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Gender differences in effectiveness of the Complete Health Improvement Program (CHIP) lifestyle intervention: an Australasian study

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Abstract

Issue addressed: Complete Health Improvement Program (CHIP) is a lifestyle modification program that promotes healthy diet, physical activity and stress management techniques. Among US CHIP participants, differences in gender responsiveness to improvements in chronic disease risk factors were demonstrated. This study examined gender differences in outcomes to the CHIP intervention in Australasia.

Methods: Changes in body weight, blood pressure (BP), blood lipid profile and fasting plasma glucose (FPG) were assessed in 925 participants (34.3% men, mean age = 56.0 ± 12.5 years; 65.7% women, mean age = 54.4 ± 13.5 years) 30 days after program commencement.

Results: Significant reductions ($P < 0.001$) in all biometrics measured were found for men and women but were greater among men for total (TC) and low-density lipoprotein cholesterol (LDL), triglycerides (TG), FPG, body mass index (BMI) and TC/high-density lipoprotein cholesterol (HDL) ratio. Participants with highest baseline classifications of BMI, systolic BP, blood lipids and FPG showed greatest reductions in 30 days.

Conclusions: CHIP more effectively reduced chronic disease risk factors among men than women. All participants, but particularly men, entering the program with the greatest risk achieved the largest reductions. Possible physiological or behavioural factors include food preferences, making commitments and differential support modes.

So what? Developers of lifestyle intervention programs should consider gender differences in physiological and behavioural factors when planning interventions. In particular, developers should manage expectations of people entering lifestyle interventions to increase awareness that men tend to respond better than women. In addition, this is a call for further research to identify the underlying mechanisms responsible for the disproportionate responsiveness of males.

Key words: Australia, chronic disease, gender, New Zealand, risk factors.

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Introduction

Chronic diseases are the leading causes of death and disability in Australasia, as they are worldwide.[1–3] Deaths from chronic diseases are projected to increase by 15% by 2020.[3] Chronic diseases place a large burden on families and communities through increased morbidity, with a subsequent major fiscal burden.[1,2]

Lifestyle modification programs have been shown to be effective in the treatment of chronic disease.[4] The Complete Health Improvement Program (CHIP), a community-based lifestyle modification program, developed in the US in 1986, has demonstrated significant reductions in selected chronic disease risk factors among large cohorts from several countries, including Australia and New Zealand.[5–8]

Gender differences in the prevalence of various chronic diseases (e.g. cardiovascular disease and obesity) and in lifestyle behaviours (e.g. dietary intake and physical activity associated with these diseases) are well recognised.[9–13] It is therefore important to differentiate the gender responsiveness to lifestyle interventions

and find effective ways to address them. While CHIP programs in Australasia have shown significant reductions in several chronic disease risk factors, the differential responsiveness of men and women to the CHIP intervention has not been performed.[5,6] The present study examined the short-term responsiveness of men and women to the CHIP intervention in Australia and New Zealand and suggests possible hormonal, behavioural (e.g. food preferences), physiological (e.g. adiposity and lean tissue mass), social (e.g. group support) and/or psychological (e.g. commitment) factors that should be considered by those responsible for the planning and delivery of lifestyle interventions.

Methods

The CHIP intervention was delivered to 925 participants, who had self-selected to participate in the program between May 2006 and September 2012. There were no inclusion/exclusion criteria other than the participant being able to pay the $300 program cost. A total of 44 CHIP programs (mean group size 21, range 5–100) were conducted at 26 locations throughout Australasia (138 participants in 9 locations in Australia; 787 in 17 locations in New Zealand) over this period. The Avondale College of Higher Education Ethics Committee approved the study.

The Australasian CHIP interventions were advertised in the local media (newspapers, radio) of the communities in which the programs were being offered and in some instances local medical practitioners recommended their patients to the program. The intervention involved 16 x two-hour group sessions, delivered four days a week, over 30 days. Each session involved viewing a one-hour pre-recorded lecture, a cooking demonstration and group discussion. Furthermore, where local health experts could be appropriately sourced, shopping tours and guided exercise sessions were also added to the sessions. Participants were educated on the aetiology of chronic disease and the benefits of positive lifestyle choices, with particular attention given to encouraging and supporting the consumption of a low-fat (<15% of calories from fat), plant-based ad libitum diet. In addition, the program advocated that participants engage daily in 30 min of moderate physical activity (walking) and practise stress management techniques (life balance, sleep, rest). Behaviour-change programs that include these components have been shown to be effective at reducing chronic disease by Ornish *et al.*[4] In addition, 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 behaviour-change maintenance were also included. For a discussion on the development and detailed description of the CHIP program, the reader is referred to the review article by Morton *et al.*[14]

The CHIP programs were conducted by volunteer facilitators, who had an interest in positively influencing the health of their local community. All volunteers were required to undergo two 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 program intervention was presented through the pre-recorded videos. The role of the volunteer facilitator was to organise the meeting and to facilitate discussions in the 16 group sessions. While the use of the supplied resources meant that program delivery was consistent in each location, the program may have varied with the addition of activities (e.g. shopping tours and exercise sessions) depending on the volunteers running these additional segments.

The participant was deemed to have completed the initial 30 days of the program if they attended 13 of the 16 sessions (more than 80% of the program) and underwent both pre- and post-assessments. Before participating in the CHIP intervention (baseline) and again at its conclusion (post-intervention), the participants' height, weight, SBP and DBP were taken and fasting (12-h) 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. The blood samples were collected by trained phlebotomists and analysed by local pathology laboratories for TC, LDL, HDL, TG and FPG levels.

Statistical analyses

The data were analysed using IBM™ Statistics (version 19) and expressed as mean ± s.d. Pearson's chi-square was used to determine the extent of differences between men and women for demographic variables. A one-way multivariate analysis of variance (MANOVA) was conducted to test the hypothesis that there would be one or more mean differences between men and women for the biometric measures. The extent of the changes (from baseline to post-intervention) in the biometric measures was then assessed for males and females separately, using paired *t*-tests. To examine whether these gender differences were statistically significant, a new variable (change from pre- to post-) was created and independent *t*-tests were performed. The McNemar's chi-square test was used to determine the extent of changes in the distribution of participants by gender, across the various risk factor categories. Participants' weight were characterised in risk categories using standard BMI cut-points for 'normal', 'overweight' and 'obese';[15] BP was classified using the 5th Joint National Committee for Hypertension guidelines;[16] and FPG was characterised according to conventional 'normal', 'impaired' and 'diabetic' levels.[17] The US National Cholesterol Education Program Adult Treatment Panel III (ATP III) classification system[16] was used to categorise the participants for all risk factors, except total cholesterol, for which the Framingham risk classification[18] was used as it includes five cholesterol categories compared with only three in the ATP III classification system. Metabolic syndrome at baseline and after intervention was classified according to the 'harmonised

definition'.[19] Participants were deemed as having this syndrome if they met three or more of the defining criteria.[19] Statistical tests were two-sided with a significance level of 5% ($P < 0.05$). Confidence intervals (95%) are also presented. In order to reduce the Type 1 error that can occur when simultaneous tests are performed in a dataset, a Bonferroni correction was applied separately to each biometric. As there were 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. Participants that did not have baseline biometrics were removed. The remaining participants were examined for missing data by sorting each variable in ascending order. Data was considered to be missing 'completely at random' when there did not appear to be a relationship between a missing data point and other values in the dataset (e.g. for a missing lipid value, values for other lipids were present, and there was no consistency in which variables had missing values).

Results

Of the 925 participants who enrolled in the program, 891 (96%) completed the initial 30 days of the intervention. Of these 891 participants, 34.3% ($n = 301$) were men and 65.7% ($n = 590$) were women. There were no significant difference in age between men and women (56.0 ± 12.5 years vs 54.4 ± 13.5 years, t(890) = 1.76, $P = 0.079$) and in the proportion reporting smoking ((4.4% vs 5.1%, ($X^2(3) = 2.23$, $P = 0.527$). More men reported being married than women (84% vs 73%, ($X^2(3) = 18.36$, $P < 0.001$). There were no differences in the proportion of men and women who attended the program each year between 2006 and 2012 ($X^2(6) = 5.30$, $P = 0.506$), in program location ($X^2(26) = 19.97$, $P = 0.793$), or across age groups (<30 years, 30–49 years, 50–69 years, 70+ years; $X^2(3) = 5.74$, $P = 0.125$).

As shown in Table 1, at program entry both men and women were representative of an at-risk population with a mean BMI in the 'obese' category and elevated BP and LDL. On average, the women had elevated TC and the men had 'prediabetic' FPG levels at program entry. There were also some baseline differences in health history, with more men than women commencing the intervention with diagnosed conditions, such as, coronary bypass (2.8% vs 0.9%, $X^2(1) = 4.29$, $P = 0.038$) and stroke (2.4% vs 0.7%, $X^2(1) = 4.21$, $P = 0.040$). Men had higher baseline weight, SBP (t(868) = 3.13, $P < 0.002$) and TC:HDL ratio, while the women had higher baseline TC (t(858) = −5.56, $P < 0.001$), LDL (t(853) = −2.91, $P = 0.004$) and HDL (t(858) = −10.52, $P < 0.001$) (Table 1). However, when the data was split by risk category, women had higher baseline TC but only in the range 4.14–5.17 mmol/L, LDL in the range 2.59–3.35 mmol/L and HDL in the intermediate range (see Table 2).

Multivariate analysis of the biometric changes showed that the covariance between men and women were assumed to be equal (Box's M of 588.91, $P < 0.001$). A statistically significant MANOVA

Table 1. Mean changes in selected risk factors for men and women from baseline to 30 days, Australia and New Zealand, 2006–2012

**pre-post- mean change significant at $P < 0.001$; [1]t-test; [††] difference in change between men and women significant at $P < 0.001$ level of significance; [†]difference in change between men and women significant at $P < 0.05$ level of significance; [‡‡]baseline difference significant at $P < 0.001$ level of significance; [‡]baseline difference significant at $P < 0.05$ level of significance; [2]% change = ((mean at 30-days − mean at baseline)/mean at baseline)*100

Risk factor	Men								Women							
	N	Baseline		30-days		Mean Change[1]	95% CI	% change[2]	N	Baseline		30-days		Mean change[1]	95% CI	% change[2]
		Mean	s.d.	Mean	s.d.					Mean	s.d.	Mean	s.d.			
Weight (kg)	298	93.9	23.8	90.0	22.4	−3.9**	−4.3, −3.6	−4.2%	585	82.83[‡‡]	20.63	80.14	19.92	−2.7**	−2.9, −2.5	−3.2%[††]
Body mass index (kg/m^2)	297	30.6	7.1	29.3	6.8	−1.3**	−1.4, −1.2	−4.2%	585	31.38	7.65	30.36	7.39	−1.0**	−1.1, −1.0	−3.3%[††]
Systolic blood pressure (mm Hg)	294	136.4	19.9	128.2	17.7	−8.2**	−10.0, −6.3	−6.0%	576	131.9[‡]	20.3	124.6	17.1	−7.3**	−8.5, −6.1	−5.5%
Diastolic blood pressure (mm Hg)	294	84.7	17.2	80.1	15.8	−4.6**	−5.7, −3.5	−5.4%	578	83.7	21.7	78.7	18.7	−4.9**	−5.8, −4.1	−5.9%
Total cholesterol (mmol/L)	295	4.91	1.09	4.01	0.89	−0.90**	−0.98, −0.82	−18.3%	565	5.31[‡‡]	1.11	4.59	0.95	−0.73**	−0.79, −0.66	−13.7%[†]
Low-density cholesterol (mmol/L)	294	3.04	0.94	2.35	0.77	−0.68**	−0.75, −0.62	−22.4%	561	3.24[‡]	0.96	2.69	0.83	−0.54**	−0.60, −0.49	−16.7%[†]
High-density cholesterol (mmol/L)	295	1.15	0.31	1.06	0.27	−0.09**	−0.11, −0.07	7.8%	565	1.41[‡‡]	0.35	1.28	0.32	−0.13**	−0.14, −0.11	9.2%[†]
Total cholesterol: HDL ratio	295	4.48	1.32	3.97	1.14	−0.51**	−0.61, −0.41	−11.4%	565	3.99[‡‡]	1.23	3.78	1.15	−0.21**	−0.26, −0.16	−5.3%[††]
Triglycerides (mmol/L)	295	1.59	1.04	1.30	0.73	−0.30**	−0.40, −0.20	−18.9%	564	1.48	0.94	1.34	0.70	−0.14**	−0.19, −0.08	−9.5%[†]
Fasting plasma glucose (mmol/L)	292	5.56	1.49	5.14	0.99	−0.42**	−0.56, −0.27	−7.6%	562	5.48	1.54	5.22	1.24	−0.26**	−0.34, −0.17	−4.7%[†]

Table 2. Changes in risk factor levels within 30 days by gender and initial risk factor classification, Australia and New Zealand, 2006–2012

**pre-post- mean change significant at $P < 0.001$; *Bonferroni correction applied: pre-post- mean change at $P < 0.0167$ level of significance with three risk categories, $P < 0.0125$ with four risk categories, $P < 0.01$ with five risk categories; [1]X^2=McNemar Chi Squared statistic; [2]*t*-test; [††]difference in change between men and women significant at $P < 0.001$ level of significance; [†]Bonferroni correction applied: difference in change between men and women at $P < 0.0167$ level of significance with three risk categories, $P < 0.0125$ with four risk categories, $P < 0.01$ with five risk categories; [‡‡]baseline difference significant at $P < 0.001$ level of significance, [‡]Bonferroni correction applied: baseline difference at $P < 0.0167$ level of significance with three risk categories, $P < 0.0125$ with four risk categories, $P < 0.01$ with five risk categories; [3]% change = ((mean at 30-days – mean at baseline)/mean at baseline)*100

Risk factor	Men											Women									
	Baseline[1]		30-days[1]		Baseline		30-days		Mean	95% CI	%	Baseline[1]		30-days[1]		Baseline		30-days		Mean	95% CI	%
	N	%	N	%	Mean	s.d.	Mean	s.d.	change[2]		change[3]	N	%	N	%	Mean	s.d.	Mean	s.d.	change[2]		change[3]
Body Mass Index (kg/m^2)	X^2 undefined											X^2 = 65.1**										
<18.5	0	0	1	0.3	19.1	0	18.4	0	N/A			1	0.2	1	0.2	17.7	0	17.6	0	−0.1	−0.1	−1.0
18.5–24.9	60	20.1	72	24.2	22.9	1.5	22.3	1.5	−0.6**	−0.8, −0.5	−2.6	120	20.5	154	26.4	22.7	1.7	22.1	1.7	−0.5**	−0.6, −0.4	−2.2
25–29.9	105	35.2	119	39.9	27.6	1.5	26.5	1.3	−1.1**	−1.2, −1.0	−4.0	163	27.9	162	27.7	27.3	1.4	26.4	1.5	−1.0**	−1.1, −0.9	−3.7
≥30	133	34.6	106	35.6	36.3	6.8	34.6	6.6	−1.7**	−1.9, −1.5	−4.7	300	51.4	267	45.7	37.1	6.2	35.9	6.1	−1.3**	−1.3, −1.2	−3.5[††]
Systolic blood pressure (mmHg)	X^2 = 50.7**											X^2 = 92.3**										
<120	53	18.0	80	27.2	110.4	8.8	112.7	11.9	2.2	−0.2, 4.7	2.0	148	25.7	225	39.1	108.9	7.9	108.8	12.1	−0.1	−2.0, 1.8	−0.1
120–139	125	42.5	152	51.7	129.3	5.3	123.9	12.2	−5.4**	−7.7, −3.2	−4.2	245	42.5	237	41.1	128.5	5.8	123.6	11.3	−5.0**	−6.4, −3.6	−3.8
140–160	89	30.3	50	17.0	149.7	6.7	138.1	17.6	−11.6**	−15.1, −8.0	−7.7	138	24.0	96	16.7	148.0	6.8	135.6	13.3	−12.3**	−14.5, −10.1	−8.3
>160	27	9.2	12	4.1	176.4	11.6	146.5	15.3	−30.0**	−35.9, −24.1	−17.0	45	7.8	18	3.1	176.5	12.3	147.8	16.9	−28.7**	−34.2, −23.2	−16.3
Diastolic blood pressure (mmHg)	X^2 = 45.6**											X^2 = 67.8**										
<80	101	34.3	143	48.6	70.1	6.2	70.2	8.7	0.1	−1.5, 1.7	0.4	264	45.7	335	58.0	69.5	6.9	69.2	9.1	−0.3	−1.3, 0.6	−0.4
80–89	108	36.7	104	35.4	83.3	3.0	78.9	7.8	−4.4**	−6.0, −2.9	−5.3	169	29.2	154	26.6	83.1	2.9	77.6	8.8	−5.5**	−6.9, −4.2	−6.6
90–100	58	19.7	29	9.9	94.0	3.8	84.8	8.4	−9.1**	−11.4, −6.9	−9.7	83	14.4	40	6.9	93.3	3.6	82.1	10.5	−11.3**	−13.6, −9.0	−12.1
>100	27	9.2	18	6.1	124.7	21.1	112.0	25.0	−12.7**	−18.2, −7.3	−10.2	62	10.7	49	8.5	132.8	27.8	118.3	24.5	−14.5**	−18.2, −10.8	−10.9
Total Cholesterol (mmol/l)	X^2 undefined											X^2 = 276.8**										
<4.14	76	25.8	177	60.0	3.61	0.45	3.15	0.61	−0.46**	−0.58, −0.35	−13.9	75	13.3	190	33.6	3.67	0.41	3.52	0.61	−0.16	−0.28, −0.03	−5.9[††]
4.14–5.17	99	33.6	90	30.5	4.61	0.28	3.90	0.54	−0.70**	−0.80, −0.61	−15.2	186	32.9	235	41.6	4.68[‡]	0.26	4.13	0.56	−0.55**	−0.63, −0.47	−10.6
5.18–6.19	82	27.8	26	8.8	5.65	0.28	4.42	0.65	−1.23**	−1.37, −1.09	−21.4	186	32.9	107	18.9	5.61	0.28	4.86	0.61	−0.75**	−0.83, −0.66	−12.5[††]
6.20–7.24	33	11.2	2	0.7	6.55	0.29	5.03	0.71	−1.52**	−1.77, −1.27	−22.7	92	16.3	28	5.0	6.57	0.30	5.35	0.79	−1.22**	−1.38, −1.06	−18.1
>7.24	5	1.7	0	0	7.94	0.58	5.76	0.73	−2.18	−3.68, −0.68	−27.8	26	4.6	5	0.9	8.01	0.91	6.26	1.08	−1.75**	−2.29, −1.22	−22.5
Low Density Lipoprotein (mmol/l)	X^2 undefined											X^2 = 200.5**										
<2.59	96	32.7	176	59.9	2.03	0.45	1.68	0.51	−0.36**	−0.44, −0.27	−20.0	139	24.8	256	45.6	2.06	0.38	1.88	0.51	−0.19**	−0.27, −0.11	−9.5†
2.59–3.35	91	31.0	87	29.6	2.91	0.24	2.31	0.47	−0.60**	−0.70, −0.50	−20.7	185	33.0	189	33.7	2.98[‡]	0.23	2.56	0.50	−0.42**	−0.49, −0.35	−13.3†
3.36–4.13	68	23.1	27	9.2	3.74	0.22	2.81	0.57	−0.93**	−1.06, −0.80	−24.3	139	24.8	86	15.3	3.71	0.24	3.02	0.59	−0.70**	−0.79, −0.60	−18.9†
4.14–4.90	31	10.5	4	1.4	4.41	0.23	3.30	0.63	−1.11**	−1.32, −0.91	−25.0	76	13.5	23	4.1	4.49	0.23	3.51	0.63	−0.98**	−1.12, −0.83	−22.2
≥4.91	8	2.7	0	0	5.23		3.40	0.63	−1.83**	−2.34, −1.31	−34.6	22	3.9	7	1.2	5.50	0.66	4.10	0.94	−1.39**	−1.87, −0.92	−25.5
High Density Lipoprotein (mmol/l)	X^2 = 34.6**											X^2 = 100.0**										
<1.03 men, <1.30 women	118	40.0	162	54.9	0.88	0.11	0.85	0.13	−0.02	−0.05, −0.00	−2.3	222	39.3	302	53.5	1.07	0.15	1.02	0.17	−0.05**	−0.07, 0.03	−4.7
1.03 men/1.30 women–1.54	152	51.5	119	40.3	1.25	0.14	1.14	0.19	−0.11**	−0.13, −0.09	−8.8	162	28.7	157	27.8	1.41[‡‡]	0.08	1.29	0.20	−0.12**	−0.15, −0.09	−8.5
≥1.55	25	8.5	14	4.7	1.82	0.29	1.49	0.34	−0.33**	−0.47, −0.18	−18.1	181	32.0	106	18.8	1.82	0.22	1.59	0.26	−0.23**	−0.26, −0.19	−12.6

Triglycerides (mmol/l)			X^2 undefined										X^2 undefined										
<1.69	194	65.8	235	79.7	1.10	0.32	1.00	0.39	−0.10**	−0.15, −0.50	−9.1	405	71.8	418	74.1	1.04	0.34	1.07	0.40	0.02	−0.01, 0.06	1.9††
1.69–2.25	54	18.3	32	10.8	1.92	0.17	1.54	0.55	−0.38**	−0.54, −0.21	−19.8	78	13.8	91	16.1	1.93	0.16	1.66	0.59	−0.27**	−0.41, −0.13	−14.0
2.26–5.64	46	15.6	28	9.5	3.03	0.65	2.26	1.05	−0.78**	−1.06, −0.49	−25.7	78	13.8	55	9.8	3.02	0.78	2.41	0.82	−0.61**	−0.81, −0.41	−20.2
≥5.65	1	0.3	0	0	13.0	0	1.50	0	N/A			3	0.5	0	0	7.90	1.65	2.02	1.00	−5.88	−12.21, 0.44	−74.4
Fasting Plasma Glucose (mmol/l)			X^2 = 45.8**										X^2 = 40.1**										
<5.50	175	59.9	229	78.4	4.83	0.48	4.78	0.65	−0.05	−0.14, 0.05	−1.0	377	67.1	428	76.2	4.82	0.37	4.79	0.49	−0.03	−0.08, 0.01	−0.6
5.50–6.90	90	30.8	49	16.8	5.87	0.35	5.35	0.54	−0.52**	−0.63, −0.41	−8.9	138	24.6	105	18.7	5.85	0.36	5.46	0.51	−0.39**	−0.47, −0.31	−6.7
>6.90	27	9.2	14	4.8	9.22	2.26	6.76	1.86	−2.46**	−3.67, −1.25	−26.7	47	8.4	29	5.2	9.63	2.44	7.97	2.55	−1.66**	−2.48, −0.83	−17.2

effect was obtained, Pillai's Trace = 0.29, F(11, 803) = 30.45, $P < 0.001$. *t*-tests were then performed to examine mean differences between men and women. The reductions were greater among the men than women for BMI, TC, LDL, TG, FPG and TC:HDL ratio, but women had greater reductions than the men for HDL (see Table 1). Furthermore, more men than women significantly decreased their classification of Metabolic Syndrome at 30 days (males: 43.2% to 32.5%, ($X^2(1) = 91.88$, $P < 0.001$; females 38.3% to 34.8%, ($X^2(1) = 259.10$ $P < 0.001$).

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 (see Table 2). Furthermore, while statistical analysis could not be performed for the proportional reduction of men compared with women in each risk category examined separately, it would appear that more men than women presenting with the highest category for each of DBP (>100 mmHg), BMI (>30 kg/m^2), TC (>7.24 mmol/l) and LDL (≥4.91 mmol/l) reduced their risk characterisation at 30 days (33% vs 21%, 20% vs 11%, 100% vs 81%, 100% vs 68%, respectively) (see Table 2). Furthermore, for FPG levels indicative of diabetes (>6.99 mmol/l), more men than women (48% vs 38%) appeared to have reduced their risk factor categorisation in the 30 days. Conversely, in the highest risk category for SBP (>160 mmHg) more women than men (60% vs 56%) appeared to no longer be in this risk category (see Table 2). An analysis of mean changes in the various biometric categories also showed that men achieved greater improvements than the women. For BMI, men experienced greater decreases than women in the highest risk category for BMI; the lowest risk categories for TC and LDL; and the lowest category for TG (see Table 2).

Discussion

Greater reductions in selected risk factors were achieved in 30 days among men using the CHIP lifestyle intervention than women. Furthermore, the majority of men in the highest risk classifications for TC, LDL, TG and FPG showed improvement of more than 25% in just 30 days; these improvements were greater than those of women, though not statistically significant, which could be due to small sample sizes in these groups. It would appear this disparity goes beyond higher baseline levels in men, as baseline levels of TC and LDL were higher for women than for men. Of note, the gender trends in baseline indices in this study are supported by national Australian data.[20] While the reasons for these differential gender outcomes were not explored in this study, several factors, including physiological and/or behavioural, could be speculated from the literature.

In terms of physiological factors, differences in the distribution of endogenous fat, may offer an explanation. Android fat around the abdomen, is more common in men, while gynoid fat around the buttocks, hips and thighs is more common in women.[21] Android fat

is more metabolically active and therefore easier to remove than gynoid fat.[21] Android fat also increases the risk of type 2 diabetes, metabolic syndrome, cardiovascular disease (CVD), dyslipidaemias and hypertension.[22] Another factor may be the greater muscle mass of men, which also contributes to their greater weight, compared with women.[22] Consequently, mechanically moving the greater weight of men means men are more likely to lose more weight than women.[23] In addition, greater muscle mass is associated with higher metabolic rate and therefore greater efficiency in expending energy.[24] Other physiological explanations may include the differing hormonal profile between men and women.[25]

Also related to physiological factors are differences in food preferences and the amount of food eaten by men and women. 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 CVD.[26] Plant foods are rich in fibre and a range of phytochemicals and antioxidants that are believed to confer these aforementioned health benefits.[26] As population surveys show that women tend to eat more fruit and vegetables than men, while men's diets tend to be higher in red and processed meats and lower in fibre,[27] there is greater scope for men's diets to include more plant foods. Furthermore, the benefit of the greater muscle mass of men also means they have a greater requirement for dietary energy than women, through greater intakes of food.[28] 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. However, this hypothesis could not be confirmed as there is no information on uptake of the program, especially relating to diet and physical activity.

From a behavioural standpoint, men may engage better with a lifestyle program once committed, although getting them to initially commit might be more challenging. Evidence from the workplace would suggest that men seem to approach making the commitment to change differently from women and are more inclined to commit to a program if the benefits outweigh the costs.[29] Results from weight-loss interventions have shown that if men value the outcome, they are more likely to achieve goals they set for themselves.[30] They are also more likely to complete a program that is prescriptive[31] particularly if advised by a health professional.[25] While women are inclined to commit to interventions for social reasons (trust, interaction and obligations to significant others), be more eager to initially change behaviour and have higher expectations of interventions than men, they are also more easily disappointed and tend to drop out before reaching their goals.[29,32] Therefore, while the literature suggests that men don't engage with behaviour-change programs (e.g. weight loss), there is anecdotal evidence that once they have assessed and evaluated the evidence and made a commitment they can effectively engage in a program[30] and achieve better results in a shorter time than women (pers. comm., Professor G Egger, Founder of Gut Busters, 13 September 2013). However, this could not be explored in this study as 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 another behavioural factor that may help explain the gender differences observed in this study. Married men are less likely to engage in unhealthy, high-risk behaviours.[33] The contention that 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.[34] Although information on marital status was collected in this study, information on attendance with a spouse was not. The effect of marital status needs to be examined further, together with other participant characteristics on the responsiveness to the CHIP lifestyle intervention.

Even though men tend to access and use health services less often than women,[35] or be aware and concerned that they are overweight,[36] this study would indicate that the CHIP program can have a positive effect on men's health and men can do especially well in 30 days compared with women. Results from the Pritikin program also showed that men achieved greater reductions in chronic disease risk factors from a lifestyle intervention than women.[37] Clearly several factors are at play, which could explain the gender differences observed in this and other studies. These include, but are not limited to, a range of physiological and behavioural factors, ranging from fat and muscle distribution, attitudes towards personal health and appearance, food consumption patterns, self-efficacy and commitment, and social support. Broader research into the gender differences that accompany lifestyle interventions may elucidate further factors.

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. Given these were small proportions (less than 5%), it is unlikely that baseline-health history would have had a major impact on the outcomes following the intervention. Another limitation of this study is missing data. However, it is not expected that missing data would have attenuated the outcomes reported in this study, as the proportion of men and women who did not return for follow-up assessment or did not have information collected on all risk factors at follow-up was small (less than 5%) but similar. Missing data was considered 'missing completely at random'.

A further limitation was the short follow-up time after which the greater benefits gained by the men may have been lost. A small

New Zealand study found that 106 CHIP participants who returned for follow-up assessment, on average four years after completion of the intervention, were able to maintain improvements in most of their biometrics.[38] Furthermore, 71% of participants reported they were still compliant to the CHIP principles after this time, but it was not clear how this differed between men and women in this study. As duration of the intervention effects and their costs-benefits, especially in comparison to non-intervention groups, is of key interest to health promoters, the CHIP intervention should be extended, for periods such as six months to five years.

Lack of information to align risk factors with various behaviours was a limitation of this study. Information on fat distribution, particularly android fat and compliance measures in relation to dietary intake and physical activity, would assist in explaining the reason for gender differences. Furthermore, socioeconomic factors, such as social class and ethnicity; who the participant attended the program with; and readiness to change are important factors that may also have contributed to the gender difference. Future studies should 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.

Notwithstanding the greater reductions among men, women did achieve substantial risk reductions for most biometrics. More than 50% of men and women with higher baseline risk levels for SBP, TC, LDL and TG reduced their risk characterisation after the 30-day intervention. The changes in TC and LDL levels compare favourably to those achieved by pharmaceutical interventions involving statins,[39] but without the risk, and are much greater than that expected from dietary interventions aimed at lowering blood lipids.[40] In addition, almost half of men and more than one-third of women characterised with diabetes reduced this characterisation. These improvements translated to a 24.6% reduction in men and a 9.3% reduction in women who were characterised with Metabolic Syndrome at baseline. Of note, risk factor characterisation 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 two or three levels, particularly if they were in the higher risk categories at baseline. As a result, the amount of change between men and women in the highest baseline levels for TC, LDL and FPG may not have reached significance because of the small numbers remaining in these risk categories at 30 days. However, without a control group, generalising on effectiveness outside the study sample is not possible.

Conclusions

The results of the present study indicate that men who participate in the CHIP lifestyle intervention appear to achieve better outcomes for reducing selected chronic disease factors in 30 days than women. Defining the gender-specific physiological and/or behavioural factors that contribute to responsiveness to lifestyle change will assist in development of more effective lifestyle interventions for both men and women. As lifestyle interventions, such as CHIP, continue to be developed more research needs to be undertaken to establish how best to meet the different needs of male and female participants.

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