

## Encouraging effects of a short-term, adapted Nordic diet intervention on skin microvascular function and skin oxygen tension in younger and older adults

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1 **Title: Encouraging effects of a short-term, adapted Nordic diet intervention on skin**  
2 **microvascular function and skin oxygen tension in younger and older adults**

3

4 **Running Head: Adapted Nordic diet and microvascular function**

5

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14

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16 MK, SM and DR analysed and interpreted the data and wrote the manuscript. All authors  
17 read and approved the final manuscript.

18

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20

21 **Tables**

22 Table 1: Nordic Foods

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28

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30 Figure 1:  $\Delta$ TcPO<sub>2</sub> pre and post intervention

31

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37

38

## Highlights

- 39 • Effects of a 4-week adapted Nordic diet on microvascular function in younger and  
40 older adults were assessed
- 41 • Laser Doppler Flowmetry measured cutaneous microvascular functioning
- 42 • Transcutaneous Oxygen monitoring measured skin oxygen tension
- 43 • Health markers were investigated concurrently
- 44 • Microvascular health, body-fat % and peak heart rate during exercise were improved  
45 followed the diet.

46

## Abstract

47 *Objective:* Microvascular benefits of regional diets are appearing in the literature however  
48 little is known about Nordic-type diets. We investigated the effects of short-term adapted  
49 Nordic diet on microvascular function in younger and older individuals at rest and during  
50 activity.

51 *Research Method & Procedures:* Thirteen young [Mean: 28, SD: (5)] and fifteen older  
52 participants [Mean: 68, SD: (6)] consumed a modified Nordic diet for four weeks. Laser  
53 Doppler Flowmetry and Transcutaneous oxygen monitoring assessed cutaneous  
54 microvascular function and oxygen tension pre and post-intervention; blood pressure, body  
55 mass, body-fat%, ratings of perceived exertion and peak heart rate during activity were  
56 examined concurrently.

57 *Results:* Axon-mediated vasodilation improved in older participants [1.17 (0.30) to 1.30  
58 (0.30);  $P < 0.05$ ]. Improvements in endothelium-dependent vasodilation were noted in young  
59 [1.67 (0.50) to 2.03 (0.62);  $P < 0.05$ ] and older participants [1.49 (0.37) to 1.63 (0.39);  $P <$   
60 0.05]. Reduced peak heart rate during activity was noted in older participants only [36.5(8.9)  
61 to 35.3(8.5);  $P < 0.05$ ] and reduced body-fat % in young participants only [young = 27.2  
62 (8.3) to 25.2 (8.8);  $P < 0.05$ ]. No other variables reached statistical significance however  
63 trends were observed.

64 *Conclusions:* We observed statistically-significant improvements in microvascular function,  
65 peak heart rate and body composition. Following an adapted Nordic diet might improve  
66 microvascular health.

67

## Keywords

68 Nordic Diet; Laser Doppler Flowmetry; Oxygen Tension

## Introduction

70 Cardiovascular disease (CVD) is the number one cause of death worldwide with 17.5 million  
71 deaths reported in 2012 (WHO, 2016). Risk factors for developing CVD include  
72 inflammatory diseases such as type II diabetes and hypertension; aging, gender and lifestyle  
73 factors such as smoking and poor nutrition (WHO 2016). Endothelial dysfunction, a  
74 pathological condition characterised by impaired vasodilation and systemic inflammation  
75 (Hadi et al. 2007), is a precursor of acute coronary syndromes, atherosclerosis and CVD  
76 (Deanfield et al. 2007). Endothelial dysfunction however appears to be reversible and  
77 endothelial health can be improved by modifying cardiovascular risk factors (Hadi et al.  
78 2005). Emerging literature has therefore sought to investigate the effects of lifestyle  
79 modifications as possible treatment strategies (Klonizakis et al. 2013) and dietary  
80 intervention is one lifestyle modification that appears to be promising (Nordman et al. 2011).

81 Dietary interventions, however, are difficult to sustain, and factors such as taste preferences,  
82 culinary habits and social acceptability might contribute to poor long-term adherence  
83 (Poulsen et al. 2015). Bere and Brug (2009) recommend that strategies tailored to regional  
84 eating preferences might lead to better long-term success, and interestingly, data is beginning  
85 to suggest that regional diets might offer health benefits. Indeed, evidence now suggests that  
86 the Mediterranean diet can reduce CVD\* risk (Nordmann et al. 2011), alleviate metabolic  
87 syndrome (Kastorini et al. 2011), reduce blood pressure and enhance weight loss (Esposito et  
88 al. 2011).

89 The Nordic diet is a regional diet that encourages the consumption of Nordic vegetables and  
90 fruits as well as whole grains, fish, rapeseed oil and low-fat dairy products. Early data

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\* **Abbreviations:** CVD, Cardiovascular Disease; NND, New Nordic Diet; LDF, Laser Doppler Flowmetry; TcPO<sub>2</sub>: Transcutaneous Oxygen monitoring; BMI, Body Mass Index; CVC, Cutaneous Vascular Conductance; RPE, Rating of Perceived Exertion; SD, Standard Deviation; NO, Nitric Oxide; MD, Mediterranean Diet; PUFA, Polyunsaturated Fatty Acids

91 suggests that this diet might lead to reduced inflammation (Kanerva et al. 2014a), improved  
92 insulin metabolism (De Mello et al. 2011) and weight loss (Poulsen et al. 2015).  
93 Cardiovascular-health benefits of the diet are also now beginning to appear in the literature:  
94 Adamsson et al. (2011) demonstrated that a 10-week intervention led to lower cholesterol,  
95 reduced blood pressure and decreased serum insulin in hypercholesterolaemic participants.  
96 To date, however, microvascular health effects of Nordic diets have yet to be explored. The  
97 integrity of the microcirculation to sustain blood flow, tissue oxygenation and nutrient  
98 delivery affects susceptibility to disease, and appears to decline with age (Tew et al. 2010).  
99 Identifying strategies that maintain or improve microvascular function are therefore important  
100 for sustaining long-term health.

101 The aim of this study was to investigate the effects of a short-term, adapted Nordic diet  
102 (AND), modified for British taste preferences, on the microvasculature, by assessing tissue  
103 oxygenation and endothelial function. The circulatory system functions differently at rest  
104 and during activity (Abraham et al. 2003) and age-related endothelial dysfunction,  
105 characterised by diminished arterial vasodilation and reduced nitric oxide supply, has been  
106 observed in older adults (Gates et al. 2009). We therefore compared the effects of the diet in  
107 younger (18-35 years old) and older sedentary participants (55-75 years old) at rest and  
108 during sub-maximal exercise. We hypothesised that the intervention would improve  
109 microvascular health and endothelial function in both groups, with older participants  
110 experiencing greater improvements.

111

## Material and Methods

### 112 *Ethical Approval*

113 Ethical approval for this research was granted by the Sheffield Hallam University's Health  
114 and Wellbeing Research Ethics Committee. This research was conducted in accordance with  
115 the Declaration of Helsinki.

### 116 *Participants*

117 Sixteen young participants aged 18-35 years [M = 28(5)] and sixteen older participants aged  
118 55-75 years [M = 64(6)] provided informed consent. Recruitment took place via posters,  
119 word of mouth and through the emailing systems of Sheffield Hallam University and the  
120 University of Sheffield. Participants' eligibility was assessed pre-intervention using physical  
121 activity and nutrition questionnaires. The long International Physical Activity Questionnaire  
122 (IPAQ) was used to assess physical activity; scores > 3000 MET minutes per week would  
123 necessitate participants' exclusion due to non-sedentariness. A validated Nordic Diet Score  
124 (NDS) questionnaire (Bjørnara et al. 2015) was used similarly, and participants scoring > 5  
125 points would also need to be excluded. Exclusion criteria also included smoking, pregnancy  
126 and chronic conditions that might affect safe participation.

### 127 *Dietary Intervention*

128 Participants were advised to adhere to Public Health England's portion size guidelines (PHE  
129 2016) but to follow the AND without restricting energy. During initial assessments,  
130 participants were briefed about AND-compliant foods (Table 1), obtained individualised diet  
131 plans, and provided with materials (recipes, etc.) and food items (root vegetables, cruciferous  
132 vegetables, fish, rye bread and apples; enough for 2 weeks) to improve adherence and foster  
133 behaviour change (Michie et al. 2011). Participants were also instructed to complete a 3-day  
134 diet diary pre and post intervention (two assessments); data was inputted into software



135 (Nutritics, Dublin, Ireland) incorporating McCance and Widdowson's UK Composition of  
 136 Food Database (2015) within its databank (Nutritics Ltd product version 1.7, Dublin Ireland),  
 137 for dietary analysis. Kcals, Total Fat, Saturated Fat, Protein, Carbohydrates, Fibre and  
 138 Omega 3 (Total *n*-3) were calculated, to measure dietary changes that might impact  
 139 microvascular function (Calder et al. 2013). Follow-up consultations were conducted via  
 140 telephone and email at weeks one and three to foster support, and a private social media  
 141 group was created to engender social support similarly (Michie et al. 2011). Participants  
 142 were advised to maintain activity as indicated by their pre-intervention IPAQ scores; no  
 143 physical activity intervention was provided.

144 **Table 1** Nordic Foods

Vegetables	Fruit	Fish/Meat	Grains	Other
Cabbages	Blueberries	Game	Wholegrain breads	Dill
Cauliflower	Blackcurrants	Poultry	Rye	Parsley
Broccoli	Redcurrants	Cod	Oats	Chive
Kale	Gooseberries	Salmon	Barley	Legumes
Onions	Apples	Herring		Rapeseed oil
Swede	Pears	Haddock		
Carrots	Plums	Mackerel		
Beetroot		Halibut		
Turnip				
Potatoes				
Parsnips				
Mushrooms				

145

146 ***Protocol***

147 We used Laser Doppler Flowmetry (LDF) and Transcutaneous Oxygen Monitoring (TcP02)  
 148 to assess microvascular function pre and post intervention, reflecting procedures described by

149 Wasilewski, Ubara and Klonizakis (2016). Laser Doppler Flowmetry was used to determine  
150 cutaneous microvascular responsiveness to local heating (Tew et al. 2011); Transcutaneous  
151 Oxygen Monitoring was used to assess tissue oxygen supply (Bajwa et al. 2014). To measure  
152 LDF and TcPO<sub>2</sub> pre and post-intervention, we required participants to attend the laboratory  
153 on two occasions, separated by a four-week intervention period, and instructed them to  
154 abstain from caffeine prior to attending, to eliminate acute vasoconstriction (Umemura et al.  
155 2006). Stature (cm) body mass (kg), body fat % and BMI (kg · m<sup>2</sup>) were measured  
156 concurrently using a segmental body-composition analyser (InBody 720, Derwent  
157 Healthcare; UK) and compared at both time points.

### 158 ***LDF Procedure***

159 Microvascular blood flow was measured as cutaneous red blood cell flux using a Laser  
160 Doppler Flowmeter (Periflux system 5000, Perimed 122 AB, Järfälla; Sweden) and a 7-point  
161 LDF probe (Probe 413, 123 Perimed AB), using procedures outline by Tew et al. (2010).  
162 Participants were acclimated to a temperature-controlled room (ambient temperature set to 22  
163 - 24 °C) before collecting data. Participants' forearms were cleansed prior to attaching the  
164 LDF probe to the skin on the underside of the right arm, avoiding veins and hair, to  
165 circumvent abnormal readings. Local thermal hyperaemia was induced using a heating disk  
166 (Model 455, Perimed AB) connected to a heating unit (Model 5020, Perimed AB) and LDF  
167 signals were recorded using PeriSoft software (PSW 9.0). Baseline blood-flow data were  
168 recorded for five minutes with the local heating disc set to 30 °C. Temperature was then  
169 increased (1° C · 10 s<sup>-1</sup>) to 42 °C to induce rapid local heating, which was then maintained  
170 for 30 minutes. After this, the probe temperature was increased to 44 °C for 10 minutes to  
171 achieve maximal vasodilation. Resting blood pressure (mmHg) and heart rate (bpm) were  
172 recorded at baseline and at every five minutes during data collection using a patient  
173 monitoring device (Dinamap Dash 2500, GE Healthcare; USA). Thermal hyperaemic data

174 were recorded during the test and expressed as cutaneous vascular conductance (CVC) at four  
175 regions (baseline, initial peak, plateau, and maximum regions) and presented as raw CVC and  
176 CVC normalised to maximum (%CVCmax: [(CVC / maximum CVC) x 100]).

### 177 *Transcutaneous Oxygen Measurement*

178 The sub-maximal exercise test (Table 2) was performed after the LDF procedure using a  
179 cycle ergometer (824E, Monark AB; Sweden). Heart Rate (HR) (Sports Tester, Polar;  
180 Finland) and Ratings of Perceived Exertion (RPE; CR10 scale, Borg, 1998) were recorded at  
181 each minute and blood pressure (mmHg) was recorded one minute into every two-minute rest  
182 period using participants' contralateral arm, using the patient monitoring device (Dinamap  
183 Dash 2500, GE Healthcare; USA). Oxygen tension was measured using a calibrated TINA  
184 TCM400 tcpO<sub>2</sub> device (Radiometer; DK) during the test. A temperature probe, set to 44.5 °C  
185 to achieve maximal skin vasodilatation, was attached to the skin of the participants' sub-  
186 scapular area using a fixation ring, which was attached to participants' back approximately 10  
187 mm below the left scapula, avoiding bone, and using contact solution. The solution was  
188 allowed to heat, causing skin dilatation. Dilatation of the skin-blood capillaries increases  
189 blood flow, causing a diffusion of oxygen through the skin into the sensor, which then  
190 measures TcPO<sub>2</sub>. After this, TcPO<sub>2</sub> measurements were temperature corrected to 37 °C by  
191 the TINA device. For the purposes of this study, TcPO<sub>2</sub> was defined as the raw oxygen  
192 perfusion values obtained directly from the TINA recordings (Table 3).

193

194 **Table 2** Submaximal Exercise Protocol

Interval : Time (mins)	Resistance (kg)	Speed (RPM: revolutions per minute	Power output (Watts)
Interval 1 : 5 mins	1kg	80 RPM	80W
Rest : 2 mins	-	-	
Interval 2: 5 mins	1.2kg	80 RPM	96W
Rest : 2 mins	-	-	
Interval 3: 5 mins	1.4kg	80 RPM	112W
Rest : 2 mins	-	-	
Interval 4: 5 mins	1.6kg	80 RPM	128W

195

196 **Table 3** TcPO2 Variables

TcPO2 Quantity	Definition
Baseline	The arithmetic mean of maximum TcPO2 at rest
TcPO2max	The greatest TcPO2 value each minute of exercise or rest.
$\Delta$ TcPO2max	The maximum change from baseline value e.g. TcPO2max – baseline.
$\Delta$ TcPO2	Average sum of change in Transcutaneous oxygen tension from baseline.

197

198 **Statistical Analysis**

199 Independent t-tests were performed on baseline physical characteristic and dietary analysis  
 200 data. A two-by-two mixed design Analysis of Variance (ANOVA) compared the effects of  
 201 the AND intervention on blood pressure (systolic and diastolic), body-mass, body-fat %, peak  
 202 heart rate, RPE,  $\Delta$ TcPO2,  $\Delta$ TcPO2max, CVC, %CVCmax and diet data (NDS, Kcals, Total  
 203 Fat, Saturated Fat, Carbohydrates, Protein, Fibre and Omega 3) in the older and younger  
 204 participants using SPSS (SPSS Inc., Chicago Illinois, version 23 for Windows). The alpha  
 205 level was set at  $P = 0.05$ . To accomplish normality or homogeneity of variance,  $\Delta$ TcPO2,  
 206 Body Fat %, Peak Heart Rate and dietary data (NDS, Kcals, Omega 3) were log transformed

207 prior to inferential analyses, after checking for and ensuring underlying assumptions.

208 Independent and dependent-samples t-tests followed up significant interactions. Data are

209 presented as mean  $\pm$  SD.

210

211

**Results**212 ***Participants***

213 Thirteen young (18 - 35 years) and fifteen older (55 – 75 years) participants completed the  
 214 study from the sixteen young and sixteen older participants originally recruited, equating to  
 215 an 82 % and 94 % completion rate. Participants' characteristics are presented in Table 4.

216 **Table 4** Participants' Characteristics Pre and Post Intervention

	<b>Group A (Young)</b>		<b>Group B (Old)</b>	
	<b>Visit 1</b>	<b>Visit 2</b>	<b>Visit 1</b>	<b>Visit 2</b>
<b>Gender</b>	5 male, 8 female		7 male, 8 female	
<b>Age (years)</b>	28 (5) <sup>†</sup>		64 (6) <sup>†</sup>	
<b>Resting BP (systolic)</b>	129 (10) <sup>†</sup>	123 (9)	150 (14) <sup>†</sup>	148 (19)
<b>Resting BP (diastolic)</b>	78 (15)	72 (8)	81(12)	79 (16)
<b>Stature (cm)</b>	171 (6.0)		168 (6.6)	
<b>Body Mass (kg)</b>	69.1 (22.1)	67.4 (22.1)	81.6(16.8)	80.6 (16.7)
<b>BMI (kg·m<sup>2</sup>)</b>	24.3 (7.9)	23.6 (7.9)	30.5 (5.4)	29.7 (5.4)
<b>Body Fat (%)</b>	27.2 (8.3)	25.2 (8.8)*	36.5(8.9)	35.3(8.5)

217 <sup>†</sup>P = <0.05 between groups (at baseline),

218 \*P = &lt;0.05 between visits (within groups)

219

220 ***Dietary Analysis***

221 Baseline Kcals [young = 1615.2 (645.6), old = 2595.2 (567.3); *P* = 0.14], Total Fat [young =  
 222 61.2 g (22.6), old = 122.0 g (56.4); *P* = 0.03], Saturated Fat [young = 22.0 g (7.3), old = 38.5  
 223 g(13.8); *P* = 0.027] and Fibre [young = 15.4 g (4.9), old = 27.7 g (4.3); *P* = 0.001] were lower  
 224 in the younger participants (Table 6). Post intervention, only Kcals [young = 1353.0 (274.3),  
 225 old = 2042.7 (676.0); *P* = 0.29] and Total Fat [young = 45.5 g (11.6), old = 87.7 g (36.5); *P* =  
 226 0.022] differed between groups. Between visits, NDS [young = 2.5 (0.8) to (5.7 (1.4); *P* =

227 0.01, old = 2.3 (1.2) to 5.2 (0.8);  $P = 0.02$ ] increased in both groups similarly. Fibre intake  
 228 increased in the younger group (15.4 g (4.9) to 24.3 g (3.0);  $P = 0.05$ ). No other dietary data  
 229 reached statistical significance (Table 5).

230 **Table 5** Raw Dietary Data Pre and Post Intervention

	Young		Old	
	Pre-NND	Post- NND	Pre-NND	Post-NND
<b>NND Score</b>	2.5 (0.8)	5.7 (1.4) *	2.3 (1.2)	5.2 (0.8) *
<b>Kcals</b>	1615.2 (645.6) †	1353.0 (274.3)	2595.2 (567.3) †	2042.7 (676.0)
<b>Total Fat</b>	61.2 (22.6) †	45.5 (11.6)	122.0 (56.4) †	87.7 (36.5)
<b>Saturated Fat</b>	22.0 (7.3) †	13.9 (7.3)	38.5 (13.8) †	22.9 (10.8)
<b>CHO</b>	194.8 (99.2)	163.8 (54.8)	237.3 (63.1)	207.3 (102.5)
<b>Protein</b>	80.7 (26.1)	81.2 (13.3)	101.3 (30.5)	92.3 (28.9)
<b>Fibre</b>	15.4 (4.9) †	24.3 (3.0) *	27.7 (4.3) †	25.1 (6.3)
<b>Omega 3</b>	0.5 (0.5)	0.5 (0.3)	3.4 (5.0)	2.4 (3.3)

231 † $P < 0.05$  between groups (at baseline)

232 \* $P < 0.05$  between visits (within groups)

233

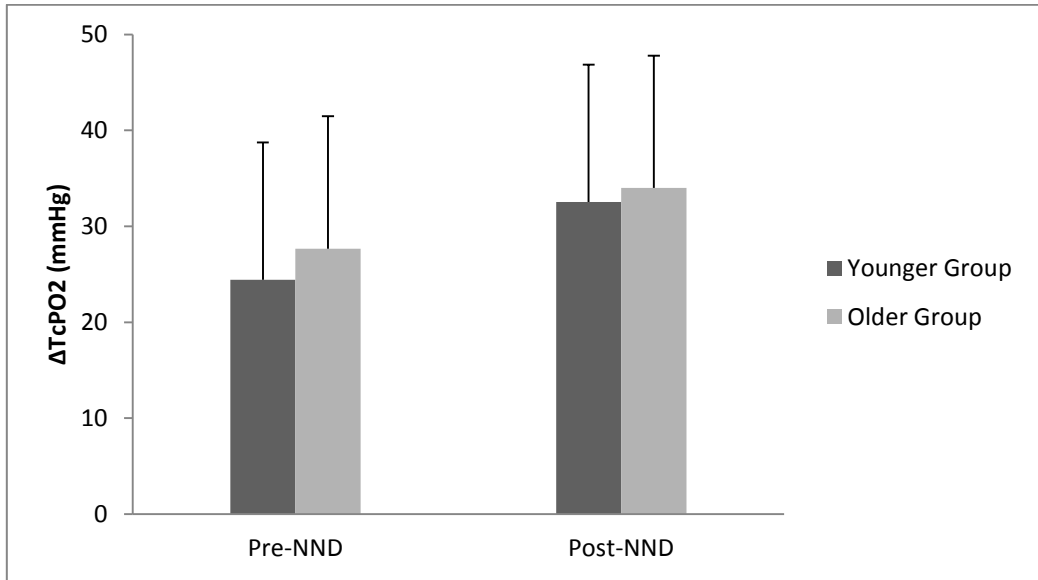
234 ***BMI, Body Mass, Body Fat and Blood Pressure***

235 No differences in BMI or Body Mass were observed in either group at any time and no  
 236 between-groups differences were noted for body fat % pre or post intervention. Only  
 237 younger participants experienced reductions in body fat % between visits [27.2 (8.3) to 25.2  
 238 (8.8);  $P = 0.028$ ] (Table 4). Baseline systolic blood pressure appeared to be lower in the  
 239 younger participants [129 (10) vs. 150 (14);  $P = 0.01$ ] however the AND had no effect on  
 240 systolic blood pressure in either group (Table 4). Further, there were no changes in diastolic  
 241 blood pressure in either group at any time (Table 4).

242 **Oxygen Tension**

243 There were no differences between the groups or changes to any of the TcPO2 variables  
244 measured despite  $\Delta$ TcPO2 appearing to increase post intervention ( $P = 0.26$ ) (Figure 1).

245 **Figure 1**  $\Delta$ TcPO2 Pre and Post Intervention



246

247

248 **Cutaneous Vascular Conductance**

249 **Baseline**

250 **Raw CVC**

251 The younger group experienced no changes to raw CVC however the older group  
252 experienced an improvement during the 2<sup>nd</sup> assessment [0.35 (0.14) to 0.42 (0.16);  $P = 0.02$ ]  
253 (Table 5). Baseline between-groups differences observed for Raw CVC were not present  
254 post-intervention (Table 6).

255 **%CVC MAX**

256 There were no differences between the groups or changes to %CVC MAX in either group at  
257 any time (Table 6).

258

259 **Initial Peak**



260 **Raw CVC**

261 Post-intervention, older participants exhibited lower Raw CVC [1.71 (0.53) vs 1.30 (0.30);  $P$   
262 = 0.01] at the initial peak compared to the young participants despite experiencing an  
263 increase from baseline [1.17 (0.30) to 1.30 (0.30);  $P = 0.01$ ] (Table 6). Pre-intervention  
264 between-groups differences were not apparent post-intervention.

265 **%CVCmax**

266 No changes in % CVCmax were observed in either group at any time (Table 6).

267 **Plateau**

268 **Raw CVC**

269 Both groups experienced improvements in raw CVC at visit 2 (Table 5). No between-groups  
270 differences were noted at the pre intervention stage for Raw CVC; however, between-groups  
271 differences became apparent at the post-intervention period (young = 2.03 (0.62), old = 1.63  
272 (0.39);  $P = 0.03$ ) (Table 6).

273 **%CVCmax**

274 Improvements to %CVCmax were experienced in the younger participants only [78.8 (12.0)  
275 to 85.0 (10.7);  $P = 0.03$ ]. Similar to Raw CVC, no between-groups differences were noted at  
276 the pre-intervention stage but were noted at the post-intervention period [young = 85.0 (10.7),  
277 old = 77.7 (7.3);  $P = 0.03$ ] (Table 6).

278

279 **Table 6** Cutaneous Vascular Conductance Pre and Post Intervention

	Group A (younger group)		Group B (older group)	
	Raw CVC	% CVC MAX	Raw CVC	% CVC MAX
<b>Baseline</b>				
Visit 1 (pre-intervention)	0.33 (0.12) <sup>†</sup>	12.7 (5.2)	0.35 (0.14) <sup>†</sup>	13.9 (4.3)
Visit 2 (post-intervention)	0.39 (0.11)	15.0 (8.2)	0.42 (0.16) <sup>*</sup>	11.0 (7.7)
<b>Initial Peak</b>				
Visit 1 (pre-intervention)	1.55 (0.47)	72.7 (10.4)	1.17 (0.30)	63.0 (16.1)
Visit 2 (post-intervention)	1.71 (0.53) <sup>†</sup>	76.0 (13.6)	1.30 (0.30) <sup>†*</sup>	71.9 (10.4)
<b>Plateau</b>				
Visit 1 (pre-intervention)	1.67 (0.50) <sup>†</sup>	78.8 (12.0) <sup>†</sup>	1.49 (0.37) <sup>†</sup>	71.7 (12.1)
Visit 2 (post-intervention)	2.03 (0.62) <sup>*</sup>	85.0 (10.7) <sup>*</sup>	1.63 (0.39) <sup>*</sup>	77.7 (7.3)

280 <sup>†</sup>P = <0.05 between groups

281 <sup>\*</sup>P<0.05 between visits (within groups)

282

283 ***Peak Heart Rate and RPE***

284 A reduction in peak heart rate was observed in the older group only [149.5 (7.9) to 146.1  
 285 (6.5); P = 0.01]. No differences between the groups or changes in RPE were noted.

286

## Discussion

288 Our study is the first to investigate the effects of a short-term, adapted Nordic diet  
289 intervention on endothelial function and tissue oxygenation in adults at rest and during mild-  
290 to-moderate exercise. These results highlight the short-term effects of the diet with respect to  
291 a number of parameters which define CVD risk and day-to-day function in older and younger  
292 individuals. Further, our findings support previous work elucidating the effects of sedentary  
293 aging on cutaneous microvascular function: Similar to Tew et al. (2010), our older  
294 participants demonstrated lower pre-intervention raw CVC during the initial peak and plateau  
295 stages, suggesting age-related vasodilation impairment in response to local skin heating. The  
296 mechanisms underpinning the weakened initial peak observed in older adults are not fully  
297 understood however evidence suggests that local sensory nerve dysfunction, diminished  
298 noradrenergic sympathetic nerve stimulation and reduced NO synthesis might attenuate the  
299 rapid skin hyperaemic response in older individuals (James et al. 2006)—reduced  
300 endothelial-mediated NO synthesis is thought to explain the diminished plateau. Age-related  
301 microvascular impairment is associated with coronary events (James et al. 2006). Strategies  
302 to improve microvascular function are clinically important therefore.

303 Tew et al. (2010) identified that maintaining aerobic fitness into advanced age might be one  
304 such strategy, while findings elsewhere (Klonizakis et al. 2013, Klonizakis et al. 2016)  
305 suggest that diet and exercise might also provide long-term benefits. Our data supports this  
306 suggestion, and highlights encouraging benefits of a short-term dietary modification. Older  
307 participants experienced improvements in raw CVC at baseline, initial peak and plateau  
308 stages, suggesting that the AND led to improvements in axon-mediated vasodilation (during  
309 the initial peak) and endothelial-mediated NO synthesis (during the plateau). Decreasing  
310 axon-mediated vasodilation indicates microcirculatory dysfunction (Nouri et al, 2012) and  
311 increased risk of cardiovascular events (Hadi et al. 2007). Our data therefore provide a

312 preliminary indication that diet might be a mechanism to attenuate this dysfunction in an  
313 aging population. Younger participants experienced improvements in a number of CVC  
314 parameters similarly, the magnitude of which appeared to be greater than the older  
315 individuals. While drawing comparisons with other studies is erroneous given the novelty  
316 and specificity of dietary-intervention research, our data reflects those observed elsewhere:  
317 Klonizakis et al. (2013, 2014) and Alkhatib and Klonizakis (2014) revealed that the MD,  
318 when coupled with exercise, led to greater improvements in endothelium-dependent  
319 vasodilation than an exercise-only condition in older sedentary individuals ( $55 \pm 4$  years).  
320 Collectively, data is beginning to suggest that wholefood; nutrient-dense diets might promote  
321 endothelial health by also increasing NO synthesis, given adequate consumption of nitrate-  
322 rich foods (Sobko et al. 2010). Indeed, age-related NO decline is also associated with CVD  
323 risk (James et al. 2006); augmenting endothelial-dependent NO production via dietary  
324 modification might mitigate such risk however.

325 The role of dietary fat in microvascular health is multifaceted: High-fat meals appear to  
326 promote endothelial dysfunction (Esposito et al. 2007) and a high-saturated-fat diet might  
327 impair endothelial vasodilation (Keogh et al. 2005). However, *n*-3 Polyunsaturated Fatty  
328 Acids (PUFA) might *improve* endothelial health (Calder et al. 2013). While our participants  
329 reduced saturated and total fat (trends only) intakes, neither group increased *n*-3 PUFA  
330 concomitantly. This was despite the AND encouraging fish consumption. Data indicates that  
331 *n*-3 PUFA might activate NO synthesis and reduce oxidative stress and inflammation (Zanetti  
332 et al. 2015). It is possible therefore that substituting a proportion of saturated and total fat in  
333 the diet for *n*-3 PUFAs might have led to superior changes to a number of microvascular  
334 parameters we assessed. Future research might need to ensure greater fish consumption for  
335 these to be realised however.

336 We adopted an incremental, sub-maximal exercise test such that tissue oxygenation could be  
337 assessed at rest and during activity. Reduced oxygen perfusion is associated with aging:  
338 Free-radical mediated endothelium-dependent NO degradation has been demonstrated in  
339 older adults, leading to arterial narrowing, increased blood pressure and cardiac  
340 complications (Gates et al. 2009). We observed no effects of the AND on any TcPO<sub>2</sub>  
341 parameters, contrasting data suggesting that short-term green tea consumption might lead to  
342 improved tissue perfusion (Wasilewski et al. 2016). This was surprising considering that the  
343 AND is a flavonoid-rich diet and that the flavonoids found in green tea explain its efficacy  
344 (Wasilewska et al. 2016). While not measured specifically, insufficient consumption of  
345 flavonoid-rich foods (e.g. berries, apples, and parsley) might explain our findings. Future  
346 studies should ensure sufficient consumption of these foods for improvements in TcPO<sub>2</sub> to be  
347 possible as part of an AND intervention.

348 We observed statistically-significant reductions in body-fat % in younger participants and  
349 improved peak heart rate in older participants, adding to existing data revealing health  
350 benefits of Nordic-type diets (Kanerva et al. 2014b). The reductions in body fat we observed  
351 (young = -2%) corresponded with a weight-loss trend (young = -2.5%). While the AND  
352 might have led to this weight loss, we cannot rule out that participating in a dietary  
353 intervention might have prompted some participants to lose weight intentionally, and that this  
354 weight loss might explain some of the improvements we noted. Nevertheless, owing to such  
355 changes being observed, and that Nordic eating appears to be associated with low obesity  
356 prevalence (Kanerva et al. 2013), and weight loss elsewhere (Poulsen et al. 2015), future  
357 research should investigate the potential of a Nordic diet for weight-management purposes  
358 specifically.

359 The efficacy of the AND to elicit improvements in microvascular function appeared to  
360 greatest in younger participants, contrasting our original hypothesis. This might be due to

361 younger participants experiencing larger changes to habitual eating patterns via compliance  
362 to the AND: while pre-to-post intervention NDS differed for both groups, a higher mean  
363 change was observed in younger participants. It is also possible that younger individuals  
364 might be more responsive to the AND: lifestyle (diet, physical activity) *and* biological factors  
365 (hormonal changes, etc.) are known to lead to endothelial dysfunction with age; older  
366 participants might have experienced dampened responsiveness due to such age-related  
367 factors. However, complex interventions (diet and physical activity) have been shown to lead  
368 to important, long-lasting improvements in microvascular function in older individuals  
369 (Klonizakis et al. 2014). Future research might need to account for older participants'  
370 responsiveness to diet-only intervention; complex interventions might be needed for greater  
371 changes to be realised.

372 Limitations of this research include the lack of objective measures to determine compliance  
373 to the AND, and the absence of control groups. Compliance measurements could be explored  
374 in future studies; the lack of control group might make inferences about the efficacy of the  
375 AND to elicit functional change to the endothelium difficult without a comparator. However,  
376 baseline measurements were used here for such comparisons, and it was felt that control  
377 groups were unnecessary owing to previous data highlighting the efficacy of dietary  
378 intervention to stimulate microvascular change in young and old groups (Wasilewski et al.  
379 2016). The short study duration, which might also be considered a limitation by some, was  
380 intentional, with the view to explore the minimum duration after which medium-term effects  
381 can be identifiable across populations. Our results have provided such indications, and so  
382 further studies with a longer-duration might now be explored. Finally, an additional limitation  
383 here is the lack of objective exercise-behaviour monitoring employed outside of testing. Such  
384 monitoring should be implemented in future investigations.

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## **Conclusion**

This study supports current evidence highlighting health benefits of regional diets. Our participants, who were sedentary, observed improvements in body composition and microvascular function by integrating Nordic foods into their diet for a 4-week period. There is now a need to investigate effects of Nordic-type diets over longer intervention periods, particularly among older individuals (55+ years), who appeared to be less responsive to the intervention. Age-related endothelial dysfunction might be a preliminary indicator of CVD events; strategies to attenuate age-related microvascular deterioration therefore require further investigation.

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