fat (OR = 8.93, $p = 0.05$), total energy (OR = 1.43, $p = 0.05$), and sodium (OR = 1.25, $p = 0.04$) increased the odds of being free from MetS compared to those who were not successful in reducing these dietary components. Participants who increased their physical activity had higher odds of being free from MetS; however, it was not statistically significant (OR = 1.61, $p = 0.26$).

Discussion

The current study provides evidence that a community-based Diabetes Prevention Program (DPP) helps to reduce the prevalence of metabolic syndrome in overweight or obese African American women without diabetes. This study also shows that minimally trained community peers (Health Coaches) can deliver the DPP intervention effectively. This finding is important since there is a need for practical and culturally relevant delivery mechanisms for the DPP intervention at the community level. Particularly because the implementation of the original DPP intervention involved components such as an individualized plan and intensive interaction with trained research staff that made it expensive and less practical in real-world settings [26, 27].

The baseline prevalence of MetS in this study was higher than the prevalence in the US population (42.1% vs. 34.2%) possibly because of the inclusion criteria of being overweight or obese. However, the prevalence of MetS reduced to 31.2% after receiving 16-weeks of DPP intervention. Lifestyle intervention trials with a longer follow-up time, such as the US DPP trial and Finnish diabetes prevention study, observed an absolute reduction of 15% and 11.4% in MetS in the lifestyle intervention group [9, 28]. Both studies used skilled professionals (nutritionists and exercise trainers, for instance) in delivering the intervention and tailored the intervention based on one's individual needs. The current study had a similar trend in reducing MetS, however using community peers, who utilized their own knowledge of their congregation to adjust curriculum discussion and examples to the preferences, social norms, and context of the African American women in their congregation was a more cost-effective and sustainable approach for community settings.

Although several lifestyle intervention studies reported significant improvements in prevention and management of MetS, the majority of these studies were comprised of Caucasians and, in those with multiethnic groups, statistical power was inadequate in drawing a definitive inference for African Americans [9, 12, 14, 29, 30]. Despite the reported differences in the prevalence of hypertension, dyslipidemia, glucose intolerance, and adiposity between AA and other racial groups [31], there are sparse data on the AA population in relation to lifestyle intervention and MetS. The Jackson Heart Study reported that the prevalence of MetS was reduced in AA adults who were more physically active [32]. The cross-sectional nature of the study and lack of dietary information limits the scope of establishing a robust association between MetS and lifestyle intervention. Another study conducted on postmenopausal AA women with a family history of breast cancer also found

that physical activity lowered the average MetS Z-score [33]. However, the generalizability of this study is limited as it only includes AA women with a family history of breast cancer.

Similar to our study, two recent meta-analyses reported that studies targeting dietary restrictions with or without physical exercise had superior improvement on metabolic syndrome than studies focused primarily on physical exercise [11, 34]. In the current study, participants received training and encouragement about healthy eating, self-monitoring of daily fat and calorie intake, ways to eat less fat and fewer calories, eating healthy while dining out, and received information on physical activity and incorporating it into their daily life [18]. At post-intervention, participants were able to substantially reduce total calories, total fat, saturated and trans-fat, carbohydrates, and sodium intake. In each case, the reduction was greater among the participants with MetS at baseline than their counterparts. We also found that the adjusted odds of being free from MetS at post-intervention were greater for those who had a reduction in dietary measures from baseline. These findings highlight the benefits of healthy eating and calorie restriction to improve the odds of being free from MetS.

Benefits of low and moderate-intensity physical exercise on metabolic markers are well established [29]. Participants in the current study increased their physical activity irrespective of their MetS. We found suggestive evidence of physical activity in reducing MetS (adjusted OR = 1.61, $p = 0.26$).

Majority of the studies that delivered lifestyle interventions (primarily diet and physical activity) observed improvements in each of the metabolic markers [9, 14, 28]. The current study in AA women also observed a significant improvement in waist circumference, blood pressure, and triglycerides for the whole sample, and improvements in fasting blood glucose and HDL cholesterol for only those who had elevated fasting blood glucose and low HDL at baseline. Given the trend of rising prevalence of MetS in the US population in general and particularly in AA women [3, 5], this evidence suggests that the delivery of evidence-based lifestyle interventions at the community level can improve this trend.

Study Limitations

This study had several limitations. First, although we observed an overall improvement in metabolic markers and a reduction in the prevalence of MetS, the absence of a no-intervention control group challenges the claim of a causal association of these favorable outcomes as a result of the DPP interventions. However, the beneficial effect of the DPP intervention on MetS is well established [9]; so, a no-intervention control group would only add incremental knowledge. On the other hand, the current study tested the translation of the DPP intervention in real-world settings by delivering a culturally tailored intervention through Health Coaches. A second limitation of this study was it only observed a short-term benefit that may arguably dissipate over time. Only a few studies investigated the long-term benefits of lifestyle interventions after 1 year or more, and each of those studies was implemented by trained professionals [9, 12, 13, 28, 34]. Therefore, long-term lifestyle interventions using a CBPR approach should be considered in future studies to develop a realistic mechanism for long-lasting benefits in reducing MetS.

To date, the strategies for chronic disease prevention through lifestyle modification in the United States have been elusive. There is no clear mechanism in place to ensure lifestyle modification for those who are at higher risk of developing cardiovascular diseases and type 2 diabetes. Despite incontrovertible evidence supporting the benefits of a healthy lifestyle to prevent chronic diseases including type 2 diabetes and cardiovascular disease, the implementation of lifestyle modification programs is suboptimal [35]. The inadequate translation of lifestyle modification interventions in practice may be partially attributed to impractical procedures, lack of cultural adaptation, nominal involvement of community members, inattention to social determinants of health and community context, and limited incentives from mainstream healthcare providers. To overcome these barriers, the Centers for Disease Control and Prevention established the National Diabetes Prevention Program (National DPP) in 2010 with the goal of translating the evidence-based findings into actions at the community-level. To add value to the success of the National DPP initiative, community-based participatory research (CBPR) addresses the mounting challenges of translating the evidence into actions by partnering with community leaders and with those directly affected by chronic diseases [17, 27]. CBPR studies have been found equally effective compared to programs implemented by trained professionals in reducing diabetes and cardiovascular disease risks [36, 37]. Therefore, by implementing a nation-wide DPP intervention through Health Coaches with minimal training and resources, the rising prevalence of MetS can be curtailed.

In conclusion, a faith adapted, culturally relevant DPP intervention held in church settings and delivered by minimally trained health coaches reduced the prevalence of metabolic syndrome among African American women who were overweight or obese but without diabetes. As lifestyle intervention is recommended to be the first line of therapeutic choice for the prevention of cardiovascular disease and type 2 diabetes, there is a need to deepen our understanding of how to deliver the intervention to the community. Lessons from the current study can be used to translate evidence into public health programs at the community level for the prevention of type 2 diabetes and cardiovascular disease.

Acknowledgements

This study was supported by a National Institutes of Health (NIH) grant R25HL125447 (to Dr. J.K.Vishwanatha) and NIH grant P20MD006882-2. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.

References:

- [1]. National Cholesterol Education Program Expert Panel on Detection, Evaluation, Treatment of High Blood Cholesterol in Adults, Third report of the national cholesterol education program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III) final report, in *Circulation*. 2002 p. 3143–421.
- [2]. Chen J, Muntner P, Hamm LL, Jones DW, Batuman V, Fonseca V, et al. The metabolic syndrome and chronic kidney disease in us adults. *Annals of Internal Medicine*. 2004; 140(3): p. 167–74. 10.7326/0003-4819-140-3-200402030-00007. [PubMed: 14757614]
- [3]. Ford ES, Giles WH, Mokdad AH. Increasing prevalence of the metabolic syndrome among u.S. Adults. *Diabetes care* 2004; 27(10): p. 2444–9. 10.2337/diacare.27.10.2444. [PubMed: 15451914]

- [4]. Hamaguchi M, Kojima T, Takeda N, Nakagawa T, Taniguchi H, Fujii K, et al. The metabolic syndrome as a predictor of nonalcoholic fatty liver disease. *Annals of Internal Medicine*. 2005; 143(10): p. 722–8. 10.7326/0003-4819-143-10-200511150-00009. [PubMed: 16287793]
- [5]. Moore JX, Chaudhary N, Akinyemiju T. Metabolic syndrome prevalence by race/ethnicity and sex in the united states, national health and nutrition examination survey, 1988–2012. *Preventing Chronic Disease*. 2017; 14 10.5888/pcd14.160287.
- [6]. Centers for Disease Control and Prevention, National diabetes statistics report, 2017. 2017, Centers for Disease Control and Prevention, U.S. Dept of Health and Human Services: Atlanta, GA.
- [7]. National Center for Health Statistics, National health interview survey. 2018, Centers for Disease Control and Prevention, U.S. Department of Health and Human Services Atlanta, GA.
- [8]. Cornier MA, Dabelea D, Hernandez TL, Lindstrom RC, Steig AJ, Stob NR, et al. The metabolic syndrome. *Endocr Rev*. 2008; 29(7): p. 777–822. 10.1210/er.2008-0024. [PubMed: 18971485]
- [9]. Orchard TJ, Tempresa M, Goldberg R, Haffner S, Ratner R, Marcovina S, et al. The effect of metformin and intensive lifestyle intervention on the metabolic syndrome: The diabetes prevention program randomized trial. *Ann Intern Med*. 2005; 142(8): p. 611–9. 10.7326/0003-4819-142-8-200504190-00009. [PubMed: 15838067]
- [10]. Grundy SM, Cleeman JI, Daniels SR, Donato KA, Eckel RH, Franklin BA, et al. Diagnosis and management of the metabolic syndrome - An American Heart Association/National Heart, Lung, and Blood Institute scientific statement. *Circulation*. 2005; 112(17): p. 2735–52. 10.1161/Circulationaha.105.169404. [PubMed: 16157765]
- [11]. van Namen M, Prendergast L, Peiris C. Supervised lifestyle intervention for people with metabolic syndrome improves outcomes and reduces individual risk factors of metabolic syndrome: A systematic review and meta-analysis. *Metabolism*. 2019; 101 10.1016/j.metabol.2019.153988.
- [12]. Bo S, Ciccone G, Baldi C, Benini L, Dusio F, Forastiere G, et al. Effectiveness of a lifestyle intervention on metabolic syndrome. A randomized controlled trial. *J Gen Intern Med*. 2007; 22(12): p. 1695–703. 10.1007/s11606-007-0399-6. [PubMed: 17922167]
- [13]. den Boer AT, Herraets IJT, Stegen J, Roumen C, Corpeleijn E, Schaper NC, et al. Prevention of the metabolic syndrome in IGT subjects in a lifestyle intervention: Results from the SLIM study. *Nutr Metab Cardiovas*. 2013; 23(11): p. 1147–53. 10.1016/j.numecd.2012.12.005.
- [14]. Poppitt SD, Keogh GF, Prentice AM, Williams DEM, Sonnemans HMW, Valk EEJ, et al. Long-term effects of ad libitum low-fat, high-carbohydrate diets on body weight and serum lipids in overweight subjects with metabolic syndrome. *Am J Clin Nutr*. 2002; 75(1): p. 11–20. 10.1093/ajcn/75.1.11. [PubMed: 11756055]
- [15]. Green LW, Brancati FL, Albright A, Primary Prevention of Diabetes Working Group. Primary prevention of type 2 diabetes: Integrative public health and primary care opportunities, challenges and strategies. *Fam Pract*. 2012; 29: p. i13–i23. 10.1093/fampra/cmz126. [PubMed: 22399542]
- [16]. Israel BA, Schulz AJ, Parker EA, Becker AB. Review of community-based research: Assessing partnership approaches to improve public health. *Annu Rev Publ Health*. 1998; 19: p. 173–202. 10.1146/annurev.publhealth.19.1.173.
- [17]. Horowitz CR, Robinson M, Seifer S. Community-based participatory research from the margin to the mainstream are researchers prepared? *Circulation*. 2009; 119(19): p. 2633–42. 10.1161/Circulationaha.107.729863. [PubMed: 19451365]
- [18]. Kitzman H, Dodgen L, Mamun A, Slater JL, King G, Slater D, et al. Community-based participatory research to design a faith-enhanced diabetes prevention program: The better me within randomized trial. *Contemp Clin Trials*. 2017; 62: p. 77–90. 10.1016/j.cct.2017.08.003. [PubMed: 28807739]
- [19]. North American Association for the Study of Obesity, National Heart, Lung, Blood Institute, and NHLBI Obesity Education Initiative. The practical guide identification, evaluation, and treatment of overweight and obesity in adults NIH publication number 00–4084. 2000: National Institute of Health 94.
- [20]. Carithers TC, Talegawkar SA, Rowser ML, Henry OR, Dubbert PM, Bogle ML, et al. Validity and calibration of food frequency questionnaires used with african-american adults in the jackson

- heart study. *J Am Diet Assoc.* 2009; 109(7): p. 1184–93. 10.1016/j.jada.2009.04.005. [PubMed: 19559135]
- [21]. Gabriel KP, McClain JJ, Schmid KK, Storti KL, Ainsworth BE. Reliability and convergent validity of the past-week modifiable activity questionnaire. *Public health nutrition.* 2011; 14(3): p. 435–42. 10.1017/S1368980010002612. [PubMed: 20843404]
- [22]. Goff DC, Lloyd-Jones DM, Bennett G, Coady S, D'Agostino RB, Gibbons R, et al. 2013 ACC/AHA guideline on the assessment of cardiovascular risk a report of the American College of Cardiology/American Heart Association task force on practice guidelines. *Circulation.* 2014; 129(25): p. S49–S73. 10.1161/01.cir.0000437741.48606.98. [PubMed: 24222018]
- [23]. Green P, MacLeod CJ. SIMR: An R package for power analysis of generalized linear mixed models by simulation. *Methods Ecol Evol.* 2016; 7(4): p. 493–8. 10.1111/2041-210x.12504.
- [24]. Little RJA. A test of missing completely at random for multivariate data with missing values. *Journal of the American Statistical Association.* 1988; 83(404): p. 1198–202. 10.2307/2290157.
- [25]. Benjamin DJ, Berger JO. Three recommendations for improving the use of p-values. *Am Stat.* 2019; 73: p. 186–91. 10.1080/00031305.2018.1543135.
- [26]. Diabetes Prevention Program Research Group. The diabetes prevention program (DPP): Description of lifestyle intervention. *Diabetes care.* 2002; 25(12): p. 2165–71. 10.2337/diacare.25.12.2165. [PubMed: 12453955]
- [27]. Boltri JM, Davis-Smith M, Okosun IS, Seale JP, Foster B. Translation of the National Institutes of Health diabetes prevention program in African American churches. *J Natl Med Assoc.* 2011; 103(3): p. 194–202. 10.1016/S0027-9684(15)30301-1. [PubMed: 21671523]
- [28]. Ilanne-Parikka P, Eriksson JG, Lindstrom J, Peltonen M, Aunola S, Hamalainen H, et al. Effect of lifestyle intervention on the occurrence of metabolic syndrome and its components in the Finnish diabetes prevention study. *Diabetes care.* 2008; 31(4): p. 805–7. 10.2337/dc07-1117. [PubMed: 18184907]
- [29]. Johnson JL, Slentz CA, Houmard JA, Samsa GP, Duscha BD, Aiken LB, et al. Exercise training amount and intensity effects on metabolic syndrome (from studies of a targeted risk reduction intervention through defined exercise). *American Journal of Cardiology.* 2007; 100(12): p. 1759–66. 10.1016/j.amjcard.2007.07.027. [PubMed: 18082522]
- [30]. Wing RR, Bahnson JL, Bray GA, Clark JM, Coday M, Egan C, et al. Long-term effects of a lifestyle intervention on weight and cardiovascular risk factors in individuals with type 2 diabetes mellitus four-year results of the look ahead trial. *Arch Intern Med.* 2010; 170(17): p. 1566–75. 10.1001/archinternmed.2010.334. [PubMed: 20876408]
- [31]. Haffner SM, Goldberg R, Ratner R, Lachin J, Temprowsa M, Orchard T, et al. Hypertension, insulin, and proinsulin in participants with impaired glucose tolerance. *Hypertension.* 2002; 40(5): p. 679–86. 10.1161/01.Hyp.0000035706.28494.09. [PubMed: 12411462]
- [32]. Newton RL, Han HM, Johnson WD, Hickson DA, Church TS, Taylor HA, et al. Steps/day and metabolic syndrome in African American adults: The Jackson Heart Study. *Preventive Medicine.* 2013; 57(6): p. 855–9. 10.1016/j.ypmed.2013.09.018.
- [33]. Dash C, Taylor TR, Makambi KH, Hicks J, Hagberg JM, Adams-Campbell LL. Effect of exercise on metabolic syndrome in black women by family history and predicted risk of breast cancer: The FIERCE Study. *Cancer.* 2018; 124(16): p. 3355–63. 10.1002/cncr.31569. [PubMed: 29975403]
- [34]. Bassi N, Karagodin I, Wang S, Vassallo P, Priyanath A, Massaro E, et al. Lifestyle modification for metabolic syndrome: A systematic review. *Am J Med.* 2014; 127(12). 10.1016/j.amjmed.2014.06.035.
- [35]. Arnett DK, Blumenthal RS, Albert MA, Buroker AB, Goldberger ZD, Hahn EJ, et al. 2019 ACC/AHA guideline on the primary prevention of cardiovascular disease: A report of the American College of Cardiology/American Heart Association task force on clinical practice guidelines. *J Am Coll Cardiol.* 2019; 74(10): p. e177–e232. 10.1016/j.jacc.2019.03.010. [PubMed: 30894318]
- [36]. Ali MK, Echouffo-Tcheugui J, Williamson DF. How effective were lifestyle interventions in real-world settings that were modeled on the diabetes prevention program? *Health Affairs.* 2012; 31(1): p. 67–75. 10.1377/hlthaff.2011.1009. [PubMed: 22232096]

- [37]. Lancaster KJ, Carter-Edwards L, Grilo S, Shen C, Schoenthaler AM. Obesity interventions in African American faith-based organizations: A systematic review. *Obesity Reviews*. 2014; 15: p. 159–76. 10.1111/obr.12207.
- [38]. Kitzman H, Mamun A, Dodgen L, King G, Slater X, DeHaven M. The better me within randomized trial: Faith-placed versus faith-based diabetes prevention program on weight loss in African American women. Unpublished results

Highlights

- Prevalence of MetS can be reduced by 25% through lifestyle intervention
- Minimally trained lay health coaches can deliver a lifestyle intervention
- Healthier selection of food is the key to preventing MetS in African American women

Figure 1. CONSORT diagram of community-based DPP trial. The figure was adopted from the Better Me Within trial [38].

Figure 2.
Prevalence of MetS risk factors at baseline and post-intervention.

Table 1.

Participant characteristics by metabolic syndrome status at baseline

	Whole sample	MetS at baseline	Did not have MetS at baseline	p-value ^a
N	221	93	128	-
Socio-demographic variables				
Age (years), mean (SD)	48.82 (11.11)	50.78 (10.95)	47.41 (11.05)	0.0147
Level of education				0.1471
High school or less, n (%)	35 (15.95)	19 (20.16)	16 (12.92)	
Technical degree or less than college, n (%)	81 (36.67)	30 (32.65)	51 (39.57)	
College degree or more, n (%)	105 (47.38)	44 (47.19)	61 (47.51)	
Menopausal status				0.0343
Pre-menopause, n (%)	100 (45.09)	35 (37.30)	64 (50.07)	
Post-menopause, n (%)	121 (54.91)	58 (62.70)	62 (49.30)	
Smoking status				0.2676
Never, n (%)	180 (81.38)	72 (77.95)	108 (83.85)	
Current or former, n (%)	41 (18.62)	20 (22.05)	21 (16.15)	
Anthropometric measures				
Body weight (lb), mean (SD)				
Baseline	215.07 (50.45)	222.30 (48.71)	209.86 (50.23)	0.0762
Post-intervention	210.24 (50.52)	217.17 (49.10)	205.25 (50.49)	0.0952
Waist circumference (in), mean (SD)				
Baseline	41.45 (6.08)	43.03 (5.49)	40.31 (6.18)	0.0045
Post-intervention	39.81 (5.85)	41.24 (5.53)	38.78 (6.04)	0.0091
BMI (kg/m ²), mean (SD)				
Baseline	36.67 (8.43)	38.03 (8.20)	35.69 (8.32)	0.0558
Post-intervention	35.85 (8.49)	37.16 (8.37)	34.91 (8.38)	0.0677
Physical activity				
Physical activity (minutes/week), mean (SD)				
Baseline	109.80 (142.50)	92.35 (132.72)	122.39 (148.02)	0.1962
Post-intervention	189.36 (133.02)	165.10 (120.77)	206.74 (160.57)	0.0843
Sedentary time (hours/week), mean (SD)				
Baseline	47.47 (10.29)	47.46 (11.91)	47.48 (12.98)	0.9942
Post-intervention	43.36 (7.53)	42.93 (9.12)	43.67 (10.09)	0.6910
Diet				
Total calories (Kcal/day), mean (SD)				
Baseline	2566.17 (1669.90)	2744.97 (1827.47)	2437.40 (1526.90)	0.2523
Post-intervention	1914.45 (1167.70)	1875.24 (982.43)	1942.53 (1323.28)	0.6703
Fat intake (g/day), mean (SD)				
Baseline	112.35 (74.69)	120.84 (82.26)	106.23 (66.69)	0.1813
Post-intervention	81.76 (51.09)	78.61 (44.54)	84.02 (59.44)	0.5138
Saturated and trans fat intake (g/day), mean (SD)				
Baseline	41.69 (28.32)	44.34 (32.47)	39.74 (24.67)	0.2450

	Whole sample	MetS at baseline	Did not have MetS at baseline	p-value ^a
Post-intervention	28.73 (20.37)	26.51 (15.64)	29.75 (22.13)	0.5412
Sodium intake (mg/day), mean (SD)				
Baseline	3997.39 (2748.30)	4280.59 (3007.09)	3793.06 (2464.28)	0.2456
Post-intervention	3000.44 (1913.24)	2854.65 (1605.08)	3105.11 (2311.51)	0.4708
Carbohydrate intake (g/day), mean (SD)				
Baseline	289.87 (211.46)	312.93 (227.09)	273.29 (196.44)	0.2953
Post-intervention	205.71 (108.23)	203.31 (121.16)	207.42 (126.53)	0.7851

^a p-value compared the statistical difference between the groups who had and did not have MetS at baseline and adjusted for cluster effects.

MetS, metabolic syndrome; SD, standard deviation; lb, pounds; in, inches; BMI, body mass index; Kcal, calories; g, gram; mg, milligram.

Table 2.

Prevalence of metabolic syndrome and its contributing factors at baseline and post-intervention, n = 221

	Baseline, mean (SD [*])	Post-intervention, mean (SD [*])	Mean (SD [*]) reduction or n (%) Change	p-value ^a (UBF)
Metabolic syndrome, n (%)	93 (42.08)	69 (31.22)	−24 (−10.86)	0.0057 (12.49)
Metabolic syndrome score	2.35 (0.98)	2.14 (0.96)	−0.21 (0.75)	0.0012 (45.58)
Waist circumference (inches)	41.45 (6.08)	39.81 (5.85)	−1.64 (2.33)	<0.0001 (>50)
SBP (mmHg)	128.20 (19.09)	124.34 (15.10)	−3.86 (13.11)	0.0016 (35.72)
DBP (mmHg)	82.22 (10.69)	80.12 (9.18)	−2.10 (7.97)	0.0037 (17.76)
Fasting blood glucose (mg/dL)	89.88 (11.54)	90.17 (9.47)	0.29 (7.09)	0.6774 (1.39)
HDL cholesterol (mg/dL)	55.87 (13.94)	53.44 (11.93)	−2.43 (6.46)	0.0012 (45.58)
Triglyceride (mg/dL)	113.63 (50.65)	97.34 (29.60)	−16.29 (42.27)	0.0007 (>50)
10-year CVD risk score	4.21 (6.86)	3.73 (5.80)	−0.47 (2.52)	0.0195 (4.79)

* Estimation of SD accounted the effect of multiple imputation in accordance with ITT analysis.

^a p-value adjusted for cluster effects and repeated measures and representing the statistical significance of mean reduction or percentage change.

UBF: upper bound of Bays factor. SBP: Systolic Blood Pressure. DBP: Diastolic Blood Pressure. HDL: High density lipoprotein. CVD: Cardiovascular disease.

Table 3.

Estimated adjusted effect of the intervention components on odds of being free from metabolic syndrome, n = 221

		Estimate of parameter (SE) ^b	Odds ratio (95% CI)	p-value (UBF)
Intercept		3.31 (1.29)		0.0106
Time	Baseline (ref)	-	-	-
	Post-intervention	0.76 (0.33)	2.14 (1.11, 4.11)	0.0222 (4.35)
Age (years)		-0.06 (0.02)	0.94 (0.90, 0.99)	0.0110 (7.42)
Intervention condition	S-DPP (ref)	-	-	-
	F-DPP	0.36 (0.50)	1.43 (0.53, 3.83)	0.4796 (1.04)
Physical activity (/week)	Less than 150 minutes (ref)	-	-	-
	More than 150 minutes	0.47 (0.42)	1.61 (0.71, 3.64)	0.2564 (1.05)
Reduction in total calorie intake (in 1000 Kcal) ^a		0.36 (0.18)	1.43 (1.00, 2.04)	0.0499 (2.46)
Reduction in total fat intake (in 100g) ^a		0.85 (0.38)	2.33 (1.10, 4.99)	0.0270 (3.77)
Reduction in saturated and trans-fat intake (in 100g) ^a		2.19 (1.13)	8.93 (0.97, 82.40)	0.0535 (2.35)
Reduction in sodium intake (in 1000 mg) ^a		0.22 (0.11)	1.25 (1.01, 1.54)	0.0387 (2.92)

^aDue to high correlation between reduction in calorie intake, fat intake, trans and saturated fat intake, and sodium intake, these variables were entered in the model one at a time along with other variables.

^bParameters were estimated using mixed effect multilevel (2-levels) logistic regression model for repeated measures with MetS (metabolic syndrome) = 'No' vs. 'Yes' as the dependent variable. Estimates of standard errors of the model parameters were adjusted for multiple imputation.

S-DPP, standard diabetes prevention program; F-DPP, faith-based diabetes prevention program; Kcal, calories; g, gram; mg, milligram; SE, standard error; CI, confidence interval; UBF, upper bound of Bays factor; ref, reference category.