

# Effects on cardiovascular risk factors of three 48-week community-based exercise interventions



Matthew Wade<sup>1</sup>, Dr Steven Mann<sup>1</sup>, Prof Alfonso Jimenez<sup>1,3</sup>, Dr Sarah Domone<sup>1</sup>, Dr Chris Beedie<sup>1,2</sup>

<sup>1</sup> ukactive Research Institute, 26–28 Bedford Row, London WC1, United Kingdom

<sup>2</sup> Centre for Applied Biological and Exercise Sciences, Faculty of Health and Life Sciences, Coventry University, Coventry, United Kingdom

<sup>3</sup> School of Human and Life Sciences, Canterbury Christ Church University, Kent, United Kingdom

## Abstract

A sedentary lifestyle is associated with cardiovascular disease (CVD). Blood pressure (BP) and blood lipids (cholesterols) are key mediators of CVD. A substantial body of evidence demonstrates reduced CVD risk following systematic physical activity (PA). Despite this evidence, CVD continues to rise, and public health PA initiatives often fail to demonstrate clinically relevant effects. In short, laboratory efficacy often fails to translate into real-world effectiveness.

**Purpose:** We investigated the effectiveness of three physical activity interventions. We did so using a Phase-IV clinical trial model, in which all treatments were administered in uncontrolled community settings, and in which all interventions and measures were delivered by, and conducted by, community health centre staff.

**Methods:** Participants were sedentary individuals receiving no medication to reduce CVD risk (n=238, age 43±5 years). Participants selected a PA or exercise (EX) pathway. Those who selected PA were randomised to either fitness centre based PA counselling delivered by an exercise professional (PAC) or a wait-list control condition (CONT). Those who selected EX were randomised to either a structured exercise program (STRUC) or unstructured fitness centre use (FREE). Measures were mean arterial pressure (MAP; mmHg) estimated using the formula Diastolic Blood Pressure (BP) + (0.33 x (Systolic BP – Diastolic BP)), and total cholesterol (TC; mmol/L). Measures were taken at baseline and 48 weeks. Data were analysed using paired-sample t-tests.

**Results:** Data analysis for cholesterol indicated a small but statistically significant increase in TC in CONT (M=0.8%, SD=0.5, p = .005). TC was however approximately equivalent to baseline in PAC and FREE and reduced, although not significantly, in STRUC. Data for blood pressure indicated a statistically significant decrease in MAP in STRUC (M=2.5%, SD=8.3, P = .004). MAP was reduced, although not significantly, in all other treatments and CONT.

**Conclusions:** Data suggest that over 48 weeks, all forms of exercise might be effective in offsetting increases in total cholesterol associated with inactivity. Furthermore, a structured exercise programme might be more effective than either unstructured exercise or physical activity counselling in reducing mean arterial blood pressure.

## Introduction

The translation of laboratory findings into practice has been described as one of the greatest challenges facing health promotion and disease prevention [1, 2]. On the basis of the above it is not unreasonable to suggest that further community based physical activity intervention trials on relevant participant groups are warranted. Arguably, to inform policy and practice, such trials should replicate real world delivery, firstly in terms of how they are communicated, delivered and managed by exercise professionals, and secondly in terms of how they are accessed and experienced by patients [3]. Data to emerge from studies adopting such designs will have relevance to public health policy and practice [4].

We report the findings of a 48-week study, in which three competing exercise interventions were tested in a previously sedentary population across multiple community fitness centres in the UK. Dependent variables were total cholesterol (TC) and mean arterial blood pressure (MAP), two commonly used metrics, each widely used in medicine, each summarising multi-component physiological variables related to cardiovascular disease.

We hypothesised that a structured exercise programme would elicit greater reductions in both MAP and TC than unstructured exercise, physical activity counselling or measurement only.

## Methods

Participants were sedentary individuals receiving no medication to reduce CVD risk (n=238, age 43±5 years). Participants selected a PA or exercise (EX) pathway. For the structured exercise programme (STRUC) participants had access to all fitness centre facilities and received an individualised exercise programme to follow which combined aerobic and resistance training. The unstructured fitness centre based exercise (FREE) allowed participants to have access to all fitness centre facilities but they received no structured programme to follow. Physical activity counselling (PAC) participants did not have access to any fitness centre facilities. Exercise professionals were instructed to meet participants once each month and deliver counselling sessions according to the 5 A's model [5]. The control group (CONT) was wait-list group that did not have access to any fitness centre facilities, nor did they receive any PAC.

Measures were mean arterial pressure (MAP; mmHg) estimated using the formula Diastolic Blood Pressure (BP) + (0.33 x (Systolic BP – Diastolic BP)), and total cholesterol (TC; mmol/L). Measures were taken at baseline and 48 weeks. Data were analysed using paired-sample t-tests.

## Results

Table 1. One-way ANOVA comparing percentage change in dependant variables at baseline and 48 weeks between treatments

Dependant variable	STRUC		FREE		PAC		CONT		F	p
	M	SD	M	SD	M	SD	M	SD		
Mean arterial pressure	-2.2	8.6	-0.9	6.9	-0.9	6.6	-1.1	0.8	0.62	.60
Total cholesterol	-0.5	11.3	0.9	11.2	1.0	11.8	4.1	10.9	2.26	.08

\* Denotes statistically significant difference between treatment groups where alpha was set at 0.05

Figure 1. Mean percentage changes in MAP and TC for each treatment group

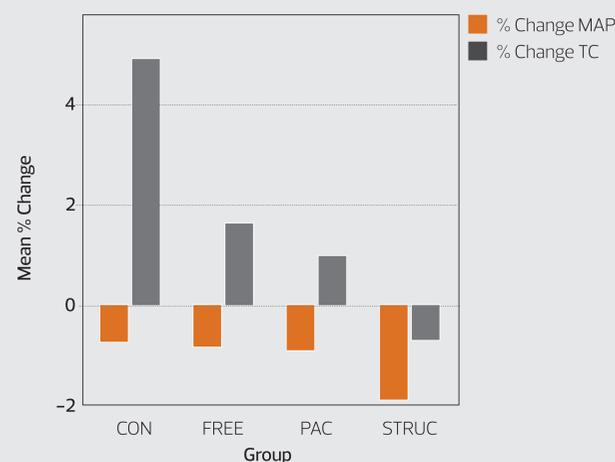


Table 2. Paired samples T-test comparing absolute changes in dependant variables at baseline and 48 weeks for each treatment

Baseline fitness group		Change over baseline		95% CI		t	df	Sig
		M	SD	Lower	Upper			
STRUC	Mean arterial pressure	-2.5	8.3	-4.1	-0.8	-2.921	96	.004*
	Total cholesterol	-0.1	0.5	-0.1	0.1	-.842	98	.402
FREE	Mean arterial pressure	-1.0	6.3	-2.3	0.2	-1.606	95	.112
	Total cholesterol	+0.1	0.5	-0.1	0.1	.006	98	.995
PAC	Mean arterial pressure	-1.2	6.5	-2.8	0.3	-1.553	67	.125
	Total cholesterol	+0.1	0.5	-0.1	0.1	.389	62	.698
CONT	Mean arterial pressure	-1.3	7.9	-3.2	0.6	-1.399	67	.167
	Total cholesterol	+0.2	0.5	0.1	0.3	2.877	71	.005*

\* Denotes statistically significant difference between treatment groups where alpha was set at 0.05

One-way ANOVA results comparing percentage change in dependant variables at baseline and 48 between treatments revealed no significant differences. Further data analysis, using a paired-samples t-test, indicated for cholesterol a small but statistically significant increase in TC in CONT (M=0.8%, SD=0.5, p = .005). TC was however approximately equivalent to baseline in PAC and FREE and reduced, although not significantly, in STRUC. Paired-sample t-test data for blood pressure indicated a statistically significant decrease in MAP in STRUC (M=2.5%, SD=8.3, P = .004). MAP was also reduced, although not significantly, in all other treatments as well as CONT.

## Summary and Conclusion

Data suggest that over 48 weeks, all forms of exercise were likely effective in offsetting increases in total cholesterol associated with inactivity, and that a structured exercise programme was more effective than either unstructured exercise or physical activity counselling in reducing mean arterial blood pressure. The magnitude of the effects observed in this study are arguably clinically relevant. In relation to TC, previous research has reported that reductions in serum cholesterol of around 0.6mmol/L can reduce the incidence of ischaemic heart disease by 54% at the age of 40 years reducing to 19% at 80 years [6]. In relation to MAP, previous research has indicated that a reduction of as little as 3mmHg will reduce the risk of coronary heart disease by 5–9%, strokes by 8–14% and all-cause mortality by 4% [7].

Future research should seek to replicate the above using direct measures of physical activity to identify the degree to which any observed effects were mediated by variations in actual physical activity levels as opposed to variations in treatment content.

## References

- [1] Kerner J, Rimer B, Emmons K. Introduction to the special section on dissemination: dissemination research and research dissemination: how can we close the gap? *Health Psychology*. 2005;24(5):443.
- [2] Sly JR, Jandorf L, Dhulkifil R et al. Challenges to Replicating Evidence-Based Research in Real-World Settings: Training African-American peers as Patient Navigators for Colon Cancer Screening. *Journal of Cancer Education*. 2012;27(4):680–6.
- [3] Beedie C, Mann S, Jimenez A et al. Death by Effectiveness: Exercise as Medicine Caught in the Efficacy Trap! *British Journal of Sports Medicine*. 2015; Published Online First: 12 Feb 2015. doi:10.1136/bjsports-2014-094389.
- [4] Hohmann AA, Shear MK. Community-based intervention research: Coping with the "noise" of real life in study design. *American Journal of Psychiatry*. 2002;159(2):201–7.
- [5] Meriwether RA, Lee JA, Lafleur AS, Wiseman P: Physical activity counseling. *Am Fam Physician* 2008, 77:1129–1136.
- [6] Law MR, Wald NJ, Thompson SG. By how much and how quickly does reduction in serum cholesterol concentration lower risk of ischaemic heart disease? *BMJ*. 1994;308(6925):367–72.
- [7] Strasser B, Schoberberger W. Evidence for resistance training as a treatment therapy in obesity. *J Obes*. 2011.

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ukactive is a not-for-profit body comprised of members and partners from across the UK physical activity sector. Our focus is a long-standing and uncompromising vision to get more people, more active, more often.

The ukactive Research Institute holds a unique partnership between academia and industry to turn the tide of physical inactivity. It co-ordinates and delivers research designed to improve the use of physical activity in everyday life. Established in 2010, the ukactive Research Institute seeks to answer 'how can we get more people, more active, more often?'

The Research Institute aims to bridge the evidence gap between traditional laboratory based 'exercise is medicine' research and real world interventions. This is achieved by conducting research assessing the effectiveness of interventions on directly measured physical activity levels, clinically relevant markers of cardiovascular and metabolic health, and other core variables in real world interventions.

These questions will in time relate as much to economic, social and political factors as to scientific and health factors. Each project undertaken will, when completed, be publishable in a peer-reviewed journal article, constitute the basis of a major policy report/insight document, or produce otherwise strategically relevant data.