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Gender Differences in Effectiveness of the Complete Health Improvement Program

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Gender Differences in Effectiveness of the Complete Health Improvement Program (CHIP)

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ABSTRACT

Objective: To determine the differential effect of gender on outcomes of the Complete Health Improvement Program, a chronic disease lifestyle intervention program.

Design: Thirty-day cohort study.

Setting: One hundred thirty-six venues around North America, 2006 to 2009.

Participants: A total of 5,046 participants (33.5% men, aged 57.9 ± 13.0 years; 66.5% women, aged 57.0 ± 12.9 years).

Intervention: Diet, exercise, and stress management.

Main Outcome Measures: Body mass index, diastolic blood pressure, systolic blood pressure, lipids, and fasting plasma glucose (FPG).

Analysis: The researchers used t test and McNemar chi-square test of proportions, at $P < .05$.

Results: Reductions were significantly greater for women for high–density lipoprotein (9.1% vs 7.6%) but greater for men for low–density lipoprotein cholesterol (16.3% vs 11.5%), total cholesterol (TC) (13.2% vs 10.1%), triglycerides (11.4% vs 5.6%), FPG (8.2% vs 5.3%), body mass index (3.5% vs 3%), diastolic blood pressure (5.5% vs 5.1%), and TC/high–density lipoprotein (6.3% vs 1.4%) but not different for systolic blood pressure (6% vs 5%). The greatest reductions were in participants with the highest baseline TC, low–density lipoprotein, triglycerides, and FPG classifications.

Conclusions and Implications: The Complete Health Improvement Program effectively reduced chronic disease risk factors among both genders, but particularly men, with the largest reductions occurring in individuals at greatest risk. Physiological or behavioral factor explanations, including differences in adiposity and hormones, dietary intake, commitment and social support, are explored. Researchers should consider addressing gender differences in food preferences and eliciting commitment and differential support modes in the development of lifestyle interventions such as the Complete Health Improvement Program.

Key Words: chronic disease, health behavior, risk factors, men, women (*J Nutr Educ Behav*. 2014;■:1-9.)

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INTRODUCTION

Chronic diseases are the leading causes of death and disability in the US, with more than half of all deaths each year attributed to heart disease, stroke, diabetes, and cancer.[1] Chronic diseases carry a major fiscal burden.

The direct (medical) and indirect (productivity) costs of cardiovascular disease alone are projected to increase from $450 billion in 2010 to more than $1 trillion by 2030.[2]

Lifestyle modification programs have been shown to be effective in the treatment of chronic disease.[3]

The Complete Health Improvement Program (CHIP) is a lifestyle modification program that is delivered by either health professionals or trained volunteers in various workplaces and community and medical settings.[4-7] Underpinning the CHIP intervention is the Theory of Planned Behavior, which asserts that behavior is driven by intentions that are in turn formed by attitudes, social norms, and perceived control.[8] The Complete Health Improvement Program includes a strong educative component to change participants' attitudes toward healthy living, group support to help foster social norms that promote a healthy lifestyle, and regular health risk assessments to increase participants' health-related self-efficacy and perceived control.

The Complete Health Improvement Program has been demonstrated to achieve meaningful reductions in

Table 1. Mean Changes in Selected Risk Factors for Men and Women From Baseline to 30 Days, North America, 2006–2009

Risk Factor	Men					Women				
	n	Baseline (mean ± SD)	30 d (mean ± SD)	Mean Change (95% confidence interval)	Change (%)	n	Baseline (mean ± SD)	30 d (mean ± SD)	Mean Change (95% confidence interval)	Change (%)
Weight, lb	1,549	214.1 ± 50.4	206.6 ± 47.7	−7.5 (−7.8, −7.2)*	3.5	3,034	181.2 ± 46.7	175.8 ± 45.0	−5.4 (−5.6, −5.3)*	3.0[a]
Body mass index, kg/m²	1,540	31.0 ± 6.7	30.0 ± 6.4	−1.1 (1.1, 1.0)*	3.5	2,972	31.0 ± 7.6	30.1 ± 7.3	−0.9 (−1.0, −0.9)*	3.0[a]
Systolic blood pressure, mm Hg	1,542	135.7 ± 18.6	128.2 ± 16.1	−7.5 (−8.2, −6.7)*	5.5	3,006	132.1 ± 19.2	125.4 ± 16.7	−6.7 (−7.2, −6.1)*	5.1
Diastolic blood pressure, mm Hg	1,542	81.6 ± 11.0	76.8 ± 10.0	−4.8 (−5.3, −4.3)*	5.9[b]	3,008	78.9 ± 11.0	75.1 ± 9.8	−3.8 (−4.2, −3.4)*	4.8[b]
Total cholesterol, mg/dL	1,563	180.8 ± 40.8	157.0 ± 35.2	−23.8 (−24.1, −22.4)*	13.2[a]	3,090	200.0 ± 40.7[c]	179.7 ± 36.8	−20.3 (−21.2, −19.3)*	10.1[a]
Low-density lipoprotein, mg/dL	1,512	124.6 ± 61.3	104.2 ± 50.8	−20.4 (−21.8, −18.9)*	16.3[a]	3,036	134.3 ± 62.1[c]	118.8 ± 56.2	−15.5 (−16.4, −14.5)*	11.5[a]
High-density lipoprotein, mg/dL	1,561	46.1 ± 21.5	42.6 ± 19.8	−3.5 (−3.9, −3.1)*	7.6	3,091	59.2 ± 26.6[c]	53.8 ± 23.8	−5.4 (−5.7, −5.1)*	9.1[a]
Ratio of total cholesterol to high-density lipoprotein	1,561	4.43 ± 1.62	4.15 ± 1.46	−0.28 (−0.32, −0.24)*	6.3	3,088	3.80 ± 1.36	3.75 ± 1.34	−0.05 (−0.08, −0.03)*	1.4[a]
Triglycerides, mg/dL	1,560	153.8 ± 102.2	136.3 ± 80.4	−17.5 (−21.0, −14.1)*	11.4	3,088	138.1 ± 82.7	130.3 ± 71.4	−7.8 (−9.7, −5.8)*	5.6[a]
Fasting plasma glucose, mg/dL	1,547	105.5 ± 32.4	96.9 ± 22.3	−8.7 (−9.7, −7.6)*	8.2	3,038	99.1 ± 26.8	93.8 ± 20.2	−5.3 (−5.9, −4.7)*	5.3[a]

*P < .001, t test; [a]Difference in change between men and women was significant at P < .001; [b]Difference in change between men and women was significant at P < .05; [c]Baseline levels were higher for women than men at P < .001.

Note: Percent change = [(mean at 30 days − mean at baseline) / mean at baseline] × 100.

selected risk factors—body mass index (BMI), blood pressure (BP), total cholesterol (TC), low-density lipoprotein (LDL), triglycerides (TG), and fasting plasma glucose (FPG)—for cardiovascular disease and type 2 diabetes mellitus among large cohorts from several countries including the US.[4-7] Furthermore, the reduction in risk factors and self-reported adherence to the health behaviors promoted in CHIP have been found to occur over the long-term, which indicates that CHIP is a useful intervention for enabling behavior change.[9,10] A detailed analyses of the differential responsiveness of males and females to the CHIP intervention has not been performed. Differences in the responsiveness of men and women might be expected because gender differences exist in a number of health outcomes and have been attributed to hormonal, behavioral, social and/or psychological factors.[11-15]

The aim of the current study was to examine differences in the short-term responsiveness of men and women to the CHIP intervention, as measured by changes in selected chronic disease risk factors.

METHODS

Study Participants

The CHIP intervention, which was previously described in detail,[4-7] was delivered to 5,046 participants who had self-selected to participate in the program between January, 2006 and October, 2009. A total of 176 CHIP interventions (mean group size, 29; range, 3−228) were conducted at 136 venues throughout North America over this period. There were no inclusion or exclusion criteria other than the participant being able to pay a $200 program cost. By comparison, weight loss diets typically cost more than this per annum.[16] Participants were invited to attend the intervention through word of mouth invitation, local media avenues, and referrals from health care providers. As indicated by baseline characteristics (Tables 1 and 2), participants comprised a more at-risk and ill demographic compared with the general North American population.[17,18] The Avondale College of

Table 2. Changes in Risk Factor Levels Within 30 Days, by Gender and Initial Risk Factor Classification, North America, 2006−2009

Risk Factor	**Men**					**Women**				
	Baseline, n (%)[a]	30 d, N (%)[a,b]	Baseline (mean ± SD)	30 d (mean ± SD)[c]	Mean Change (95% confidence interval) (%)	Baseline, n (%)[a]	30 d, N (%)[a,b]	Baseline (mean ± SD)	30 d (mean ± SD)[c]	Mean Change (95% confidence interval) (%)
Body mass index, kg/m²	χ^2 = 187.71, P < .001					χ^2 = 300.53, P < .001				
< 18.5	9 (0.6)	9 (0.6)	17.5 ± 1.1	17.4 ± 0.9	−0.1 (−0.5, 0.3) (0.4)	18 (0.6)	23 (0.8)	17.6 ± 0.8	17.5 ± 0.7	−0.2 (−0.4, 0.1) (1.0)
18.5−24.9	230 (14.9)	317 (20.6)	23.2 ± 1.4	22.7 ± 1.3	−0.5 (−0.5, −0.4)* (2.0)	641 (21.6)	777 (26.1)	22.6 ± 1.7	22.0 ± 1.6	−0.5 (−0.6, −0.5)* (2.3)
25−29.9	547 (35.5)	570 (37.0)	27.5 ± 1.4	26.6 ± 1.5	−1.0 (1.0, −0.9)* (3.4)	841 (28.3)	867 (29.2)	27.4 ± 1.4	26.6 ± 1.5	−0.8 (−0.9, −0.8)* (3.0)[d]
≥ 30	754 (49.0)	644 (41.8)	36.2 ± 5.9	34.8 ± 5.6	−1.4 (−1.5, −1.3)* (3.8)	1,472 (49.5)	1,305 (43.9)	36.9 ± 6.2	35.7 ± 6.1	−1.2 (−1.2, −1.1)** (3.2)[d]
Systolic blood pressure, mm Hg	χ^2 = 267.33, P < .001					χ^2 = 387.43, P < .001				
< 120	261 (16.9)	409 (26.5)	110.8 ± 6.3	115.3 ± 13.0	4.4 (2.8, 6.1)* (−4.0)	749 (24.9)	1,107 (36.8)	109.8 ± 7.6	111.7 ± 11.5	1.9 (1.1, 2.6)* (−1.7)[e]
120−139	680 (44.1)	793 (51.4)	129.0 ± 5.9	124.0 ± 11.3	−5.1 (−5.9, −4.3)* (4.0)	1,281 (42.6)	1,317 (43.8)	128.5 ± 5.8	124.0 ± 12.6	−4.4 (−5.1, −3.7)* (3.4)
140−160	429 (27.8)	274 (17.8)	147.5 ± 5.9	135.0 ± 12.4	−12.6 (−13.8, −11.3)* (8.5)	695 (23.1)	467 (15.5)	147.0 ± 5.8	134.0 ± 13.6	−13.0 (−14.1, −12.0)* (8.9)
> 160	172 (11.2)	66 (4.3)	170.6 ± 12.0	148.3 ± 18.3	−22.4 (−25.0, −19.7)* (13.1)	281 (9.3)	115 (3.8)	170.6 ± 11.9	146.7 ± 17.2	−23.9 (−26.1, −21.8)* (14.0)
Diastolic blood pressure, mm Hg	χ^2 = 263.87, P < .001					χ^2 = 324.93, P < .001				
< 80	599 (38.8)	904 (58.6)	71.1 ± 6.1	71.4 ± 8.9	0.2 (−0.5, 1.0) (−0.3)	1,486 (49.4)	1,937 (64.4)	70.3 ± 6.5	70.7 ± 8.8	0.4 (−0.1, 0.9) (−0.6)
80−89	571 (37)	465 (30.2)	83.3 ± 3.0	77.2 ± 7.6	−6.1 (−6.8, −5.5)* (7.4)	1,001 (33.3)	855 (28.4)	83.2 ± 2.9	77.5 ± 8.0	−5.7(−6.2, −5.2)* (6.8)
90−100	282 (18.3)	147 (9.5)	92.9 ± 3.0	83.6 ± 8.3	−9.3 (−10.3, −8.4)* (10.0)	406 (13.5)	174 (5.8)	92.9 ± 3.0	82.1 ± 8.4	−10.7 (−11.6, −9.9)* (11.6)
> 100	90 (5.8)	26 (1.7)	104.7 ± 6.9	89.1 ± 10.5	−15.6 (−17.7, −13.6)* (14.9)	115 (3.8)	42 (1.4)	104.4 ± 7.7	86.8 ± 10.2	−17.5 (−19.6, −15.4)* (16.8)
Total cholesterol, mg/dL	χ^2 = 539.01, P < .001					χ^2 = 991.32, P < .001				
< 160	484 (31%)	866 (55.4)	137.2 ± 16.4	127.4 ± 22.9	−9.8 (−11.5, −8.0)* (7.1)	473 (15.3)	946 (30.6)	142.5 ± 14.3	137.1 ± 23.0	−5.5 (−7.3, −3.6)* (3.8)[e]
160−199	617 (39.5)	515 (33)	178.6 ± 11.0	156.8 ± 22.6	−21.8 (−23.4, −20.1)* (12.2)	1,161 (37.6)	1,304 (42.2)	180.8 ± 11.1	167.3 ± 22.3	−13.5 (−14.7, −12.3)* (7.5)[d]
200−239	336 (21.5)	155 (9.9)	215.9 ± 11.1	180.4 ± 25.8	−35.5 (−38.2, −32.9)* (16.5)	950 (30.8)	650 (21)	217.5 ± 11.3	192.7 ± 24.6	−24.8 (−26.3, −23.3)* (11.4)[d]
240−280	103 (6.6)	20 (1.3)	255.6 ± 12.0	203.3 ± 32.9	−52.3 (−59.1, −45.5)* (20.5)	400 (13)	165 (5.3)	254.9 ± 10.6	218.1 ± 28.9	−36.9 (−39.7, −34.1)* (14.5)[d]
> 280	22 (1.4)	6 (0.4)	314.4 ± 37.3	240.3 ± 51.0	−74.3 (−98.3, −50.3)* (23.6)	104 (3.4)	23 (0.7)	306.3 ± 24.6	247.9 ± 42.2	−58.4 (−67.1, −49.7)* (19.1)
Low-density lipoprotein, mg/dL	χ^2 = 405.43, P < .001					χ^2 = 629.70, P < .001				
< 100	565 (37.4)	844 (55.8)	78.3 ± 16.0	70.6 ± 19.3	−7.7 (−9.2, −6.3)* (9.8)	837 (27.6)	1,226 (40.4)	81.1 ± 14.1	77.8 ± 21.5	−3.3 (−4.6, −2.1)* (4.1)[d]
100−129	434 (28.7)	394 (26.1)	113.3 ± 8.3	96.4 ± 19.5	−17.0 (−18.8, −15.1)* (15.0)	951 (31.3)	967 (31.9)	114.6 ± 8.7	103.9 ± 20.2	−10.7 (−11.8, −9.5)* (9.3)[d]
130−159	276 (18.3)	131 (8.7)	141.8 ± 8.5	114.3 ± 21.4	−27.5 (−29.9, −25.0)* (19.4)	624 (20.6)	451 (14.9)	142.6 ± 8.5	122.5 ± 21.4	−20.2 (−21.8, −18.6)* (14.2)[d]
160−189	87 (5.8)	46 (3)	171.8 ± 7.8	136.8 ± 26.6	−35.0 (−40.8, −29.1)* (20.4)	287 (9.5)	147 (4.8)	172.1 ± 8.3	142.8 ± 27.1	−29.3 (−32.3, −26.3)* (17.0)[d]
≥ 190	150 (9.9)	97 (6.4)	272.6 ± 66.3	216.3 ± 67.2	−56.4 (−64.4, −48.4)* (20.7)	337 (11.1)	245 (8.1)	274.5 ± 68.8	236.0 ± 74.9	−38.5 (−43.5, −33.5)* (14.0)[d]
High-density lipoprotein, mg/dL	χ^2 = 148.51, P < .001					χ^2 = 378.12, P < .001				
< 40 men, < 50 women	728 (46.6)	925 (59.3)	32.9 ± 4.8	31.6 ± 6.5	−1.3 (−1.7, −0.9)* (3.8)	467 (15.1)	750 (24.3)	34.8 ± 4.1	34.4 ± 7.3	−0.4 (−1.0, 0.2)* (1.2)
40−59	622 (39.8)	453 (29)	46.6 ± 5.3	42.5 ± 7.8	−4.1 (−4.6, −3.6)* (8.9)	1,581 (51.1)	1,583 (51.2)	49.1 ± 5.5	45.5 ± 7.7	−3.6 (−3.9, −3.3)* (7.3)
≥ 60	211 (13.5)	183 (11.7)	90.4 ± 26.4	81.1 ± 26.2	−9.3 (−11.4, −7.3)* (10.3)	1,043 (33.7)	758 (24.5)	85.6 ± 30.4	75.2 ± 28.9	−10.4 (−11.1, −9.7)* (12.2)

(continued)

Table 2. Continued

Risk Factor	Men					Women				
	Baseline, n (%)[a]	30 d, N (%)[a,b]	Baseline (mean ± SD)	30 d (mean ± SD)[c]	Mean Change (95% confidence interval) (%)	Baseline, n (%)[a]	30 d, N (%)[a,b]	Baseline (mean ± SD)	30 d (mean ± SD)[c]	Mean Change (95% confidence interval) (%)
Triglycerides, mg/dL	$\chi^2 = 67.92$, $P < .001$					$\chi^2 = 45.09$, $P < .001$				
< 150	967 (62)	1,052 (67.4)	97.2 ± 29.2	99.7 ± 43.6	2.5 (0.1, 5.0)* (−2.6)	2,075 (67.2)	2,170 (70.3)	94.6 ± 29.8	99.5 ± 40.8	4.9 (3.4, 6.3) (−5.2)
150–199	251 (16.1)	260 (16.7)	171.9 ± 13.8	154.3 ± 49.3	−17.6 (−23.9, −11.3)* (10.2)	498 (16.1)	499 (16.2)	171.8 ± 13.8	159.6 ± 54.2	−12.2 (−16.9, −7.5)* (7.1)
200–499	318 (20.4)	241 (15.4)	274.7 ± 66.5	216.5 ± 88.1	−58.2 (−67.1, −49.2)* (21.2)	496 (16.1)	415 (13.4)	267.8 ± 59.6	222.3 ± 77.5	−45.5 (−51.8, −39.1)* (17.0)
≥ 500	24 (1.5)	7 (0.4)	647.9 ± 120.6	360.7 ± 153.9	−287.2 (−367.6, −206.8)* (44.3)	19 (0.6)	4 (0.1)	618.1 ± 106.3	327.0 ± 166.5	−291.1 (−375.5, −206.7)* (47.1)
Fasting plasma glucose, mg/dL	$\chi^2 = 124.73$, $P < .001$					$\chi^2 = 148.36$, $P < .001$				
< 110	1,157 (74.8)	1,298 (83.9)	92.1 ± 9.8	89.2 ± 10.93	−2.9 (−3.5, −2.3) (3.2)	2,517 (82.9)	2,690 (88.5)	90.2 ± 9.6	88.2 ± 10.3	−2.0 (−2.4, −1.7)* (2.2)[e]
110–125	163 (10.5)	120 (7.8)	116.6 ± 4.8	105.4 ± 19.4	−11.3 (−14.1, −8.4)* (9.6)	224 (7.4)	179 (5.9)	115.7 ± 4.6	106.3 ± 11.9	−9.3 (−10.8, −7.9)* (8.1)
> 125	227 (14.7)	129 (8.3)	166.4 ± 43.9	130.1 ± 32.3	−36.3 (−41.1, −31.5)* (21.8)	297 (9.8)	169 (5.6)	162.3 ± 40.9	132.4 ± 36.1	−29.9 (−33.9, −25.8)* (18.4)

*$P < .001$, t test; [a]McNemar chi-square test; [b]Number of participants in that risk category at 30 days; [c]Mean for each biometric risk category at 30 days; [d]Difference in change between men and women significant at $P < .001$; [e]Bonferroni correction applied: difference in change between men and women at $P < .017$ with 3 risk categories, $P < .012$ with 4 risk categories, and $P < .010$ with 5 risk categories.

Higher Education Ethics Committee approved the study.

Facilitator Information

The CHIP programs were conducted by volunteer facilitators sourced primarily through the Seventh-Day Adventist Church, who had an interest in positively influencing the health of their local community. All volunteers were required to undergo 2 days of training to learn about the CHIP intervention and develop group facilitation skills. There were no educational requirements or selection criteria for the volunteer facilitators. The educational component of the CHIP intervention was presented through a set of prerecorded videos. The role of the volunteer facilitator was to organize the meetings and facilitate discussion.

Description of CHIP

The CHIP intervention, founded in 1986, involved 16 group sessions conducted in a community setting over 30 days.[5,6] Sessions were structured around a process of learning, experiencing, and reflecting. Each of the 16-group sessions, delivered 4 d/wk for 30 days, was approximately 1 hour in duration, with approximately half of the session involving the viewing of a prerecorded educational video and the other half constituting group activities such as cooking demonstrations, physical exercises, and discussion. Participants were educated on the etiology of chronic disease and the benefits of positive lifestyle choices, with particular attention given to diet and physical activity. The program advocated a predominately whole-food, plant-based diet that is high in nutrient density and fiber yet low in energy density. As such, the program did not restrict the volume of food that participants consumed. Sessions on overcoming barriers to change, developing emotional intelligence, and providing participants with strategies (self-monitoring, goal setting, and problem solving, including addressing unsupportive social and physical influences) for behavior change maintenance were also included. Use of the supplied resources meant that the

program delivery was consistent in each location.

The program encouraged and supported participants to move toward a low-fat (< 15% of calories from fat), ad libitum plant-based diet over 30 days, with emphasis on the daily whole-foods consumption of grains, legumes, fruits and vegetables, and water (2–2.5 L) while limiting intake of added sugar, sodium, and cholesterol (40 g, 2,000 mg, and 50 mg, respectively). In addition, the program advocated that participants engage in 30 minutes of moderate physical activity daily and practice stress management techniques. These behaviors have previously been shown to be efficacious for preventing and treating chronic disease.[3] Participants were deemed to have completed the intervention if they attended a minimum of 13 of the 16 sessions and completed the baseline and 30-day assessments. After completion, participants were invited to attend ongoing monthly follow-up sessions to reinforce lifestyle behavior changes and build a network of support and ongoing education. Biometric data were not collected following the 30-day assessment, and so evaluation of the biometric outcomes during the follow-up sessions is beyond the scope of the current study.

Data Collection and Reporting

Before participating in the CHIP intervention (baseline) and again at its conclusion (postintervention), participants' height, weight, systolic blood pressure (SBP), and DBP were taken, and fasting (12-hour) blood samples were collected by registered health professionals. The same scales and sphygmomanometer were used for taking measurements at baseline and again at 30 days. Blood samples were collected by trained phlebotomists and analyzed by local pathology laboratories for TC, LDL, high-density lipoprotein (HDL), TG, and FPG levels. Self-report health conditions were also collected at baseline but not at follow-up.

The researchers analyzed data using SPSS Statistics (version 19, IBM Corp, Armonk, NY, 2010). The biometric data was initially explored for outliers that were beyond reasonable physiological values, and removed. The extent of the changes (from baseline to postintervention) in the biometric measures was assessed for males and females separately, using paired t tests. Findings were reported as mean ± SD, because the data were deemed to approximate normality because of the large sample size.[19] Only participants with complete pre- and postintervention data were included for analysis of each biometric. About 7% of men and women (113 men and 224 women) did not have follow-up data for any biometric at 30 days. Furthermore, some participants did not have follow-up data for some risk factors; hence, there is variation in the number (n) listed in Table 1 between the biometrics. McNemar chi-square test was used to determine changes in the distribution of participants by gender, across the various risk factor categories. Participants' weight was characterized using standard BMI cut points for normal, overweight, and obese[20]; BP was classified using the Fifth Joint National Committee for Hypertension guidelines[21]; and FPG was characterized according to conventional normal, impaired, and diabetic levels.[22] The National Cholesterol Education Program Adult Treatment Panel III classification system[22] was used to categorize the participants for all risk factors except total cholesterol, for which the Framingham risk classification[23] was used. The Framingham classification includes 5 cholesterol categories compared with only 3 in the National Cholesterol Education Program Adult Treatment Panel III classification system, and thus allowed a more detailed analysis of the effect of the intervention on the highest-risk participants. Metabolic syndrome at baseline and after intervention was classified according to the harmonized definition.[24] Participants were deemed as having this syndrome if they met ≥ 3 of the defining criteria.[24] P < .05 was considered significant. To reduce the Type 1 error that can occur when simultaneous tests are performed in a data set, Bonferroni correction was applied to each biometric separately. Because there was a different number of risk category comparisons for each biometric, the correction applied was 0.05/n, where n was the number of categories within each biometric.

RESULTS

Of the 5,046 participants, 33.5% (n = 1,690) were men and 66.5% (n = 3,392) were women, with only 21% reporting to be Seventh-Day Adventists. The men (57.9 ± 13.0 years) were marginally older than the women (57.0 ± 12.9 years) (P = .027) and more men than women reported being smokers (2.6% vs 2.0%; P < .001) and married (85% vs 68%; P < .001). There were also baseline differences in health history, with more men commencing the intervention with diagnosed heart conditions such as angina (4.9% vs 2.7%; P < .001), myocardial infarction (6.6% vs 3.0%; P < .001), coronary bypass (4.1% vs 1.2%; P < .001), and diabetes (16.6% vs 12.7%; P < .001). There were no differences for history of stroke (2.3% vs 1.8%; P = .241) or cancer (9.0% vs 8.5%; P = .556).

Men had higher baseline SBP, DBP, TG, and FPG than women (P < .001), whereas the women had higher baseline TC, LDL, and HDL (P < .001) (Table 1). There was no difference in baseline BMI between the men and women (P = .867) (Table 1).

Both genders achieved significant reductions in all biometrics after the intervention, but the reductions were greater among men than women for all biometrics except SBP, for which there was no difference, and HDL, for which women had greater reductions than the men (Table 1). The TC to HDL ratio also significantly decreased within 30 days for all participants combined and the reduction was greater for men than for women (Table 1). The proportion of men and women classified with metabolic syndrome at baseline decreased significantly (P < .001) at 30 days (males, 46.5% to 37.5%; females, 39.2% to 33.1%).

Stratification of risk factors showed substantive changes in the distribution of men and women across the various categories, with the largest reductions among participants with the highest risk classifications at baseline (Table 2). More men than women presenting with the highest category SBP (> 160 mm Hg), DBP (> 100 mg/dL), BMI (> 30 kg/m²), and LDL (≥ 190 mg/dL) reduced their risk characterization at 30 days (62% vs 59%, 71% vs 63%, 15% vs 11%, and 35% vs

27%, respectively) (Table 2). However, in the highest risk categories for TC (> 280 mg/dL), TG (≥ 500 mg/dL), and HDL (< 40 mg/dL), more women than men (78% vs 73%, 79% vs 71%, and 61% vs 27%, respectively) were no longer in these risk categories (Table 2). For FPG levels indicative of diabetes (> 125 mg/dL), equal proportions of men and women (43%) reduced their risk factor categorization in the 30 days.

An analysis of mean changes in the various biometric categories also indicated that men tended to achieve greater improvements than the women. For BMI, men experienced greater decreases than women in the baseline overweight and obese categories. For SBP and FPG, only in 1 baseline category (< 120 mm Hg and < 110 mg/dL, respectively) was the mean change greater for men than women. For other risk factors, men experienced greater mean change than women in a number of baseline categories: LDL, all categories, except 160–189 mg/dL; and TC, all categories, except the highest (Table 2).

DISCUSSION

Substantial reductions in selected risk factors were achieved in 30 days using the CHIP lifestyle intervention, with greater reductions seen among the men than women. In 30 days, the majority of men in the highest risk classifications for TC, LDL, TG, and FPG, but only TG for women, were able to show improvement by more than 20%. Results from the Pritikin program also showed that men achieved greater reductions in chronic disease risk factors from a lifestyle intervention than women.[25] A seemingly adverse outcome of the CHIP intervention is the reduction in HDL among both men and women, which has also been observed in other lifestyle interventions that promote a plant-based eating pattern.[3] However, this reduction in HDL is not considered detrimental to the risk of chronic disease, as discussed by Kent et al.[26] The reason for the difference observed between men and women, although not explored in this study, could be speculated from the literature to relate to physiological and/or behavioral factors.

In terms of physiological factors, differences in adiposity distribution may offer an explanation. The propensity for men to store fat in the abdominal region, which is more metabolically active and therefore easier to remove than fat on the hips and thighs, which is more common in women, may offer a physiological explanation.[27,28] It is well established that central obesity increases the risk of type 2 diabetes, metabolic syndrome, cardiovascular disease, dyslipidemias, and hypertension.[29] Other physiological explanations may include the differing hormonal profile between men and women and the heavier weight of men; heavier people tend to lose more weight.[30,31] Furthermore, men are also more likely to have greater muscle mass and therefore a higher metabolic rate than women.[32,33]

In terms of the other metabolic risk factors, differences in food preferences and the amount of food eaten by men and women may offer physiological explanations for the observed differences at 30 days. Diets high in whole-plant foods and low in red and processed meat may provide benefits for the prevention and treatment of not only obesity but other chronic health problems, including type 2 diabetes and cardiovascular disease.[34] Plant foods are rich in fiber and a range of phytochemicals and antioxidants, which are believed to confer these health benefits.[35] Because women tend to eat more fruit and vegetables than do men and men's diets tend to be higher in red and processed meats and lower in fiber,[35,36] there is greater scope for changes in men's diets to include more plant foods. However, this study did not collect information on dietary intake to examine this more closely. The benefit of the greater muscle mass of men also means they have a greater requirement for dietary energy than women.[37] Consequently, the higher intake of health-promoting plant foods required to meet the energy demands of men, together with increases in physical activity, as promoted by the CHIP intervention, are therefore expected to more quickly ameliorate the adverse effect of a previously poor lifestyle compared with the lower intakes required by women.

Women tend to participate in commercial weight loss programs such as Weight Watchers or be treated for weight problems by a primary care provider more so than men.[30] Indeed, women are more likely to be aware of or concerned that they are overweight.[30,38] Certainly, two thirds of the CHIP participants were women. The way men and women engage with lifestyle interventions may offer a behavioral explanation for why men might achieve better outcomes. Men seem to approach making the commitment to change differently from women.[39] Men are more inclined to commit to a program if the benefits outweigh the costs.[39] Indeed, once men have made a decision, they are more likely to complete a program that is prescriptive[40] and achieve their goals,[41] particularly if advised by a health professional.[30] On the other hand, women are inclined to commit to interventions for social reasons (trust, interaction, and obligations to significant others).[39] However, whereas women tend to have higher expectations and be more eager to initially change behavior than men,[39] they are also inclined to be more easily disappointed and risk dropping out before reaching their target.[42] Therefore, although the literature suggests men seem to take longer to engage with the program, there is anecdotal evidence that once they have assessed and evaluated the evidence and made a commitment, they appear to achieve better results in a shorter time than women (Professor G. Egger, Founder of Gut Busters, personal communication). However, this could not be explored in this study because information on commitment was not collected. The difference in men's and women's engagement in the CHIP intervention program needs to be explored in further research.

Having supportive relationships may be a behavioral factor that explains the greater effect on men than women observed in this study. Men who are married are less likely to engage in unhealthy, high-risk behaviors and therefore experience better health through the influence of their wives.[43] In the current study, there is anecdotal evidence also supported by the literature[44] that men are persuaded to attend CHIP with their

wives, which according to the stages of change theory suggests that they enter the program as pre-contemplators, whereas women enter at the preparation stage.[45] The supposition that the married men who attended the CHIP intervention with their spouse benefited from the ensuing household changes made by the women is supported by the literature.[46] Indeed, an intervention delivered in a group setting offers the social support needed to foster new social norms and accountability. Group programs have been shown to be more effective for achieving weight loss than individual programs, even for those who claim to prefer individual programs.[47]

Other factors relating to program content and structure may have contributed to the outcomes observed in this study. One may be the intensiveness of the CHIP intervention. Other studies have shown a dose response between intervention intensity and health outcomes.[3,48] Further research is required to determine the most efficacious dosages of lifestyle interventions with regard to the number of sessions, program duration, and type and magnitude of lifestyle modifications targeted for men and women. Other factors may be the strong educative component and repeated health risk assessments. These are believed to increase participants' health related self-efficacy and perceived control. Future research should explore self-efficacy/perceived behavioral control among men and women participating in CHIP resulting from the increased knowledge that comes from the educational videos.

Study Limitations and Strengths

In this study, a greater proportion of men entered the program with previously diagnosed health conditions, which may have contributed to the higher baseline risk factor levels of men compared with women.

However, < 5% of each gender entered the program with cardiovascular disease conditions, and so it is unlikely that baseline health history would have had a major impact on the outcomes of the intervention. Another limitation of this study is missing data. However, it is not ex-pected that the missing data would have attenuated the outcomes reported in this study, because the proportion was similar (≤ 7%) for both men and women.

The ability of self-selected participants to pay the program fee could have led to selection bias of persons with economic advantage over those who could not afford the fee, or of persons interested in health compared with those not interested in making changes and therefore not willing to pay. Because the interventions are sourced primarily through the Seventh-Day Adventist church, this may have biased the selection process and therefore the outcomes. However, because only one-fifth of participants reported being Seventh-Day Adventists, it is not likely that motivation to participate could be attributed to membership of this health-conscious religious denomination. The generalizability of the findings to less motivated populations and with differing baseline health characteristics, eg, biometrics and smoking status needs to be determined.

Information on fat distribution, particularly android fat; compliance measures, particularly for dietary intake and physical activity; socioeconomic factors such as social class and ethnicity, and who the participant attended the program with; and readiness to change are important factors that may have contributed to the gender difference and are lacking in this study. Lack of information to align risk factors with behaviors is a limitation of this study. Future studies will gather valid measures of psychosocial factors and the various lifestyle changes made by participants during the CHIP program to elucidate their contribution to the results achieved.

Despite these limitations, the results of this study, which show the value of CHIP to lower risk factors, are noteworthy given the size of the sample and the large reductions observed, particularly among individuals commencing the program with the greatest risk. Risk factor characterization of all biometrics was reduced to at least the next lower level for both men and women, but more so for men, with some participants reducing 2 or 3 levels, particularly if they were in the higher risk categories at baseline. Therefore, reporting the mean change at each risk level actually underestimated the actual change in this study as participants reduced their risk characterization.

Notwithstanding the greater reductions among men, women achieved substantial risk reductions for most biometrics and the intervention appears to be beneficial for both genders. More than 60% of men and women with highest baseline risk levels for SBP, DBP, TC, and TG reduced their risk characterization after the 30-day intervention. In addition, almost half of men and women characterized with diabetes and high baseline levels of LDL reduced these characterizations. These improvements translated to a 19.4% reduction in men and a 15.7% reduction in women who were characterized with metabolic syndrome at baseline. Furthermore, the changes in TC and LDL levels compare favorably with those achieved by pharmaceutical interventions involving statins[49] but without the risk, and are much greater than those expected from dietary interventions aimed at lowering blood lipids.[50]

The strength of the CHIP intervention is the strong education component that focuses on the etiology of chronic disease and the benefits of positive lifestyle choices, with particular attention given to diet, physical activity, sleep, and stress; overcoming barriers; maintaining behavior change; environmental influences on lifestyle practices; mental and emotional health; and self-worth and personal flourishing.

IMPLICATIONS FOR RESEARCH AND PRACTICE

The educationally centered CHIP lifestyle intervention is effective at reducing selected chronic disease factors in 30 days for both men and women, but especially for men. However, it appears that male and female participants react differently to these interventions. Although this study did not examine the physiological and behavioral reasons for this difference, the literature suggests that lifestyle interventions targeting women should be delivered in a socially interactive group setting. For men, the literature suggests that interventions

should involve a prescriptive regime. Both men and women appear to benefit from programs that focus on an eating regime that is high in minimally processed, whole, plant foods, because this is nutrient dense and satiating, yet low in calories. This eating regime not only benefits weight loss but improves other chronic disease risk factors. In addition, lifestyle interventions should incorporate physical activity, including strength training routines to build lean muscle mass and increase metabolic rate in programs that include women. Interventions delivered through an entertaining, evidence-based educative video in an interactive setting, with regular health risk assessments, appear to be efficacious in fostering behavior change by developing perceived control and self-efficacy.

As lifestyle interventions such as CHIP continue to expand globally, more research needs to be undertaken to establish how best to meet the different needs of male and female participants.

REFERENCES

1. Centers for Disease Control and Prevention. Chronic diseases and health promotion: United States, 2012. http://www.cdc.gov/chronicdisease/overview/index.htm. Accessed October 1, 2013.

2. Heidenreich PA, Trogdon JG, Khavjou OA, et al. Forecasting the future of cardiovascular disease in the United States: a policy statement from the American Heart Association. *Circulation*. 2011;123:933-944.

3. Ornish D, Scherwitz LW, Billings JH, et al. Intensive lifestyle changes for reversal of coronary heart disease. *JAMA*. 1998;280:2001-2007.

4. Aldana SG, Greenlaw RL, Diehl HA, et al. A video-based lifestyle intervention and changes in coronary risk. *Health Educ Res*. 2008;23:115-124.

5. Diehl HA. Coronary risk reduction through intensive community-based lifestyle intervention: the CHIP experience. *Am J Cardiol*. 1998;82:83T-87T.

6. Englert HS, Diehl HA, Greenlaw RL, et al. The effect of a community-based coronary risk reduction: the Rockford CHIP. *Prevent Med*. 2007;44:513-519.

7. Morton DP, Rankin P, Morey P, et al. The effectiveness of the Complete Health Improvement Program (CHIP) in Australasia for reducing selected chronic disease risk factors: a feasibility study. *N Z Med J*. 2013;126:43-54.

8. Morton D, Rankin P, Kent L, Dysinger W. The Complete Health Improvement Program (CHIP): history, evaluation, and outcomes. *Am J Lifestyle Med*; 2014. http://dx.doi.org/10.1177/1559827614531391.

9. Kent L, Morton D, Hurlow T, et al. Long-term effectiveness of the community-based complete Health Improvement Program (CHIP) lifestyle intervention: a cohort study. *BMJ Open*. 2013;3:e003751.

10. Merrill RM, Aldana SG, Greenlaw RL, et al. Can newly acquired healthy behaviors persist? An analysis of health behavior decay. *Prev Chronic Dis*. 2008;5(1).

11. Denton M, Prus S, Walters V. Gender differences in health: a Canadian study of the psycho-social, structural and behavioural determinants of health. *Soc Sci Med*. 2004;58:2586-2600.

12. Parikh NI. Sex differences in the risk of cardiovascular disease. *BMJ*. 2011;343: d5526.

13. Barrett-Connor E. Sex differences in coronary heart disease: why are women so superior? The 1995 Ancel Keys Lecture. *Circulation*. 1997;95:252-264.

14. Kent L, Worsley A. Trends in BMI, diet and lifestyle between 1976 and 2005 in North Sydney. *Asia Pac J Clin Res*. 2009;18:453-461.

15. Kent L, Worsley A. Breakfast size is related to body mass index for men, but not women. *Nutr Res*. 2010;30:240-245.

16. ABC News. Which diet gives the best bang for your buck? July 23, 2014. http://abcnews.go.com/Health/diet-best-bang-buck/story?id=24677053. Accessed July 26, 2014.

17. Weight-control Information Network. Overweight and obesity statistics. 2014. http://win.niddk.nih.gov/statistics/. Accessed July 26, 2014.

18. Centers for Disease Control and Prevention. Cholesterol. http://www.cdc.gov/cholesterol/facts.htm. Accessed July 26, 2014.

19. Kuzma JW, Bohnenblust SE. *Basic Statistics for the Health Sciences*. 4th ed. Singapore: McGraw-Hill; 2001.

20. National Heart, Lung, and Blood Institute. Calculate body mass index. http://www.nhlbi.nih.gov/health/public/heart/obesity/wecan/healthy-weight-basics/body-mass-index.htm. Accessed May 13, 2014.

21. Joint National Committee. The fifth report of the Joint National Committee on detection, evaluation, and treatment of high blood pressure (JNC V). *Arch Intern Med*. 1993;153:154-183.

22. National Cholesterol Education Program (NCEP) Expert Panel on Detection. Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). 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. *Circulation*. 2002;106:3143-3421.

23. Wilson PWF, D'Agostino RB, Levy D, et al. Prediction of coronary heart disease using risk factor categories. *Circulation*. 1998;97:1837-1847.

24. Alberti KG, Eckel RH, Grundy SM, et al. Harmonizing the metabolic syndrome: a joint interim statement of the International Diabetes Federation Task Force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute, American Heart Association; World Heart Federation; International Atherosclerosis Society and International Association for the Study of Obesity. *Circulation*. 2009;120: 1640-1645.

25. Barnard RJ. Effects of life-style modification on serum lipids. *Arch Intern Med*. 1991;151:1389-1394.

26. Kent L, Morton D, Rankin P, et al. The effect of a low-fat, plant-based lifestyle intervention (CHIP) on serum HDL levels and the implications for metabolic syndrome status—a cohort study. *Nutr Metab*. 2013;10:58-63.

27. Chaston TB, Dixon JB. Factors associated with percent change in visceral versus subcutaneous abdominal fat during weight loss: findings from a systematic review. *Int J Obes*. 2008;32:619-628.

28. Regitz-Zagrosek V, Lehmkuhl E, Weickert MO. Gender differences in the metabolic syndrome and their role for cardiovascular disease. *Clin Res Cardiol*. 2006;95:136-147.

29. Cameron AJ, Dunstan DW, Owen N, et al. Health and mortality consequences of abdominal obesity: evidence from the AusDiab study. *Med J Aust*. 2009;191:202-208.

30. Wardle J, Johnson F. Weight and dieting: examining levels of weight concern in British adults. *Int J Obesity*. 2002;26: 1144-1149.

31. Egger G. The Australian experience: from GutBusters to Professor Trim's.

In: White A, Pettifer M, eds. *Hazardous Waist: Tackling Overweight and Obesity in Men*. Oxford, UK: Radcliffe; 2007:205-212.

32. Stiegler P, Cunliffe A. The role of diet and exercise for the maintenance of fat-free mass and resting metabolic rate during weight loss. *Sports Med*. 2006;36:239-262.

33. Janssen J, Heymsfield SB, Wang Z, et al. Skeletal muscle mass and distribution in 468 men and women aged 18–88 yr. *J Appl Physiol*. 2000;89:81-88.

34. Friel S, Kelleher CC, Nolan G, et al. Social diversity of Irish adults nutritional intake. *Eur J Clin Nutr*. 2003;57: 865-875.

35. Marsh K, Zeuschner C, Saunders A. Health implications of a vegetarian diet: a review. *Am J Lifestyle Med*. 2012;6:250-267.

36. Blanck HM, Gillespie C, Kimmons JE, et al. Trends in fruit and vegetable consumption among U.S. men and women, 1994–2005. *Prev Chronic Dis*. 2008;5:A35.

37. Lassek WD, Gaulin SJC. Costs and benefits of fat-free muscle mass in men: relationship to mating success, dietary requirements, and native immunity. *Evol Hum Behav*. 2009;30:322-328.

38. Wilkins D, Payne S, Granville G, et al. The Gender and Access to Health Services Study. *United Kingdom*. 2008. http://www.academia.edu/1254973/The_Gender_and_Access_to_Health_Services_Study_Final_Report. Accessed May 1, 2014.

39. Vlerick Leuven Gent Working Paper Series. Gender differences in commitment to change: impacted by gender or by being part of a minority group? *Finland*. 2012. https://public.vlerick.com/Publications/057e56cf-11ab-e111-8afa-005056a635ed.pdf. Accessed May 5, 2014.

40. Young MD, Morgan PJ, Plotnikoff RC, et al. Effectiveness of male-only weight loss and weight loss maintenance interventions: a systematic review with meta-analysis. *Obes Rev*. 2012;13: 393-408.

41. Bautista-Castano I, Molina-Cabrillana J, Montoya-Alonso JA, et al. Variables predictive of adherence to diet and physical activity recommendations in the treatment of obesity and overweight in a group of Spanish subjects. *Int J Obes*. 2004;28:697-705.

42. Linne Y, Hemmingsso E, Adolfsson B, et al. Patient expectations of obesity treatment: the experience from a day-care unit. *Int J Obes*. 2002;26:739-741.

43. Donoho C, Crimmins EM, Seeman TE. Marital quality, gender, and markers of inflammation in the MIDUS cohort. *J Marriage Fam*. 2013; 75:127-141.

44. Seymour-Smith S, Wetherell M, Phoenix A. "My wife ordered me to come!": a discursive analysis of doctors' and nurses' accounts of men's use of general practitioners. *J Health Psychol*. 2002;7: 253-267.

45. Norcross JC, Krebs PM, Prochaska JO. Stages of change. *J Clin Psychol*. 2011; 67:143-154.

46. Gorin AA, Raynor HA, Fava J, et al. Randomized controlled trial of a comprehensive home environment-focused weight loss program for adults. *Health Psychol*. 2013;32:128-137.

47. Renjilian DA, Perri MG, Nezu AM, et al. Individual versus group therapy for obesity: effects of matching participants to their treatment preferences. *J Consult Clin Psychol*. 2001;69:717-721.

48. Barnard RJ, Guzy P, Rosenberg J, et al. Effects of an intensive exercise and nutrition program on patients with coronary artery disease: five-year follow-up. *J Cardiac Rehabil*. 1983;3:183-190.

49. Gould AL, Davies GM, Alemao E, et al. Cholesterol reduction yields clinical benefits: meta-analysis including recent trials. *Clin Ther*. 2007;29:778-794.

50. Tang JL, Armitage JM, Lancaster T, et al. Systematic review of dietary intervention trials to lower blood total cholesterol in free-living subjects. *BMJ*. 1998;316:1213-1220.