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Relationship between long-chain omega-3 polyunsaturated fatty acid intake and ankle brachial index, pulse wave velocity and resting heart rate in a sample of overweight adults: A secondary analysis of baseline data in the HealthTrack study

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Abstract

Aim: The present study aimed to explore the association between dietary long-chain omega-3 polyunsaturated fatty acid (LCn3PUFA) intake and cardiovascular risk indicators (ankle brachial index, resting heart rate and brachial-ankle pulse wave velocity) in a clinical sample of overweight and obese participants volunteering for a weight loss trial.

Methods: This was a secondary analysis of baseline data from the HealthTrack study (n = 351). LCn3PUFA intake was calculated via a diet history and the association with ankle brachial index, resting heart rate and brachio-ankle pulse wave velocity was explored using linear regression after controlling for covariates.

Results: LCn3PUFA intake was inversely associated with ankle brachial index (R^2change = 0.021, F change (1, 339) = 8.864, P < 0.05) and resting heart rate (R^2change = 0.014, F change (1, 342) = 5.337, P < 0.05) but not with brachio-ankle pulse wave velocity (R^2change = 0.001, F change (1, 339) = 0.725, P > 0.05).

Conclusions: In this clinical sample of overweight adults, LCn3PUFA consumption was significantly associated with a lower resting heart rate, adding to the current evidence on the potential benefits of LCn3PUFA consumption. It also supports the value of targeting a diet rich in this nutrient when planning future dietetic approaches. Relationships with ankle brachial index and pulse wave velocity require further investigation. Future research should assess the effect of changes in dietary LCn3PUFA intake on novel cardiovascular risk indicators.

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Methods: This was a secondary analysis of baseline data from the HealthTrack study (n=351). Long chain omega-3 polyunsaturated fatty acid intake was calculated via a diet history and the association with ankle brachial index, resting heart rate, and brachio-ankle pulse wave velocity was explored using linear regression after controlling for covariates.

Results: Long chain omega-3 polyunsaturated fatty acid intake was inversely associated with ankle brachial index (R square change=0.021, F change (1, 339)=8.864, $p<0.05$) and resting heart rate (R square change=0.014, F change (1, 342)=5.337, $p<0.05$) but not with brachio-ankle pulse wave velocity (R square change=0.001, F change (1, 339)=0.725, $p>0.05$).

Conclusion: In this clinical sample of overweight adults, long chain omega-3 polyunsaturated fatty acid consumption was significantly associated with a lower resting heart rate, adding to the current evidence on the potential benefits of long chain omega-3 polyunsaturated fatty acid consumption. It also supports the value of targeting a diet rich in this nutrient when planning future dietetic approaches. Relationships with ankle brachial index and pulse wave velocity require further investigation. Future research should assess the effect of changes in dietary long chain omega-3 polyunsaturated fatty acid intake on novel cardiovascular risk indicators.

Keywords:

Ankle-brachial index

Cardiovascular risk factors

Diet history

LCn3PUFA

Pulse wave velocity

Resting heart rate

Introduction

Cardiovascular disease (CVD) is considered to be a global health concern, responsible for 17.7 million deaths, which represented 31% of global deaths in 2015.[1] Exploring novel, non-invasive physiological risk factors for CVD provides insight into disease risk and progression and can be used to explore the effect of lifestyle modifications on CVD risk. Heart rate (HR), arterial stiffness, and peripheral arterial disease (PAD) are now considered to be independently associated with high risk of CVD.[2-14] Epidemiological studies have reported strong, independent, graded correlations between elevated resting HR and CVD.[13, 17] Lower resting HR is associated with a lower CVD risk compared to increased HR.[11, 18] In comparison, arterial stiffness is defined as a reduction of the distending ability of arteries due to pathological changes in the vessel wall. The "gold standard" measurement of arterial stiffness is pulse wave velocity (PWV).[19] Increased stiffness or elevated PWV promotes endothelial damage and increases back-pressure to the left ventricle of the heart, causing left ventricular hypertrophy and coronary ischemia, ultimately resulting in CVD.[19 21] Improving arterial stiffness (i.e. reducing PWV) aids CVD prevention and treatment in clinical practice.[22, 23] Peripheral arterial disease (PAD) is the blockage or narrowing of medium to small arteries supplying limbs, mainly the lower extremities, and is primarily diagnosed by ankle brachial index (ABI) in clinical practice. The main cause of PAD is atherosclerosis.[24-27] Coexisting severe coronary atherosclerosis and similar lesions can be found elsewhere in the arterial system in patients with PAD or low ABI.[2, 3, 5, 28, 29] Investigation of modifiable factors which can impact these risk factors is required.

Dietary modifications may play a role in influencing physiological risk factors for CVD including those described above. The effect of consumption of long chain omega 3 polyunsaturated fatty acids (LCn3PUFA) on CVD has been studied extensively during the last few decades. LCn3PUFAs are a group of fatty acids abundant in oily fish and produced in minute amounts in the human body from desaturation of alpha-linolenic acid, which is an essential fatty acid.[30, 31] Research suggests supplementation of LCn3PUFA may have CVD protective and mortality reduction effects by improving endothelial function, reducing CVD risk factors such as blood pressure, heart rate, and serum triglyceride levels, and reducing ventricular arrhythmias and chronic inflammation.[32-41] There is currently a paucity of evidence on the effects of dietary modification including LCn3PUFA intake on forms of CVD such as PAD. As a result, the body of evidence for the effects of LCn3PUFA consumption on risk factors including ABI remains inconclusive. While previous research has explored the relationship between LCn3PUFA intake and CVD risk factors, there has been a paucity of research investigating this relationship in the clinical context. Exploration of the relationship between consumption of LCn3PUFA and risk factors for CVD in a clinical sample provides an opportunity to investigate the relevance of this relationship in clinical practice. This also provides insight into potential dietetic strategies for improving CVD risk in clinical populations.

This study aimed to explore the association between reported LCn3PUFA intake and cardiovascular risk indicators (ankle brachial index, resting HR and brachial-ankle PWV) in a sample of overweight and obese adults (25-54 years) volunteering for a clinical trial.

Methods

The present study is a secondary analysis of baseline data on participants randomised to the HealthTrack study.[42]The HealthTrack study was a 12-month randomised controlled trial conducted in the Illawarra region, 70km south of Sydney, Australia. Study subjects were overweight or obese (body mass index (BMI) 25 to 40 kg /m^2) adults aged between 25 – 54 years. The HealthTrack study exclusion criteria included being unable to communicate in English; severe medical

conditions which impaired the ability to participate in the study; immune deficiencies; survival from illnesses predicted to be less than 1 year; reported illegal drug use; regular alcohol intake associated with alcoholism (>50g/day), or having difficulties or hindrances in participating for study components. From recruitment, 377 participants were randomised for baseline analysis, intervention and follow-up. Randomised participants were grouped into three arms to examine the interdisciplinary approach of weight reduction with usual care. The primary outcome was weight and secondary outcomes included disease risk factors such as fasting blood lipids, glucose, HbA1c, systolic blood pressure and behaviour (diet, activity, and psychological factors).[42]

Ethical approval was obtained from the '[removed for blind peer review]' and the study was registered with the '[removed for blind peer review]'.

All physiological data were collected in a laboratory which was calm and quiet to minimise external stimulation. Participants were not fasted prior to the collection of physiological data. Resting HR, brachial-ankle PWV (baPWV) and ABI data were measured using an Omron BP-203RPEIII VP-1000 device (Omron Health Care, Kyoto, Japan) and cleaned using American Heart Association guidelines.[41] Measurements were taken following a 5-minute resting period in the supine position. Two measurements were taken and the second was used as the actual measurement for the study. Blood pressure taken at the same time as the ABI measurement was utilised for the calculation of mean arterial blood pressure (MBP). The following equation was used to calculate MBP as a covariate for baPWV.[44, 45]

$$MBP = [\text{Systolic pressure} + 2\ (\text{diastolic pressure})]/3$$

PWV was cleaned according to European Society of Cardiology guidelines.[44]

Dietary intake data was collected using diet history interviews (DH) conducted by a team of Accredited Practising Dietitians (APD), using a validated interview protocol[46], with support from food models and household measures. Dietary data was entered into FoodWorks nutrient analysis software (version 7.0, 2012 Xyris Software, Highgate Hill, QLD, Australia) using AUSNUT 2007.[47] Where a food item was not found in the AUSNUT 2007 database, an appropriate

substitution was made, or if possible, a new product was created using label data. Where substitutions were required, a log of substituted products was kept to improve reliability, and all dietary data was checked by an independent researcher. Dietary intake of LCn3PUFA was then calculated. We have previously found reported intake of LCn3PUFA collected using this method to be associated with objective measures of LCn3PUFA intake.[48] LCn3PUFA intake was compared to the National Health and Medical Research Council (NHMRC) Nutrient Reference Values Suggested Dietary Target (SDT) (males: 610mg; females: 430mg per day.[49] Detailed data on quantity and frequency of LCn3PUFA supplement consumption was not available, however during the baseline assessment participants reported if they took supplements, and this data was used in the current analysis. Body weight (kg) and height were measured at baseline to determine BMI. Weight was measured with participants in an upright position, with no shoes and minimal clothing (Tanita TBF-662, Wedderburn Pty Ltd, Ingleburn, NSW, Australia), with the height measured using a stadiometer.

During baseline screening, participants reported whether they had previously been diagnosed by a doctor with type 2 diabetes, cardiovascular disease, or hypertension. Fasting blood samples were collected by a registered pathological service (Southern IML Pathology) at baseline. Cholesterol / HDL ratio data was utilised in the current study because this measure has the greater predictive ability of atherosclerotic vascular disease than other blood cholesterol measurements and is less modified by LCn3PUFA intake.[50, 51]

The International Physical Activity Questionnaire (IPAQ) short form, a validated assessment tool for use in the Australian community, was used to assess participant's physical activity.[52]

Statistical analysis was conducted using SPSS (version 22.0, IBM Corp, 2013, New York). The distribution of all continuous data was explored for normality (Kolmogorov-Smirnov, Shapiro-Wilk and graph) and log-transformed if found to be non-parametric (LCn3PUFA - FR and DH, baPWV, ABI, resting HR, Cholesterol / HDL ratio and total energy). Data which could not be transformed (age) were categorised into groups. All categorical data were arranged into binominal groups.

Descriptive statistics of central tendency were calculated for all parameters. Results were presented with mean and standard deviation if variables were continuous and parametric, with median and interquartile range (25^{th} and 75^{th} percentile) reported if continuous variables were nonparametric. Categorical variables were presented as percentages.

Hierarchical linear regression was used to determine whether LCn3PUFA intake predicted the variability of ABI, resting HR, and baPWV when covariates (MBP, HR, Cholesterol / HDL Ratio, age, gender, BMI, whether participants reported taking fish oil supplements, total energy intake and CVD related comorbidities such as heart disease, hypertension and diabetes mellitus) were controlled. As the accuracy of baPWV may be reduced in the case of lower limb artery stenosis [53, 54], the analysis between LCn3PUFA and baPWV was repeated with participants with ABI <0.9 excluded. Preliminary analysis were conducted to detect violations of normality, linearity, multicollinearity and homoscedasticity, with no violations of assumptions found.

To further explore the relationship between LCn3PUFA intake and HR, participants were categorised as those with a HR below 69 beats per minutes (<69bpm) and those with a HR of 69 beats per minutes (69bpm) or above (>=69 bpm). These cut-offs were selected based on the findings of a previous meta-analysis which observed greater effects of fish oil on HR in populations with a mean baseline HR of 69bpm or greater.[55] An independent sample T-test was used to compare intake of LCn3PUFA (transformed) between HR groups. The two tailed *p* value of <0.05 was taken as statistically significant for all analyses.

Results

Table 1 summarises characteristics of study participants at baseline. The study sample for this analysis was n=351 participants (Figure 1). A total of 24.9% of participants reported consuming LCn3PUFA above the SDT.

Covariates were entered in step 1 of the hierarchical linear regression to compare LCn3PUFA intake and ABI, explaining 40.1% of the variability in ABI. In step 2 after entering LCn3PUFA intake, 42.6% of the variance was explained ($F(12, 339)=6.277$, $p<0.05$). LCn3PUFA intake

explained an additional 3.6% of the variance in ABI, after controlling for other variables (R square change=0.021, F change (1, 339)=8.864, p<0.05). In the final model, LCn3PUFA intake was statistically significant, with a low beta value (beta=-0.036, p<0.05) (Table 2).

During analysis of LCn3PUFA intake and baPWV, covariates were entered in step 1, explaining 73% of the variability in baPWV. In step 2 after entering LCn3PUFA intake, there was no change of variance (F (12, 339)=32.190, p<0.05) after controlling for other variables (R square change=0.001, F change (1, 339)=0.725, p>0.05). In the final model, LCn3PUFA intake was not statistically significant, with a low beta value (beta=-0.006, p>0.05) (Table 2). Exclusion of participants with ABI <0.9 from this analysis did not change the relationship observed (beta=-0.007, p>0.05).

While analysing variability of LCn3PUFA intake and HR**,** all covariates were entered in step 1, explaining 35.1% of the variability in HR. After entering LCn3PUFA intake in step 2 explained variance was 37% (F (9, 342)=5.341, p<0.05). Adding LCn3PUFA intake explained an extra 2.1% variance in HR, after controlling for other variables (R square change=0.014, F change (1, 342)=5.337, p<0.05). LCn3PUFA intake was statistically significant, with a low beta value (beta=-0.021, p<0.05) (Table 2).

The independent-samples t-test indicated that participants with a HR of 69 bpm or higher had significantly lower intakes of LCn3PUFA than those with a HR less than 69 bpm (t [349] = -2.471, p=0.014, two-tailed) (Table 3). The magnitude of the differences in the means (mean difference = -0.1, 95% CI: –0.18 to -0.02) was very small (eta squared = 0.017).

Discussion

In this secondary analysis of baseline data on overweight and obese individuals from a clinical trial, LCn3PUFA intake was inversely associated with ABI and resting HR. This finding confirms that the favourable relationship between LCn3PUFA and resting HR observed in previous research[55, 56-] can also be observed in the clinical setting. The relationship between ABI and LCn3PUFA observed in this study should be interpreted with caution. While there is evidence suggesting that lower ABI

may indicate higher risk of cardiovascular disease [2, 5], low ABI in the younger participants in this study may not reflect lower extremity arterial disease. Furthermore, there was no association observed in this study between reported LCn3PUFA and baPWV.

While the relationship between LCn3PUFA and ABI should be interpreted with caution, there are a number of mechanisms by which LCn3PUFA consumption may be associated with reduced ABI and HR. LCn3PUFA intake may influence ABI by reducing inflammatory cytokine production through incorporation into the cell membrane.[57, 58] Furthermore, LCn3PUFA derived EPA has been found to improve endothelial function via nitrous oxide-dependent vascular relaxation and DHA modifies lipid composition and the structure of the vessel wall by altering adhesion molecules, ultimately improving endothelial and vessel compliance.[58] It is possible that LCn3PUFA consumption may reduce ABI by means of inflammatory reduction and modification of endothelial and vascular function.

Findings of animal studies have suggested LCn3PUFA consumption may alter the automaticity of heart muscles cells similar to class 1 antiarrhythmic medications.[59] LCn3PUFAs can alter the resting membrane potential of heart muscle cells, predominantly the SA node, by direct action on the cell membrane. This effect can increase membrane threshold resulting in a delay in the next autogenerated impulse, leading to a reduction of resting HR.[59] Increased consumption of LCn3PUFA via fish oil was associated with a reduction of HR by 2.5 beats per minutes (bpm) in individuals with a baseline HR of 69 beats per minute or higher which is associated with reduced cardiac morbidity.[55] This observation may be suggestive of a cardiac protective effect of LCn3PUFA consumption via reducing arrhythmogenicity and improved reserved capacity. However, in this study, the magnitude of the association between LCn3PUFA intake and HR was very small. The median intake, even in the group with a resting heart rate <69 beats per minute, was still below the prefereable 250 mg per day (EPA +DHA) supported by longitudinal evidence for cardiac benefits [60] In our study population, there were other significantly associated risk factors

contributing to resting HR such as age and BMI (Table 2), however, these results suggest that LCn3PUFA intake are a potential target for dietary modification within clinical practice.

Given the association between overweight and obesity and PWV[61], it is relevant to explore dietary components associated with improved PWV in this at risk population, in order to identify potential dietetic strategies for reducing cardiovascular risk. In contrast to the findings for ABI and HR, we did not observe an association between LCn3PUFA consumption and baPWV in this setting. This finding does not align with those of previous studies using LCn3PUFA supplements with therapeutic doses.[62, 63] The disparity in findings may be explained by variations in the study population, the amount of LCn3PUFA intake and dietary assessment methods used. For example, age is a well known major determinant of vascular stiffness, which increases significantly after the age of 55.[64, 65] The median age in the current study group was 45 years. Our population may be too young to demonstrate a significant association between vascular stiffness (baPWV) and higher LCn3PUFA consumption. Furthermore, baPWV was used to measure vascular stiffness in the current study. However, cfPWV is the gold standard measurement of aortic stiffness and is considered to be a prognostic indicator of CVD risk,[7, 10, 66, 67] with baPWV validated as a cardiovascular risk factor in Asian communities only. This may be another reason for the disparity in results between studies as they used inconsistent PWV measuring methods. Whilst some research suggest cfPWV and baPWV may similarly predict CVD risk,[68] baPWV results should be generalised to European communities with caution.

Previous studies and reviews have demonstrated therapeutic effects on the endothelial and vascular system in different doses of LCn3PUFA supplements, between 0.45 to 3g/day.[65, 69-72] However, the cardiac effects of LCn3PUFA are evident at lower doses such as 1g/day or less.[37, 60, 71, 73] In the current analysis resting HR was significantly associated with LCn3PUFA at an even lower consumption level. Importantly, this relationship was observed at intake levels associated with moderate consumption of dietary sources of LCn3PUFA. In contrast however, in the current study, LCn3PUFA intake may not have been sufficient to be associated with a lower baPWV. Reseach

findings provide insight into potential dietetic strategies for improving cardiovascular risk. These findings further appear to be reflective of the inconclusive nature of the body of evidence surrounding the impact of LCn3PUFA on cardiovascular outcomes more broadly, as highlighted by a recent systematic review and meta-analysis on the impact of LCn3PUFA supplements on coronary heart disease.[74] Though there are beneficial cardiovascular effects obsevered in therapeutic doses of LCn3PUFA, further research is needed exploring the impact of LCn3PUFA from dietary sources on cardiovascular measures specifically vascular indicators such as baPWV and ABI. The current study had some limitations which may have affected the results. This study was a baseline secondary analysis of data from the HealthTrack study, which was not designed to assess LCn3PUFA intake and cardiovascular outcomes. As such the HealthTrack study was not powered to address this specific question, which may have affected our results. As this study utilised baseline data, it was a cross-sectional analysis and therefore cannot draw conclusions regarding causation. The DH used was not standardised for LCn3PUFA intake assessment and there was no objective measure of LCn3PUFA intake available such as erythrocyte LCn3PUFA levels. Estimation of LCn3PUFA intake may have also been limited by the availability of food products within AUSNUT 2007. Furthermore, this study was not able to quantify the LCn3PUFA supplement intake by the study population, which has been suggested to play a major role in Australians achieving the SDT for LCn3PUFA.[47] However, whether participants reported taking LCn3PUFA or fish oil supplements was included as a covariate during the analysis to alleviate this limitation. The HealthTrack study used AUSNUT 2007, the most recent food composition database available at the beginning of the study; however AUSNUT 2007 only reports total LCn3PUFA rather than EPA and DHA separately. This may limit comparisons with LCn3PUFA studies in the literature.[75-78] Measurement of baPWV and ABI did not follow standard operational procedures published by the American Heart Association for vascular research,[65] and the device used to measure baPWV and ABI was only standardised for the Japanese population.[65] However, the HealthTrack study was designed to be aligned with clinical practice, and thus may correspond with methods used in the

clinical setting. Finally, the HealthTrack study involved overweight and obese self-selected volunteers from regional New South Wales, therefore results may not be generalisable to the broader population.

This secondary analysis of baseline data from a weight loss trial confirms that the favourable relationship between LCn3PUFA intake and CVD risk factorHR can also be observed in the clinical setting. In contrast, relationships with ankle brachial index and pulse wave velocity require further investigation. These results add to the current evidence surrounding the potential benefits of LCn3PUFA consumption and highlight the importance of targeting food sources of this nutrient in clinical dietetic practice.

Given the findings of this cross-sectional analysis, it will be beneficial to explore these results further in randomised controlled trials to assess the effect of changes in dietary LCn3PUFA intake on novel cardiovascular risk indicators.

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Figures:

Figure 1 Flow diagram for baseline data

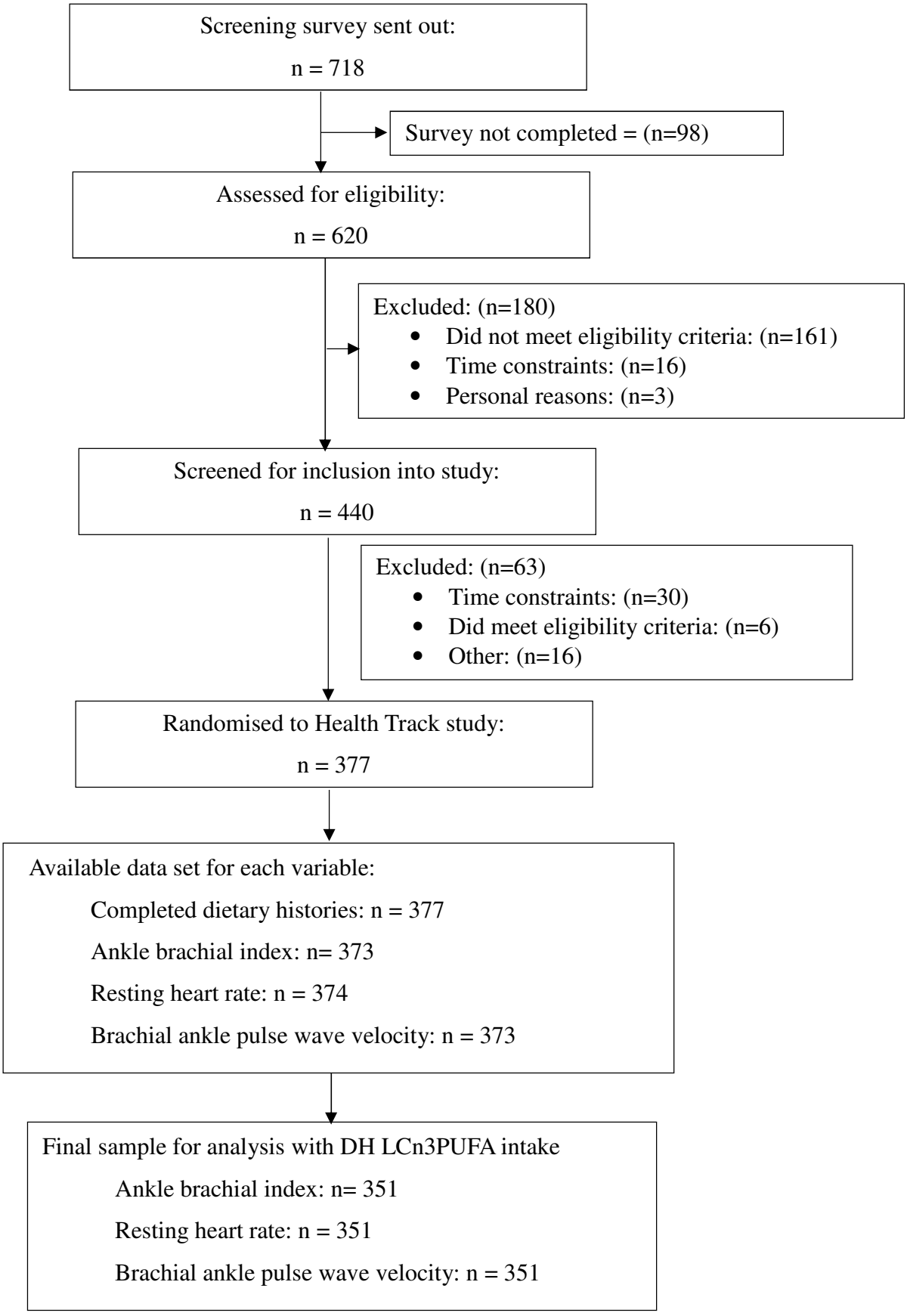

Figure 1 Flow diagram for baseline data analysis

515 **Table**

516 **Table 1** Characteristics of the study participants at baseline

517 **Table 2** Regression analysis summary table of ABI[(a)], HR[(a)] and PWV[(a)]

518 **Table 3.** LCn3PUFA[(a)] intake between HR[(a)] categories

519 **Table 1** Characteristics of the study participants at baseline

Variables	Subsample for analysis			
	N	%	Median	IQR
Age, (years)	351		45	37 – 51
Gender	351			
Male	92	26.3		
Female	259	73.7		
BMI[(a)], (kg/m^2)	351		32.1	29.3 – 35.7
Self-reported medical history of baseline survey	351			
Comorbidities (CVD-related)	104	29.4		
Heart disease	4	1.1		
Diabetes mellitus	21	5.9		
Hypertension	93	26.3		
Fish oil supplements	351			
Taking supplements	20	5.7		
Not taking supplements	331	94.3		
MBP[(a)], (mmHg)	351		90.67	82 – 97.33
Cholesterol / HDL[(a)] ratio	351		3.6	3 – 4.4
IPAQ (MET mins/week)[(a)]	351		984	466.5 - 1751
Total energy intake, (kJ)	351		9098	7476 – 11187
LCn3PUFA consumption, (mg)	351		287.6	159.7 – 518.4
ABI[(a)]	351		1.06	1.01 – 1.12
baPWV[(a)], (cm/s)	351		1180	1084 – 1313.5
HR[(a)], (bpm)	351		65	59 - 72

520 [(a)]Abbreviations: IQR: interquartile range, BMI: Body mass index; CVD: Cardiovascular disease; MBP: Mean blood pressure; HDL: High-density lipoproteins; IPAQ: International
521 physical activity questionnaire; LCn3PUFA: Long chain omega 3 polyunsaturated fatty acid; ABI: Ankle brachial index; baPWV: Brachial-ankle pulse wave velocity; HR: Heart
522 rate.

523 **Table 2** Regression analysis summary table of ABI, HR and PWV

		ABI (n=351)			**HR (n=351)**			**baPWV (n=351)**		
		B	SE B	β	B	SE B	β	B	SE B	β
Step 1	(Constant)	1.897*	.212		1.361*	.137		1.560*	.146	
	Age 20 to 30	-.029*	.016	-.103	.029*	.012	.139	-.057*	.009	-.268
	Age 30 to 40	-.008	.011	-.039	.006	.009	.045	-.033*	.007	-.225
	Age 50 to 60	.010	.011	.056	-.004	.008	-.032	.027*	.006	.204
	Log Energy	-.004	.036	-.006	.018	.027	.036	.035	.020	.070
	Log BMI[(a)]	-.084	.082	-.055	.258*	.058	.233	-.050	.048	-.044
	Log Cholesterol/HDL[(a)] ratio	.026	.036	.040				.028	.021	.057
	Gender (Male)	.041*	.011	.213	-.006	.008	-.040	.012	.006	.080
	Comorbidities	.004	.010	.023	.027*	.007	.195	.013*	.006	.092
	Log MBP[(a)]							.549*	.053	.461
	Log HR[(a)]	-.392*	.074	-.286				.221*	.042	.214
	Log IPAQ[(a)]	.004	.005	.040	-.006	.004	-.083	.003	.003	.039
	Supplements	.015	.017	.045	.007	.012	.030	.012	.009	.046
Step 2	(Constant)	1.974*	.211		1.392*	.137		1.573*	.147	
	Age 20 to 30	-.031*	.015	-.111	.026*	.012	.125	-.058*	.009	-.272
	Age 30 to 40	-.010	.011	-.052	.001	.008	.005	-.034*	.007	-.231
	Age 50 to 60	.013	.011	.069	-.003	.009	-.024	.028*	.006	.207
	Log Energy	.005	.036	.008	.022	.027	.045	.037	.020	.073
	Log BMI[(a)]	-.069	.081	-.045	.259*	.058	.234	-.048	.048	-.042
	Log Cholesterol/HDL[(a)] ratio	.011	.036	-.017				.026	.021	.053
	Gender (Male)	.044*	.011	.229	-.004	.008	-.031	.012	.006	.083
	Comorbidities	.002	.010	.013	.025*	.007	.184	.013*	.006	.090
	Log MBP[(a)]							.548*	.053	.461
	Log HR[(a)]	-.415	.073	-.303				.217*	.042	.211

	Log IPAQ[(a)]	.003	.005	.031	-.005	.004	-.073	.003	.003	.042
	Supplements	.017	.017	.051	.008	.012	.034	.012	.009	.047
	Log LCn3PUFA[(a)]	-.036*	.012	-.152	-.021*	.009	-.120	-.006	.007	-.033

[(a)]Abbreviations: Log: logarithmic; BMI: Body mass index; MBP: Mean blood pressure; HDL: High-density lipoproteins; IPAQ: International physical activity questionnaire; LCn3PUFA: Long chain omega 3 polyunsaturated fatty acid; ABI: Ankle brachial index; baPWV: Brachial-ankle pulse wave velocity; HR: Heart rate.

** p<0.05 (Significantly associated)*

Table3. LCn3PUFA[(a)] intake between HR[(a)] categories

Pulse category	**Median intake (mg/dl)**	**IQR[(a)] (mg/dl)**
HR[(a)] less than 69bpm	228	138-455.23
HR[(a)] of 69 bpm or higher	176.90	581.59

[(a)]Abbreviations: LCn3PUFA: Long chain omega 3 polyunsaturated fatty acid; HR: Heart rate; IQR: Interquartile range; SD: Standard deviation

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