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Health and Wellness Coaching for 5-Year Projected Cardiovascular Health

A Randomized Controlled Trial

Rita V. Krishnamurthi, BSc, MApplSc, PhD*, Alain C. Vandal, PhD, Suzanne Barker-Collo, MA, PhD, Susan Mahon, PhD, P. Alan Barber, MD, PhD, Bruce Arroll, MD, PhD, Elaine Rush, PhD, Hinemoa Elder, PhD, and Valery L. Feigin, MD, PhD, FAAN*

Correspondence
Dr. Feigin
valery.feigin@aut.ac.nz

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Abstract

Background and Objectives

Evidence of effective multifactorial lifestyle interventions for primary stroke prevention is lacking, despite the significant contribution of lifestyle to stroke burden. We aimed to determine the efficacy of health and wellness coaching (HWC) for primary stroke and cardiovascular disease (CVD) prevention in adults at a moderate-to-high CVD risk.

Methods

This was a parallel, 2-arm, open-label, single-blinded, phase III randomized controlled trial to determine the efficacy of HWC for primary stroke prevention in individuals 30 years and older with a 5-year CVD risk ≥10% as measured by 5-year absolute CVD risk (as measured by the PREDICT tool) at 9 months post-randomization. Eligible participants were those with a 5-year CVD risk ≥10%, with no history of stroke, transient ischemic attack, or myocardial infarction. The relative risk reduction (RRR) and odds ratios (OR) were evaluated separately in those at moderate (10%–14%) 5-year CVD risk and those at high risk (≥15%) at baseline. The Life's Simple 7 (LS7) score for lifestyle-related CVD risk, as the indicator of cardiovascular health, was a key secondary outcome.

Results

Of a total of 320 participants, 161 were randomized to the HWC group and 159 to the usual care (UC) group. HWC resulted in a statistically significant RRR of -10.9 (95% CI −21.0 to −0.9) in 5-year CVD risk in the higher CVD risk group but no change in the moderate risk group. An improvement in the total LS7 score was seen in the HWC group compared with the UC group (absolute difference = 0.485, 95% CI [0.073 to 0.897], $p = 0.02$). Improvement in blood pressure scores was statistically significantly greater in the HWC group than in the UC group for those at high risk of CVD (OR 2.28 [95% CI 1.12 to 4.63] and 1.55 [0.80 to 3.01], respectively). No statistically significant differences in mood scores, medication adherence, quality of life, and satisfaction with life scores over time or between groups were seen.

Discussion

Health and wellness coaching resulted in a significant RRR in the 5-year CVD risk compared with UC at 9 months post-randomization in patients with a high baseline CVD risk. There was no improvement in CVD risk in the moderate risk group; hence, this study did not meet the primary hypothesis. However, this treatment effect is clinically significant (number needed to treat was 43). The findings suggest that HWC has potential if further refined to improve lifestyle risk factors of stroke.

*These authors wrote the first draft of the manuscript and equally contributed to this work.

National Institute for Stroke and Applied Neurosciences (RVK, SM, ER, VLF), School of Clinical Sciences, Auckland University of Technology; Department of Statistics (ACV), The University of Auckland; Ko Awatea, Counties Manukau Health; Department of Psychology (SB-C); Department of Neurology (PAB); Department of General Practice (BA), The University of Auckland; and Te Whare Wānanga o Awanuiārangi (HE), Auckland, New Zealand.

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Introduction

The presence of ideal healthy lifestyle factors such as healthy diet, not smoking, adequate physical activity, normal body weight, and low alcohol consumption has been shown to substantially reduce risk of stroke, coronary artery disease, myocardial infarction, and overall cardiovascular disease (CVD) mortality.[1-4] Behavioral interventions and health coaching aimed at improving lifestyle behavior have been suggested for CVD prevention.[5,6] Health and wellness coaching (HWC) is a health-focused client-centric intervention based on the principles of positive psychology[7] that aims to enhance health and well-being and improve lifestyle behaviors.[8-11] Previous studies have shown that HWC has produced positive effects on health and improved well-being of patients with chronic disease,[10,12,13] but there were no previous trials of HWC for primary stroke and/or cardiovascular disease (CVD) prevention.

This study aimed to determine the efficacy of HWC for primary relative risk reduction (RRR) in adults with a moderate-to-high risk of CVD, as determined by CVD risk screening in primary care.

Methods

Trial Design Overview

We conducted a parallel, 2-arm, open-label, single-blinded randomized controlled trial, based on the PROBE design,[14] to determine the efficacy of HWC on primary stroke/CVD prevention in moderate-to-high CVD risk adults, as determined by absolute 5-year CVD risk.[15] The detailed methods of the trial have been described previously.[16] The trial was approved by the National Health and Disability Ethics Committee (ref: 16/NTA/36) and the Auckland University of Technology Ethics Committee (ref: 16/174) and registered with the Australian New Zealand Clinical Trial Registry (ACTRN N12616000379415) on 23/03/2016. Only individuals who met inclusion criteria, did not have exclusion criteria and gave written informed consent to participate in the study were randomized. The study participants were enrolled over the two-year period after trial registration (2016–2018).

The primary hypothesis was that a 15-session HWC intervention conducted over a 9-month period would result in a clinically significant 10% RRR among those at moderate 5-year CVD risk (10%–14%) and a 25% RRR among those at high risk (≥15%) with 5-year absolute risk of CVD ≥10% at baseline as compared with usual care. The secondary hypotheses were that HWC would result in (1) an improvement in the LS7 total cardiovascular risk score[17] overall and on its individual lifestyle components (blood pressure, blood glucose, blood cholesterol, weight, physical activity, smoking status, and diet) at the 9-month follow-up; (2) self-reported improvement in adherence to medication; (3) reduced cardiovascular events (new stroke or coronary heart disease, both fatal or non-fatal); (4) improved health-related quality of life; (5) reduced scores for depression; and (6) an increase in participant life satisfaction at 3, 6, 9, and 12 months post-randomization. We also registered all new stroke, transient ischemic attack (TIA), and acute CVD events.

Participants and Procedures

Potentially eligible participants were identified by clinicians through Auckland general practice clinics using preselection eligibility criteria (for details on the study protocol, see Supplementary materials, eSAP 1, links.lww.com/CPJ/A481). A list of potential participants who consented to be contacted by the research team was provided by medical practice staff to the study manager. Participants were randomized between May 25, 2016, and July 9, 2019. Eligibility criteria included individuals with a five-year CVD risk ≥10% as determined by a risk assessment by their family doctor using the online PREDICT CVD risk assessment tool,[18,19] those who were 30 years and older, those who had no history of stroke or myocardial infarction, and those who were able to provide written informed consent. Participants were not eligible if they were unable to converse in English, had significant comorbid conditions deeming them unsuitable for the trial as determined by their doctor, or had moderate-to-severe depression or anxiety as determined by the Patient Health Questionnaire (PHQ-9) cutoff score (>18).[20]

Of the 1,116 potentially eligible people, 767 were excluded from the study. Of them, 160 (21) did not meet the inclusion criteria, 255 (33%) declined to participate, 381 (50%) were not able to be contacted, and 29 (4%) had exclusion criteria (Figure). The remaining 320 participants (28.7%) were randomly assigned using an online randomization tool in the Active Research Technology (ART) database system[21] to either the HWC group (n = 161) or UC group (n = 159). Age, sex, ethnicity, level of education, marital status, employment and living status, and medical conditions were identified from questionnaires and medical records (Table 1). A CONSORT flowchart of how participants moved through the trial is presented in the Figure. There was no statistically significant difference in the study participant baseline characteristics between those "lost to follow-up" in the HWC group (22%; n = 35) and control group (11%; n = 17).

Participants were grouped into 4 equal strata according to the 4 main ethnic groups in New Zealand (NZ): Māori, Pacific, Asian, and NZ Europeans/others. Computerized online stratified minimization randomization was used to balance ethnic groups for possible prognostic factors: age (30–55 years, ≥56 years), sex, and 5-year CVD risk (10%–14% [moderate risk] and ≥15% [high risk]). Each participant was informed of their allocation and asked not to disclose this to their assessor.

Coaches attended an intensive six-week coaching course facilitated by a certified health and wellness coach. Training included core coaching competencies and code of ethics, developed by the International Coach Federation (ICF). ICF coaching is an internationally recognized approach effectively

Table 1 Demographics of the Study Participants

Characteristics	Health and wellness coaching (n = 159)	Usual care (n = 161)
Age (mean, SD)	60.8 (9.5)	60.8 (8.6)
Older than 35 years (n, %)	159 (100.0)	159 (98.8)
Sex: Male, n (%)	98 (61.6)	99 (61.5)
Ethnicity (n, %)		
Māori	34 (21.1)	34 (21.1)
Pacific	36 (23.0)	37 (23.0)
Asian	29 (18.2)	28 (17.4)
NZ European/other	60 (37.7)	62 (38.5)
Marital status: Married, n (%)	117 (73.6)	107 (66.5)
Education: University, n (%)	49 (30.8)	49 (30.4)
Employment: [a]Employed, n (%)	112 (70.4)	107 (66.5)
Living, n (%)		
With partner/family	137 (86.2)	131 (81.4)
Alone	15 (9.4)	20 (12.4)
With others	7 (4.4)	10 (6.2)
Dwelling, n (%)		
Other	5 (3.1)	15 (9.3)
Own home	106 (66.7)	92 (57.1)
Renting	44 (27.7)	49 (30.4)
Rest/nursing home, boarding house	2 (1.3)	4 (2.5)
Retirement village	2 (1.3)	2 (1.3)
Medical conditions, n (%)		
Hyperlipidemia	56 (35.2)	50 (31.0)
High BP (≥140/90 mm Hg)	57 (35.8)	51 (31.6)
Diabetes mellitus	47 (29.6)	35 (21.7)
Heart disease (except myocardial infarction)	7 (4.4)	3 (1.9)
Peripheral vascular disease	1 (0.6)	0 (0)
Epilepsy	1 (0.6)	1 (0.6)
Traumatic brain injury	1 (0.6)	1 (0.6)
Other medical conditions	15 (9.4)	17 (10.6)

[a] Includes retired, unemployed, volunteer, and sickness beneficiary.

used in various settings. Coaches received regular group supervision, facilitated by the trainer, using a small-group approach.[16] The intervention combined educational material and intensive HWC (eTable 1, links.lww.com/CPJ/A480). The details of the intervention have been described using the TIDieR guidelines[22] (eTable 2).

Those in the HWC group were assigned a coach and allocated 15 sessions with their coach. The first 2 sessions and the final session were conducted in-person and the remaining sessions over the telephone. The first 12 sessions were conducted every 2 weeks and then the remaining 3 were conducted monthly, with the coaching sessions taking place over the course of 9 months. Coaching sessions initially lasted up to 1 hour during the in-person sessions, with later telephone sessions lasting 30–40 minutes. Between the final coaching session and the 9-month assessment, HWC participants received a single short (<10 minute) telephone 'booster' call from their coach to encourage maintenance of behavior change. The outline and content of the coaching sessions are summarized in eTable 1 (links.lww.com/CPJ/A480). UC study participants received usual medical care at the discretion of treating clinicians. The type and quantity of the care received by all participants were recorded through questionnaires and documentation at each assessment. The UC group was contacted at the same frequency as the HWC group during the study for assessments. All participants were provided the NZ Heart Foundation booklet, available to the general public through their doctor or online, which included recommendations and guidelines on heathy lifestyle.[23]

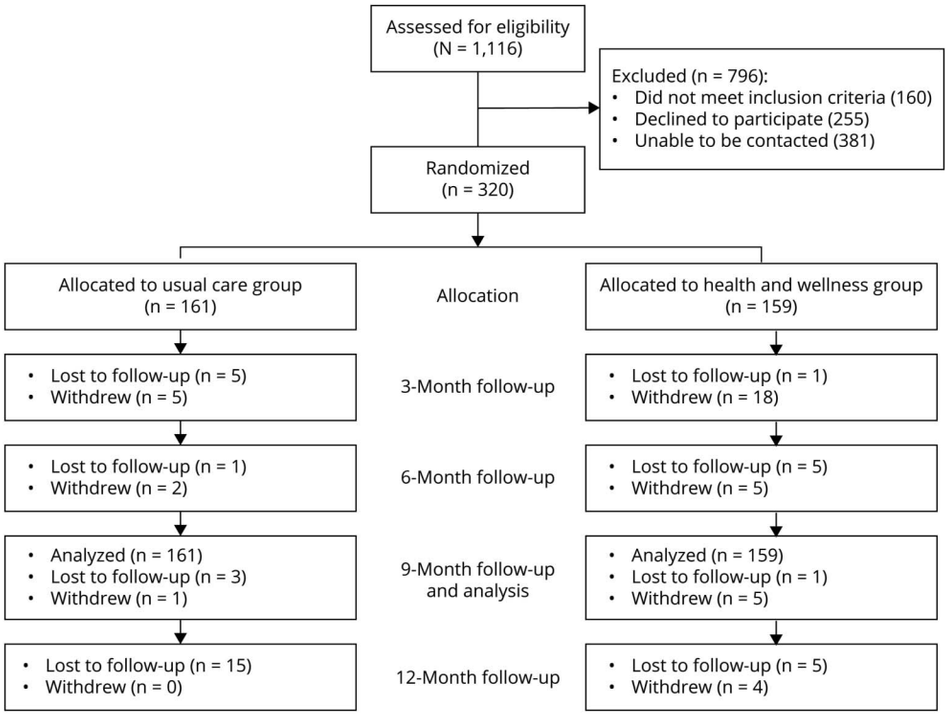
Study Outcomes

The primary outcome was a clinically significant 10% RRR among those at moderate 5-year CVD risk (10%–14%) and a 25% RRR among those at high risk (≥15%) with 5-year absolute risk of CVD ≥10% at baseline as compared with usual care assessed using the PREDICT tool (using the licensed PREDICT software)[18] at 9 months post-randomization. The key prespecified secondary outcomes were (1) the LS7[17] total score of CVD risk (total possible score of 14, with higher scores indicating better cardiovascular health) and scores on the individual lifestyle components contributing 0 for poor, 1 for intermediate, and 2 for good cardiovascular health (blood pressure, blood glucose, blood cholesterol, BMI, physical activity, smoking status, and diet) at the 9-month follow-up; (2) self-reported changes in adherence to medication[24]; (3) health-related quality of life as assessed using EQ5D[25]; (4) screening for depression using the PHQ9[20]; (5) level of TIA/stroke awareness based on open-ended questions; and (6) life satisfaction[26] at baseline, 3, 6, 9, and 12 months (eTable 3, links.lww.com/CPJ/A480). Secondary outcomes also included acute CVD events (new stroke, TIA, or myocardial infarction, both fatal and non-fatal) during the 12-month follow-up period. All outcome measures were collected in a standardized manner by the trained assessors blinded to the treatment allocation.

Statistical Considerations

Analysis was performed according to intention-to-treat principles. In the adjusted analyses, all tests of significance of hypotheses concerning treatment effect parameters were performed using a level of significance of 5% and two-sided alternatives. Covariates were included in the model for the purpose of avoiding chance bias, reducing variance in the study sample, and, in the case of logit models, to avoid bias (eTable 4, links.lww.com/CPJ/A480). Baseline values of an outcome

Figure CONSORT Study Flowchart



were always included in the model as a covariate. The 5-year CVD risk score was analyzed using censored regression treating individuals with scores greater than 30% as right-censored. Changes in relative CVD risk due to randomization arms were computed as the ratio of the effect estimate and the mean baseline CVD risk. Numbers needed to treat or harm were computed as the reciprocal of the adjusted estimated absolute risk reduction. Odds ratios for LS7 scores were also calculated. 95% confidence intervals (CIs) for all treatment effects were estimated. Continuous outcomes were analyzed using linear regression, dichotomous outcomes using logistic regression, and ordered categorical outcomes using proportional odds regression. Participant-level random effects were used in the presence of repeated measures. The effect at a given time point on a repeatedly measured outcome was estimated from the full mixed-effects model. The time point was modeled as a factor in all analyses. A likelihood ratio test of heteroscedasticity across treatment arms was conducted after unblinding, and different variances modeled if homoscedasticity was rejected at the 5% level. No baseline covariates with more than 15% missingness were retained for adjustment. Covariates with 15% values missing or less were imputed using all available selectable baseline covariates to form a model. Missing outcome data were assumed missing at random. Under this assumption, longitudinal modeling when repeated measures are available yields unbiased effect estimates.[27] When outcome data were only available at baseline and 9 months, we pooled the analyses over 15 multiply imputed data sets. Sensitivity analyses were conducted including adjusted analyses on complete-case data, unadjusted analyses on multiply imputed data using the allocated arm, and adjusted analyses on multiply imputed data with usual care used as effective allocation (for imputation only) when outcomes were missing.

The significance level was set at 0.05 against two-sided alternatives. We assumed 20% noncompliance and loss to follow-up[28] and plausible values of the baseline means and standard deviations of the absolute 5-year CVD risk. With these assumptions, 27 participants per arm in the moderate (10%–15%) CVD risk group and 13 participants per arm in the high (≥15%) CVD risk group provided 80% power to detect clinically significant[29,30] 10% and 25% RRR[31-34] in the absolute 5-year CVD risk between the HWC and UC arms 9 months after randomization in each group, respectively, without accounting for multiple testing: This was the targeted power in each ethnicity subgroup. Overall, 320 participants provided 90% power to detect approximate RRRs of 6% and 17% in the moderate and high-risk subgroups, respectively. Reporting of the trial results was done according to the CONSORT guidelines.[35]

Results

Of a total of 320 participants, 161 were randomized to the HWC group and 159 to the UC group. The demographics, baseline characteristics (Table 1), stratified mean CVD risks,

and other variables (Tables 2 and 3) of the study participants in the HWC and UC groups were similar. NZ Europeans/other was the largest ethnic group (38%), and each of the 4 other ethnic group accounted for approximately 20% of participants. Most (83%) of the sample was in the moderate CVD risk category. Participants were older by 3 to 5 years in the high-risk group compared with the moderate-risk group. The overall mean (SD) CVD risk at baseline was 16.08 (6.31) in the HWC group and 16.04 (5.95) in the UC group.

Primary Outcome

In the higher CVD risk group, there was a statistically significant adjusted reduction in the 5-year CVD risk of −2.33 percentage points (95% CI [−4.47 to −0.18], p = 0.034), equating to a 10.9% RRR (95% CI [−21.0 to −0.9]) in CVD risk in this group (Table 4). The corresponding number needed to treat was 43 (95% CI [3 to 82]). No statistically significant change (+0.65 percentage points, 95% CI [−1.37 to +2.66], p = 0.53) was seen in the moderate CVD risk group. The corresponding number needed to harm was 154 (95% CI [−324 to 632]). The 5-year CVD risk in the HWC group changed from 16.1% (6.3) at baseline to 14.6% (6.7) at 9 months while in UC, it changed from 16.0% (6.0) at baseline to 14.9% (6.8) at 9 months (complete cases, unadjusted results, Table 3). Subgroup analyses by ethnic groups showed no statistically significant difference between HWC and UC groups by ethnicity in 5-year CVD risk, or total LS7 score, except in the moderate-risk Asian group, in which there was a significant increase in the LS7 score in favor of the HWC intervention (1.78; 95% CI 0.24 to 3.33) (eTable 5, links.lww.com/CPJ/A480; additional details of the inferential analysis are present in the link provided in the eAppendix, p. 12).

Secondary Outcomes

Adjusted regression analyses showed a statistically significant positive difference in change from baseline in the total LS7 score between the HWC group and the UC group (0.49, 95% CI [0.07,0.90], p = 0.02) (Table 4), but this difference was only statistically significant in the moderate CVD risk group (0.72, 95% CI [0.07 to 1.38], p = 0.03). There was also statistically significantly greater improvement in blood pressure control (OR = 1.83, 95% CI [1.13 to 2.97], p = 0.01), especially in the high CVD risk group (OR = 2.28, 95% CI [1.12 to 4.63], p = 0.02), where there was also better control of blood glucose (OR = 2.28, 95% CI [1.12 to 4.63], p = 0.02). No statistically significant group differences were found in mood scores, medication adherence (including dosage and types of the medications for the treatment of arterial hypertension, diabetes, and elevated blood cholesterol), quality of life, and satisfaction with life scores (Table 3). No stroke, TIA, or acute CVD event was observed in either group during the follow-up assessments.

Systolic and diastolic blood pressure increased from baseline to 9 months follow-up in both HWC and UC groups (Table 5), but the increases in the HWC group (5 mm Hg and 4 mm Hg, respectively) were statistically significantly smaller than those in the UC group (11 mm Hg and 7 mm Hg, respectively; p = 0.019 and 0.015, respectively), after adjustment for baseline values. At the 9-month post-randomization follow-up, there were no statistically significant changes in mean BMI or concentrations of fasting blood glucose and total cholesterol between the groups. No unintended effects of the HWC intervention were registered; however, the "lost to follow up" rate was twice as high in the HWC group compared with the UC group.

Lost to Follow-up and Missed Assessments at 9 Months

Fifty-seven baseline characteristics were assessed using t-tests and chi-square tests between participants lost to follow-up or with all data missing at 9 months ("Missing"; n = 52) and those who completed the 9-month assessment ("Assessed"; n = 268). Five of these characteristics displayed a raw p-value smaller than 5% (Table 6).

Table 2 Demographic Characteristics and Baseline CVD Risk Stratified by Randomization Arm and Baseline CVD Risk

	Health and wellness coaching		Usual care	
Baseline CVD risk	Moderate CVD risk (n = 85)	High CVD risk (n = 74)	Moderate CVD risk (n = 85)	High CVD risk (n = 76)
Mean age	58.3 (8.4)	62.6 (10.0)	59.5 (7.3)	60.7 (10.0)
Sex (male %)	63.5	59.5	60.0	63.2
Ethnicity (%)				
Māori	22.4	20.3	21.2	21.1
Pacific	22.4	23.0	18.8	27.6
Asian	16.5	20.3	18.8	15.8
European/other	38.8	36.5	41.2	35.5
CVD risk (%) baseline	11.6 (1.2)	21.2 (5.9)	12.1 (1.3)	20.5 (5.9)
Overall mean risk (%)	16.08 (6.31)		16.04 (5.95)	

Table 3 Outcome Means and Standard Deviations by Treatment Arm at Baseline and 9 Months (Unadjusted)

	Health and wellness coaching		Usual care	
Outcome measure, means (SD)	Baseline	9-mo	Baseline	9-mo
5-y CVD risk (%)	16.08 (6.31)	14.60 (6.69)	16.04 (5.95)	14.88 (6.75)
5-y CVD risk median (Q1, Q3)	14 (11, 18.5)	13 (10, 17)	14 (12, 17)	14 (10, 19)
LS7 scores				
Total	7.27 (2.12)	7.42 (2.00)	7.43 (2.22)	7.12 (2.38)
Smoking	1.65 (0.75)	1.75 (0.64)	1.57 (0.81)	1.65 (0.74)
Body mass index	0.62 (0.74)	0.64 (0.74)	0.67 (0.73)	0.73 (0.78)
Physical activity	1.04 (0.87)	1.07 (0.76)	1.09 (0.85)	0.96 (0.83)
Health diet	1.06 (0.64)	1.06 (0.69)	1.09 (0.67)	1.09 (0.69)
Total cholesterol	1.14 (0.61)	1.27 (0.65)	1.12 (0.66)	1.11 (0.64)
Blood pressure	0.75 (0.64)	0.70 (0.68)	0.72 (0.64)	0.43 (0.60)
Fasting glucose	1.01 (0.79)	0.98 (0.82)	1.17 (0.78)	1.13 (0.82)
Alcohol use 3 mo (yes; %)	56 (50)	50 (50)	55 (50)	58 (50)
Alcohol frequency				
Once or more a day	16 (16.7)	6 (10.0)	19 (18.4)	15 (18.3)
Every 2–10 d	43 (44.8)	29 (48.3)	48 (46.6)	38 (46.3)
Once a fortnight or less	37 (38.5)	24 (40.0)	36 (35.0)	29 (35.4)
Don't know/refuse	0 (0.0)	1 (1.7)	0 (0.0)	0 (0.0)
Satisfaction with life (Satisfaction with Life Scale score)	23.83 (6.33)	25.61 (6.24)	23.52 (6.12)	26.22 (5.71)
EuroQol-5 dimensions	75.42 (16.58)	77.22 (13.88)	76.50 (16.43)	77.70 (15.48)
Patient Health Questionnaire (PHQ9)	5.03 (4.69)	3.44 (4.00)	4.66 (5.18)	2.87 (3.72)
Adherence (Morisky Medical Adherence Scale score)	2.07 (1.49)	1.84 (1.18)	2.00 (1.28)	1.86 (1.29)

After adjusting for multiple testing using the Hochberg procedure[36] (which is less conservative than the Holm,[37] Benjamini and Hochberg,[38] or Bonferroni procedures[39]), none of these differences proved significant at the 5% level. The full set of results is available in the Supplementary materials.

Discussion

This study has not borne out the primary hypothesis because there was no improvement in CVD risk in the moderate-risk group. In patients with a high baseline CVD risk, health and wellness coaching resulted in a 10.9% RRR in the 5-year CVD risk ($p = 0.03$) compared with UC at 9 months post-randomization. This reduction is inferior to the one posited in the primary hypothesis. However, this treatment effect is clinically significant (number needed to treat was 43). The positive effects of the intervention in the high CVD risk group were consistent with the positive effects of HWC on health and improved well-being of patients with some chronic diseases.[10,12,13] However, in the moderate-risk group, there was no improvement in CVD risk possibly because these people were 3–5 years younger than the high-risk group, leading to differences in risk factor and demographic profiles. Because there was no significant difference in the adherence, types, and dosages of medications used for blood pressure and lipid-lowering therapy between the HWC and UC groups, the treatment effect achieved is likely to be related to the behavioral components of the HWC intervention. Age has the greatest effect on CVD risk,[40] and it is likely that in high CVD risk people, smaller differences in the exposure to risk factors (for example, the observed better blood pressure control) result in greater reductions in CVD risk than the same level of reductions in people with moderate CVD risk. It is also possible that the PREDICT algorithm did not pick up changes in several of the areas being targeted by the intervention such as lifestyle factors other than smoking, because this content is not well represented in the outcome measure. A recent meta-analysis of multifactorial lifestyle interventions for primary prevention of CVD also reported better CVD risk improvement in high CVD risk groups.[41]

Table 4 Adjusted Regression Analysis of Primary and Secondary Outcomes at 9 Months Post-Randomization

Outcome measures	Effect type	Overall			Baseline CVD risk 10%–14%			Baseline CVD risk ≥15%		
		Effect estimate	95% CI	*p* Value	Effect estimate	95% CI	*p* Value	Effect estimate	95% CI	*p* Value
5-y CVD risk (% points)	Difference[a]	−0.68	−2.18 to +0.82	0.37	0.65	−1.37 to 2.66	0.53	−2.33	−4.47 to −0.18	0.034
	Change in RR[b]	−4.2%	−13.6 to +5.1		5.5%	−11.5 to +22.5		−10.9%	−21.0 to −0.9	
LS7 components										
Smoking status	Odds ratio[c]	1.11	0.05 to 23.00	0.95	1.62	0.02 to 148	0.83	0.65	0.01 to 51	0.85
BMI	Odds ratio[c]	1.23	0.64 to 2.37	0.53	1.17	0.66 to 2.08	0.59	0.80	0.43 to 1.49	0.48
Physical activity	Odds ratio[c]	1.53	0.88 to 2.66	0.13	1.47	0.69 to 3.13	0.31	1.59	0.70 to 3.64	0.27
Healthy diet score	Odds ratio[d]	0.93	0.52 to 1.68	0.81	1.22	0.56 to 2.63	0.61	0.66	0.27 to 1.60	0.35
Total cholesterol	Odds ratio[c]	1.49	0.95 to 2.33	0.09	1.44	0.79 to 2.64	0.23	1.55	0.82 to 2.96	0.18
Blood pressure	Odds ratio[d]	1.83	1.13 to 2.97	0.014	1.55	0.80 to 3.01	0.20	2.28	1.12 to 4.63	0.024
Fasting glucose	Odds ratio[c]	0.90	0.56 to 1.44	0.66	0.99	0.55 to 1.77	0.96	0.51	0.27 to 0.97	0.042
Total LS7 score	Difference[a]	0.49	0.07 to 0.90	0.02	0.72	0.07 to 1.38	0.03	−0.04	−0.74 to 0.66	0.91
PhQ9 (Mood)	Difference[a]	0.34	−0.33 to 1.02	0.32	0.38	−0.68 to 1.43	0.48	0.85	−0.27 to 1.97	0.14
MMAS (Medication adherence)	Difference[a]	−0.16	−0.45 to 0.13	0.27	No subgroup analyses planned					
Quality of life EQ5D VAS	Difference[a]	0.25	−2.57 to 3.08	0.86						
SWL satisfaction with life	Difference[a]	−0.23	−1.21 to 0.76	0.65						

Abbreviations: CVD = cardiovascular disease; HWC = Health and Wellness Coaching.
[a] Adjusted absolute difference at 9 mo between HWC and UC arms in mean change from baseline from linear regression.
[b] Change in RR based on the mean CVD risk at baseline.
[c] Odds ratio at 9 mo from proportional odds regression. The subscores were treated as 3-level ordinal variables. The OR is the factor by which the odds of an outcome increasing by one level (moderate vs poor, good vs moderate) are multiplied in the intervention arm compared with the control arm. The ORs are the same regardless of the starting level (poor or moderate). An OR >1 indicates benefit.
[d] Odds ratio at 9 mo from logistic regression. The subscores were dichotomized. The OR represents the factor by which the odds of a positive outcome are multiplied in the intervention arm as compared with the control arm. An OR >1 indicates benefit.

Elevated blood pressure is the single most important modifiable risk factor of stroke, and our findings of improved of blood pressure control in the HWC group compared with the UC group (overall and in moderate and high CVD risk groups separately) highlights the potential of the HWC intervention for primary stroke prevention. Although blood pressure increased in both groups over the duration of the study, this increase was smaller in the HWC group. Because the percentage of study participants receiving blood pressure-lowering medications was not statistically significantly

Table 5 Changes in LS7 Metabolic Risk Measures

Metric	Usual care		Health and wellness coaching		Adjusted *p* value[a]
	Baseline	9 mo	Baseline	9 mo	
Systolic blood pressure, mean mm Hg (SD)	133 (13.2)	144 (19.1)	132 (14.8)	137 (19.5)	0.019
Diastolic blood pressure, mean mm Hg (SD)	80 (8.6)	87 (11.7)	78 (9.6)	82 (11.3)	0.015
Blood glucose, mean (SD)	6.9 (3.1)	6.6 (2.7)	7.0 (3.1)	7.0 (2.7)	0.212
Total blood cholesterol, mean (SD)	5.2 (1.3)	5.1 (1.2)	5.0 (1.2)	4.7 (1.3)	0.071
BMI, mean (SD)	31.7 (7.7)	31.7 (8.0)	32.3 (10.2)	32.8 (10.5)	0.160

[a] *p*-value to test that the differences in changes from baseline is different from 0, adjusted for baseline.

Table 6 Baseline Characteristics That Differ at the 5% Level Between Participants Missing and Assessed at 9 Months

	Missing (n = 52)	Assessed (n = 268)		
Outcome at baseline	Mean (SE)	Mean (SE)	Direction of benefit	Unadjusted *p* value
LS7 smoking score (out of 3)	1.35 (0.93)	1.66 (0.74)	↑	0.007
LS7 cholesterol score (out of 3)	1.33 (0.65)	1.09 (0.63)	↑	0.013
LS7 glucose score (out of 3)	1.31 (0.81)	1.04 (0.78)	↑	0.025
CVD risk	0.14 (0.05)	0.16 (0.06)	↓	0.045
	Count (%)	**Count (%)**		
No medical condition	15 (29%)	42 (16%)	↑	0.038

Abbreviations: CVD = cardiovascular disease; SE = standard error.

different between the groups, the reason for increasing blood pressure in both groups remains unclear. The *p*-values were not corrected for multiple comparisons, and the difference may be due to chance. Our trial was not powered to determine the effect of the intervention on other individual lifestyle risks. The findings of an approximately 0.5-point increase in the overall LS7 score and a 0.7-point increase in the moderate-risk group are equivalent to a 4% and 5% annual stroke incidence reduction, respectively.[17]

We did not find any significant differences in mood, satisfaction with life, quality of life, or medication adherence scores. This may be due to the floor effect of screening, as those with depression and anxiety at screening were not eligible to participate. Hence, the mean mood scores in both groups at baseline were in the normal-to-mild range, which means further improvement was unlikely. The medication adherence score at baseline was low in both groups and did not improve at 9 months. The reasons for this are unclear, and ways to target medication adherence in HWC need to be explored further. One possibility is that participants' behavior may have been modified by knowing they were in a trial and would be asked about their medications.

The study had several strengths and limitations. The main strength was that the sample was ethnically diverse and covered both men and women of age 35+ years, thus enhancing generalizability of the findings for people with moderate-to-high CVD risk. HW coaches were community workers demonstrating the ability of non-health professionals to be trained to deliver HWC. The HWC intervention was able to be delivered predominantly by telephone, making it more feasible to roll out into the community. Finally, the target sample population was at an increased risk of CVD, hence appropriate for a multifactorial lifestyle modification intervention. The main weakness of the study was that while CVD risk was the primary outcome, the Framingham-based calculation of CVD risk assigns greater weight to age and ethnicity, which are not amenable to lifestyle changes. In hindsight, addressing primary stroke prevention in this population required a metric focused on lifestyle. The LS7 and more detailed measures of lifestyle risk may be more appropriate measures in future studies, with a longer term of follow-up (ideally, with the hard primary end points, such as new strokes/TIA and acute CVD events). The intervention was not tailored to be culturally responsive to Māori, Pacific, and Asian groups, and future HWC interventions should be co-designed with relevant cultural advisors, so they are better tailored to different cultural groups.

In conclusion, HWC has the potential to improve cardiovascular health in those with high cardiovascular disease risk. Further work is required to improve the efficacy of HWC in those at moderate cardiovascular disease risk.

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Appendix Authors

Name	Location	Contribution
Rita V. Krishnamurthi, BSc, MApplSc, PhD	National Institute for Stroke and Applied Neurosciences, School of Clinical Sciences, Auckland University of Technology, New Zealand	Drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data; study concept or design; analysis or interpretation of data
Alain C. Vandal, PhD	Department of Statistics, The University of Auckland; Ko Awatea, Counties Manukau Health, New Zealand	Analysis or interpretation of data
Suzanne Barker-Collo, MA, PhD	Department of Psychology, The University of Auckland, New Zealand	Study concept or design; analysis or interpretation of data
Susan Mahon, PhD	National Institute for Stroke and Applied Neurosciences, School of Clinical Sciences, Auckland University of Technology, New Zealand	Major role in the acquisition of data; analysis or interpretation of data
P. Alan Barber, MD, PhD	Department of Neurology, The University of Auckland, New Zealand	Study concept or design; analysis or interpretation of data
Bruce Arroll, MD, PhD	Department of General Practice, The University of Auckland, New Zealand	Study concept or design; analysis or interpretation of data
Elaine Rush, PhD	National Institute for Stroke and Applied Neurosciences, School of Clinical Sciences, Auckland University of Technology, New Zealand	Study concept or design; analysis or interpretation of data
Hinemoa Elder, PhD	Te Whare Wānanga o Awanuiārangi, Auckland, New Zealand	Study concept or design; analysis or interpretation of data
Valery L. Feigin, MD, PhD, FAAN	National Institute for Stroke and Applied Neurosciences, School of Clinical Sciences, Auckland University of Technology, New Zealand	Drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data; study concept or design; analysis or interpretation of data

References

1. Larsson SC, Akesson A, Wolk A. Primary prevention of stroke by a healthy lifestyle in a high-risk group. *Neurology*. 2015;84(22):2224-2228. doi:10.1212/WNL.0000000000001637
2. Gorelick PB. Primary prevention of stroke: impact of healthy lifestyle. *Circulation*. 2008;118(9):904-906. doi:10.1161/CIRCULATIONAHA.108.800169
3. Chiuve SE, Rexrode KM, Spiegelman D, Logroscino G, Manson JE, Rimm EB. Primary prevention of stroke by healthy lifestyle. *Circulation*. 2008;118(9):947-954. doi:10.1161/CIRCULATIONAHA.108.781062
4. Lin MP, Ovbiagele B, Markovic D, Towfighi A. "Life's Simple 7" and long-term mortality after stroke. *J Am Heart Assoc*. 2015;4(11):e001470. doi:10.1161/JAHA.114.001470
5. LeFevre ML, U.S. Preventive Services Task Force. Behavioral counseling to promote a healthful diet and physical activity for cardiovascular disease prevention in adults with cardiovascular risk factors: U.S. Preventive Services Task Force recommendation statement. *Ann Intern Med*. 2014;161(8):587-593. doi:10.7326/M14-1796
6. Linden A, Butterworth SW, Prochaska JO. Motivational interviewing-based health coaching as a chronic care intervention. *J Eval Clin Pract*. 2010;16(1):166-174. doi:doi:10.1111/j.1365-2753.2009.01300.x
7. Seligman ME, Steen TA, Park N, Peterson C. Positive psychology progress: empirical validation of interventions. *Am Psychologist*. 2005;60(5):410-421. doi:10.1037/0003-066X.60.5.410
8. Huffman MH. Health coaching: a fresh approach for improving health outcomes and reducing costs. *AAOHN J*. 2010;58(6):245-250; quiz 251-252. doi:10.3928/08910162-20100526-02
9. Jefferson V, Jaser SS, Lindemann E, et al. Coping skills training in a telephone health coaching program for youth at risk for type 2 diabetes. *J Pediatr Health Care*. 2011;25(3):153-161. doi:10.1016/j.pedhc.2009.12.003
10. Kivelä K, Elo S, Kyngäs H, Kääriäinen M. The effects of health coaching on adult patients with chronic diseases: a systematic review. *Patient Educ Couns*. 2014;97(2):147-157. doi:10.1016/j.pec.2014.07.026
11. Kreitzer MJ, Sierpina VS, Lawson K. Health coaching: innovative education and clinical programs emerging. *Explore (NY)*. 2008;4(2):154-155. doi:10.1016/j.explore.2007.12.007
12. Wolever RQ, Eisenberg DM. What is health coaching anyway? Standards needed to enable rigorous research. *Arch Intern Med*. 2011;171(22):2017-2018. doi:10.1001/archinternmed.2011.508
13. Vale MJ, Jelinek MV, Best JD, et al; COACH Study Group. Coaching patients on achieving cardiovascular health (COACH): a multicenter randomized trial in patients with coronary heart disease. *Arch Intern Med*. 2003;163(22):2775-2783. doi:10.1001/archinte.163.22.2775
14. Hansson L, Hedner T, Dahlöf B. Prospective randomized open blinded end-point (PROBE) study. A novel design for intervention trials. Prospective Randomized Open Blinded End-Point. *Blood Press*. 1992;1(2):113-119. doi:10.3109/08037059209077502
15. New Zealand Guidelines Group. *Evidence-based Best Practice Guideline: The Assessment and Management of Cardiovascular Risk*. New Zealand Guidelines Group; 2003.
16. Mahon S, Krishnamurthi R, Vandal A, et al. Primary prevention of stroke and cardiovascular disease in the community (PREVENTS): methodology of a health wellness coaching intervention to reduce stroke and cardiovascular disease risk, a randomized clinical trial. *Int J Stroke*. 2018;13(2):223-232. doi:10.1177/1747493017730759
17. Kulshreshtha A, Vaccarino V, Judd SE, et al. Life's simple 7 and risk of incident stroke: the reasons for geographic and racial differences in stroke study. *Stroke*. 2013;44:1909-1914. doi:10.1161/STROKEAHA.111.000352
18. Pylypchuk R, Wells S, Kerr A, et al. Cardiovascular disease risk prediction equations in 400 000 primary care patients in New Zealand: a derivation and validation study. *Lancet*. 2018;391(10133):1897-1907. doi:10.1016/S0140-6736(18)30664-0
19. Wells S, Riddell T, Kerr A, et al. Cohort profile: the PREDICT cardiovascular disease cohort in New Zealand primary care (PREDICT-CVD 19). *Int J Epidemiol*. 2017;46(1):22. doi:10.1093/ije/dyv312
20. Kroenke K, Spitzer R, Williams J. The PHQ-9: validity of a brief depression severity measure. *J Gen Intern Med*. 2001;16(9):606-613. doi:10.1046/j.1525-1497.2001.016009606.x
21. Witt E. *Overview of ART Database*. AUT University; 2017.
22. Hoffmann TC, Glasziou PP, Boutron I, et al. Better reporting of interventions: template for intervention description and replication (TIDieR) checklist and guide. *BMJ*. 2014;348:g1687. doi:10.1136/bmj.g1687
23. Heart Foundation of New Zealand. *Lowering Your Risk of Heart Attack and Stroke—Booklet*. Accessed August 26, 2019. heartfoundation.org.nz/resources/lowering-your-risk-of-heart-attack-and-stroke-booklet.
24. Morisky DE, Green LW, Levine DM. Concurrent and predictive validity of a self-reported measure of medication adherence. *Med Care*. 1986;24(1):67-74. doi:10.1097/00005650-198601000-00007
25. Szende A, Janssen M, Cabases J, Ramos Goñi J. Self-reported population health: an international perspective based on EQ-5D. *Value in Health*. 2013;16(7):A464. doi:10.1016/j.jval.2013.08.812
26. Diener E, Chan M. Happy people live longer: subjective well-being contributes to health and longevity. *Appl Psychol Health Well-Being*. 2011;3:1-43. doi:doi:10.1111/j.1758-0854.2010.01045.x
27. Carpenter JR, Kenward MG. Missing data in randomised controlled trials: a practical guide. In: *Health Technology Assessment Methodology Programme*; 2007.
28. Wittes J. Sample size calculations for randomized controlled trials. *Epidemiol Rev*. 2002;24(1):39-53. doi:10.1093/epirev/24.1.39
29. Cox JL, Carr B, Vallis TM, Szpilfogel C, O'Neill BJ. A novel approach to cardiovascular health by optimizing risk management (ANCHOR): a primary prevention initiative examining the impact of health risk factor assessment and management on cardiac wellness. *Can Journal Cardiol*. 2011;27(6):809-817. doi:10.1016/j.cjca.2011.04.013
30. Mc Namara KP, George J, O'Reilly SL, et al. Engaging community pharmacists in the primary prevention of cardiovascular disease: protocol for the pharmacist assessment of adherence, risk and treatment in cardiovascular disease (PAART CVD) pilot study. *BMC Health Serv Res*. 2010;10:264. doi:10.1186/1472-6963-10-264
31. Jackson R, Lawes CMM, Bennett DA, Milne RJ, Rodgers A. Treatment with drugs to lower blood pressure and blood cholesterol based on an individual's absolute cardiovascular risk. *Lancet*. 2005;365(9457):434-441. doi:10.1016/S0140-6736(05)17833-7
32. Wald NJ, Law MR. A strategy to reduce cardiovascular disease by more than 80%. *BMJ*. 2003;326:1419. doi:10.1136/bmj.326.7404.1419
33. Czernichow S, Zanchetti A, Turnbull F, et al; Blood Pressure Lowering Treatment Trialists' Collaboration. The effects of blood pressure reduction and of different blood pressure-lowering regimens on major cardiovascular events according to baseline blood pressure: meta-analysis of randomized trials. *J Hypertens*. 2011;29(1):4-16. doi:10.1097/HJH.0b013e32834000be
34. Turnbull F, Blood Pressure Lowering Treatment Trialists' Collaboration. Effects of different blood-pressure-lowering regimens on major cardiovascular events: results of prospectively-designed overviews of randomised trials. *Lancet*. 2003;362(9395):1527-1535. doi:10.1016/s0140-6736(03)14739-3

35. Blom JW, Muth C, Glasziou P, et al. Describing deprescribing trials better: an elaboration of the CONSORT statement. *J Clin Epidemiol.* 2020;127:87-95. doi:10.1016/j.jclinepi.2020.07.011
36. Hochberg Y. A sharper bonferroni procedure for multiple tests of significance. *Biometrika.* 1988;75(4):800-802. doi:10.2307/2336325
37. Holm S. A simple sequentially rejective multiple test procedure. *Scand J Stat.* 1979;6:65-70.
38. Benjamini Y, Hochberg Y. Controlling the false discovery rate: a practical and powerful approach to multiple testing. *J R Stat Soc Ser B (Methodol).* 1995;57(1):289-300. doi:10.1111/j.2517-6161.1995.tb02031.x
39. Dunn OJ. Multiple comparisons among means. *J Am Stat Assoc.* 1961;56(293):52-64. doi:10.2307/2282330
40. Dhingra R, Vasan RS. Age as a risk factor. *Med Clin North Am.* 2012;96(1):87-91. doi:10.1016/j.mcna.2011.11.003
41. Sisti L, Dajko M, Campanella P, Shkurti E, Ricciardi W, De Waure C. The effect of multifactorial lifestyle interventions on cardiovascular risk factors: a systematic review and meta-analysis of trials conducted in the general population and high risk groups. *Prev Med.* 2018;109:82-97. doi:10.1016/j.ypmed.2017.12.027

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