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Accumulated brisk walking and cardiovascular risk in an 'at risk' population: a preliminary investigation into the novel effects on HDL functionality

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Overweight/obesity now dominate the standard risk factors for cardiovascular disease (CVD)[1]. Regular physical activity, even without weight loss, is associated with reduced CVD-risk. It is recommended that adults undertake at least 150 min of moderate intensity activity per week, which can be accumulated in 10 min bouts. Emerging evidence suggests that high density lipoproteins (HDL) can become dysfunctional in the presence of inflammation, reducing its ability to protect against CVD. The inflammatory molecule serum amyloid A (SAA) displaces apolipoprotein AI from the HDL molecule, which may reduce LCAT activity, thus influencing reverse cholesterol transport. The effects of accumulated moderate intensity exercise (e.g. brisk walking) on HDL functionality in the absence of weight loss has yet to be determined in a population 'at risk' of CVD. This preliminary study investigated whether accumulated brisk walking in overweight/obese subjects (i.e. a group at risk of CVD) without weight loss could influence functional aspects of HDL.

77 overweight/obese sedentary individuals [19M, 58 F; age 45·6 (±6·55) years; BMI 29·2 (±4·27) kg/m^2] were randomly allocated to one of three groups; two groups completed 30 min of accumulated walking for 6 months with either weekly or monthly telephone support; the third group (control) performed stretching exercises. The walking groups were combined and telephone support included as a covariate. As previously reported, following 6 months of brisk walking, arterial stiffness was reduced in the walking groups along with an increase in fitness, effects which were sustained 4 months post-intervention[2]. These changes were observed despite no change in BMI, body fatness, blood pressure or fasting blood lipids (total cholesterol, HDL cholesterol, LDL cholesterol and triglycerides) across the study period[2].To investigate the potential effects of accumulated brisk walking on functional aspects of HDL, HDL_2 and HDL_3 were isolated from fasting serum by rapid ultracentrifugation[3]. SAA (by ELISA),activities of HDL associated enzymes; LCAT and CETP (fluorometric assays) and PON-1 (spectrophotometric assay) were assessed in both fasting serum and isolated HDL sub-fractions. The lipid content of HDL_2 and HDL_3 was assessed spectrophotometrically. Only individuals with a complete dataset were included in analysis (Control group n19: Walking group n40). Data were analysed using repeated measures two-way analysis of variance (ANOVA) with $P < 0{\cdot}05$ considered significant. No significant changes were observed for the lipid profile of HDL_2 and HDL_3 across the study period (all $P > 0{\cdot}05$). Similarly, no significant change in the inflammatory marker SAA or in HDL-associated enzyme activities of CETP or PON-1were identified (all $P > 0{\cdot}05$). A trend towards increased serum LCAT activity was observed in the walking group, although this did not reach significance and was not observed in HDL_2 and HDL_3 (see table).

LCAT Activity	Serum (390:470 nm)					HDL_2 (µratio/mg protein)					HDL_3 (µratio/mg protein)				
	Control		Walking			Control		Walking			Control		Walking		
	Mean	SD	Mean	SD	P*	Mean	SD	Mean	SD	P*	Mean	SD	Mean	SD	P*
Baseline	0·9	0·18	0·9	0·38[a]	0·98	32·0	12·39	28·9	14·58	0·25	0·2	12·39	0·2	0·08	0·52
End	1·0	0·22	1·0	0·42[a]	0·80	31·5	8·34	29·6	13·59	0·25	0·3	8·34	0·3	0·09	0·75
Post	1·0	0·15	1·1	0·52[b]	0·05	32·7	11·33	29·5	14·21	0·19	0·3	11·33	0·3	0·11	0·28
P^+	0·69		0·06			0·93		0·91			0·16		0·19		

Values are mean (SD) at baseline, end of intervention (end) and 4 months post-intervention (post)for control group (n19) and walking group (n40).*P (time), +P(within groups). Different superscripts indicate $P < 0{\cdot}05$ between time points.

Retrospective power calculations based on this preliminary data indicate that 100 participants would be required to elicit a significant change to these measures of functional aspects of HDL, and future appropriately powered studies are needed to determine whether accumulated exercise in the absence of weight loss impacts on the antiatherogenic properties of HDL.

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