The Effects of Supplemental Fish Oil on Blood Pressure and Morning Cortisol in Normotensive Adults: A Pilot Study

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The Effects of Supplemental Fish Oil on Blood Pressure and Morning Cortisol in Normotensive Adults: A Pilot Study

Keywords
omega 3 fatty acids, pulse pressure, fish oil, dietary supplements, Eicosapentaenoic Acid, Docosahexaenoic Acid, automated oscillometric devise

Abstract
Purpose: To determine the effects of 6wk of supplementation with fish oil (FO) on blood pressure and the morning salivary cortisol concentration in normotensive adults.

Methods: Testing was performed following an overnight fast. Subjects (n=40; 35+/-13y, mean+/-SD) rested supine for 40 min, at which time blood pressure and heart rate were measured. Saliva was collected and analyzed for cortisol. Subjects were then randomly assigned to either: 4g/d of Safflower Oil (SO); pr 4g/d of FO supplying 1,600mg/d eicosapentaenoic acid and 800mg/d docosahexaenoic acid. Testing was repeated following 6wk treatment.

Results: Compared to SO, there was a significant decrease in systolic blood pressure with FO (SO= 1.3+/-5.8 mmHg; FO= -6.8+/-10.2 mmHg; p=0.004), a significant reduction in pulse pressure with FO (SO= 0.2+/-7.8 mmHg; FO= -6.4+/-8.8 mmHg; p=0.02), and a tendency for a decrease in mean arterial pressure (SO= 1.2+/-5.3 mmHg; FO= -2.5+/-7.3 mmHg; p=0.08). There was a tendency for salivary cortisol to decrease with FO (SO= 0.005+/-0.129 µg/dL; FO= -0.068+/-0.148 µg/dL; p=0.072), however, this change was not significant;y correlated with the change in systolic blood pressure (r=0.021, p=0.929).

Conclusion: 6wk of supplementation with FO significantly decreases systolic blood pressure in normotensive adults and this change was not significantly correlated with a reduction in salivary cortisol.

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The Effects of Supplemental Fish Oil on Blood Pressure and Morning Cortisol in Normotensive Adults: a Pilot Study

Eric E. Noreen and Josef Brandauer

Abstract

PURPOSE: To determine the effects of 6wk of supplementation with fish oil (FO) on blood pressure and the morning salivary cortisol concentration in normotensive adults. METHODS: Testing was performed following an overnight fast. Subjects (n=40; 35 +/- 13y, mean +/- SD) rested supine for 40min, at which time blood pressure and heart rate were measured. Saliva was collected and analyzed for cortisol. Subjects were then randomly assigned to either: 4g/d of Safflower Oil (SO); or 4g/d of FO supplying 1,600mg/d eicosapentaenoic acid and 800mg/d docosahexaenoic acid. Testing was repeated following 6wk of treatment. RESULTS: Compared to SO, there was a significant decrease in systolic blood pressure with FO (SO=1.3 +/- 5.8 mmHg; FO=-6.8 +/- 10.2 mmHg; p=0.004), a significant reduction in pulse pressure (SO=0.2 +/- 7.8 mmHg; FO=-6.4 +/- -8.8 mmHg; p=0.02), and a tendency for a decrease in mean arterial pressure (SO=1.2 +/- 5.3 mmHg; FO=-2.5 +/- 7.3 mmHg; p=0.08). There was a tendency for salivary cortisol to decrease with FO (SO=0.005 +/- 0.129 µg/dL; FO=-0.068 +/- 0.148 µg/dL; p=0.072), however, this change was not significantly correlated with the change in systolic blood pressure (r=0.021, p=0.929). CONCLUSION: 6wk of supplementation with FO significantly decreases systolic blood pressure in normotensive adults and this change was not significantly correlated with a reduction in salivary cortisol.

KEYWORDS: eicosapentaenoic acid, docosahexaenoic acid, omega 3 fatty acids, pulse pressure, automated oscillometric device

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INTRODUCTION

It is generally acknowledged that an increased dietary intake of fish, or fish oil that is rich in long chain omega 3 fatty acids, results in a modest, yet significant reduction in blood pressure (Appel, Miller, Seidler, & Whelton, 1993; Geleijnse, Giltay, Grobbee, Donders, & Kok, 2002; Morris, Sacks, & Rosner, 1993). However, the majority of the studies examining the effects of fish oil on blood pressure have been performed in individuals with either primary or secondary hypertension, and/or in individuals with a known disease. Currently very little is known about the effects of fish oil on blood pressure in healthy, normotensive subjects. While a few studies have examined the effects of fish oil on blood pressure in subjects with untreated borderline hypertension, the results have been equivocal. Prisco, et al. (1998) observed a significant 6mm Hg decrease in systolic blood pressure and a significant 5mm Hg decrease in diastolic blood pressure following 2 months of fish oil treatment, however, Sacks et al. (1994) found no effect of 6 months of fish oil treatment on blood pressure in borderline hypertensives. Similarly, the results of fish oil treatment in healthy normotensive adults have also been equivocal. Mortensen et al. (1983) observed a significant decrease in systolic blood pressure of ~ 4mm Hg following 4wks of fish oil treatment. Conversely, other studies have found no effect of fish oil on blood pressure in normotensive subjects (Bruckner, Webb, Greenwell, Chow, & Richardson, 1987; Prisco et al., 1998; Ryu, Lerner, & Sullivan, 1990). Flaten et al. (1990) similarly concluded that there was no effect of fish oil on blood pressure in normotensive subjects following 6wks of treatment. They did observe a significant decrease in systolic blood pressure of ~ 4mm Hg following 6wks of treatment with fish oil, but they largely dismissed this change since they also observed a similar change in the group that received the olive oil “placebo.” However, recent work has shown that olive oil can lower blood pressure when it is given to subjects who do not routinely consume an olive oil rich diet (Bondia-Pons et al., 2007; Perona et al., 2009). This strongly suggests that caution should be used when interpreting results from studies that have used olive oil as the placebo treatment.

The exact mechanism(s) by which dietary long chain omega 3 fatty acids reduce blood pressure are not fully understood. One potentially novel mechanism explaining the hypotensive effect of dietary omega 3 fatty acids could be a reduction in blood pressure secondary to a reduction in cortisol. The effects of dietary fish oil on cortisol concentration are poorly understood, however, there is limited evidence that an increased consumption of fish oil results in a reduction in cortisol concentration (Delarue et al., 2003; Eguchi et al., 2011; Michaeli, Berger, Revelly, Tappy, & Chiolero, 2007).
Although the adrenal hormone cortisol is essential for maintaining normal blood pressure, an excess of cortisol results in hypertension (Whitworth, Mangos, & Kelly, 2000; Whitworth, Williamson, Mangos, & Kelly, 2005). The exact mechanism(s) by which an excess of cortisol results in hypertension is unclear, but research has pointed to an inhibition of the vasodilator nitric oxide (Kelly, Tam, Williamson, Lawson, & Whitworth, 1998), and/or an increase in the concentration of the vasoconstrictor erythropoietin (Kelly, Martin, & Whitworth, 2000). Historically, cortisol induced hypertension was thought to be clinically relevant only to patients with Cushing’s disease, or those using exogenous glucocorticoid therapy (Whitworth, et al., 2000). However, it has been suggested by some researchers that elevated cortisol may also contribute to essential hypertension (Soro, Ingram, Tonolo, Glorioso, & Fraser, 1995; Walker, Best, Shackleton, Padfield, & Edwards, 1996). In support of this, several studies have found a correlation between elevated morning cortisol levels and hypertension (Bose, Olivan, & LaFerrere, 2009; Filipovsky et al., 1996; Kidambi et al., 2007; Phillips et al., 2005; Schoorlemmer, Peeters, van Schoor, & Lips, 2009). However, the relationship between morning cortisol levels and blood pressure in normotensive individuals has not been clearly established.

Therefore, the purpose of this pilot study was to assess the effects of fish oil on blood pressure and morning cortisol concentrations, as well as the relationship between changes in morning cortisol concentrations and blood pressure in healthy normotensive adults following 6wk of an increased dietary intake of fish oil.

METHODS

Prior to all testing, approval for the study was obtained from the institutional review board at Gettysburg College, and written informed consent was obtained from all subjects.

Healthy adults (19-55y) were recruited through word of mouth and flyers posted at Gettysburg College and the surrounding community. Subjects were healthy and active, but not engaged in consistent, systematic exercise training. Individuals who ate fatty fish at least 3 times a month, or were supplementing their diet with omega 3 fatty acids were excluded from the study. Subjects were also excluded if they had a doctor diagnosed metabolic or endocrine disorder, or if they were taking any prescription medications, with the exception of birth control hormones and allergy or asthma medications. Individuals with a baseline systolic blood pressure measurement greater than 140 mmHg, or a diastolic blood pressure measurement greater than 90 mmHg were excluded from the study. In total, 40 individuals volunteered to participate (Table 1). Subjects were asked to maintain their current diet and exercise practices throughout the study. Additionally,
subjects were instructed to keep a diet and activity log for the 24h period prior to the baseline testing and to duplicate these for the 24h period prior to the post testing.

Experimental Protocol

Subjects reported to the laboratory first thing in the morning immediately after waking. Subjects were not allowed to eat or drink anything other than water starting 10h before their scheduled testing time. This included the use of mouthwash or toothpaste in the morning since this could interfere with the salivary cortisol measurements. Upon arrival at the laboratory, subjects were asked to void if they could, and then they rested quietly in a supine position for 40 minutes in a darkened room, after which blood pressure and heart rate were measured using an automated oscillometric device (Omron HEM-907, Omron Healthcare, Kyoto Japan) that automatically took the average of two readings. Following these tests, a saliva sample was taken via passive drool and later analyzed for cortisol content (see below for details). Subjects were then randomly assigned in a double blind manner to one of two groups based on the order of entrance into the study.

The two groups were:

**Safflower oil (SO):** 4g/d of safflower oil (Genuine Health Corporation, Toronto, Ontario, CA) administered in 4 enteric coated capsules. Each capsule provided 1g of cold pressed, high linoleic acid, safflower oil. See appendix A for a breakdown of fatty acids found in the safflower oil capsules. Safflower oil is a rich source of long chain polyunsaturated omega 6 fatty acids. Since the average western diet contains considerable amounts of omega 6 fatty acids, the safflower oil is believed to be a good placebo.

**Fish oil (FO):** 4g/d concentrated fish oil (o3 mega extra strength, Genuine Health Corporation, Toronto, Ontario, CA) administered in 4 enteric coated capsules. Each capsule provided 400mg eicosapentaenoic acid and 200mg of docosahexaenoic acid. See appendix A for a breakdown of the fatty acids found in the fish oil capsules.

Subjects were instructed to take 2 capsules with breakfast and 2 capsules with dinner for a 6wk period. Subject compliance was monitored by asking the subjects to return any left over pills at the completion of the study. Subjects were also asked to write down any possible side effects noted during the 6wk treatment.
Subjects were instructed to maintain their normal diet and exercise patterns during the treatment period. All testing was repeated following 6wk of treatment.

**Salivary Analysis**

Subjects rinsed their mouth with water prior to all saliva collections to minimize contamination of the samples. Saliva was collected in a polypropylene vial via passive drool through a short straw and stored at -80 °C until analyzed. Prior to analysis, samples were thawed and centrifuged at 10,000g for 20 minutes to remove mucins and analyzed in duplicate for cortisol concentration using a commercially available enzyme immunoassay kit (Salimetrics, State College, PA, USA). Salivary cortisol is a sensitive marker of activation the hypothalamus-pituitary-adrenal system’s response to stress, which correlates very well to plasma cortisol concentrations (Hellhammer, Wust, & Kudielka, 2009). The normal morning salivary cortisol concentrations range from 0.094 – 1.551 μg/dL for healthy adults in the age range used in this study (Aardal & Holm, 1995).

**Blood Pressure Variables**

Heart rate, systolic and diastolic blood pressure were automatically determined by the Omron HEM-907 automated oscillometric device. Pulse pressure was calculated as the difference between systolic and diastolic blood pressure, and the mean arterial pressure (MAP) was calculated as \( \frac{2}{3} DBP + \frac{1}{3} SBP \).

**Statistical Analysis**

Data were analyzed using the Statistical Package for the Social Sciences version 13 (SPSS Inc., Chicago, IL). Salivary cortisol concentrations were not normally distributed so they were log transformed prior to analysis. A group by time, repeated measures ANOVA was used to evaluate significant differences, and a standard Pearson’s r was used to evaluate correlations. For all analysis, the alpha level was set at \( p \leq 0.05 \). All values are presented as mean ± standard deviation.

**RESULTS**

**Blood Pressure**

The results from the blood pressure measurements are presented in Table 2. There were no significant differences between groups at baseline for any values. A group by time repeated measures ANOVA revealed a significant decrease in
systolic blood pressure (Table 2: SO = 1.3±5.8 mmHg; FO = -6.8±10.2 mmHg; p=0.004), and pulse pressure (Table 2: SO = 0.2±7.8 mmHg; FO = -6.4±8.8 mmHg; p=0.02), following fish oil treatment. Although MAP decreased following fish oil treatment, this change was not significant (SO = 1.2±5.3 mmHg; FO = -2.5±7.3 mmHg; p=0.08). There were no significant differences observed between the groups for heart rate or diastolic blood pressure.

**Salivary Cortisol Concentrations**

Salivary cortisol concentrations decreased following fish oil treatment, however the change was not significant (Table 2: SO = 0.005±0.129 μg/dL; FO = -0.068±0.148 μg/dL; p=0.072).

**Correlations**

There were no significant relationships observed between the baseline morning salivary cortisol concentration and the blood pressure variables (Table 3). Following treatment, there were no significant relationships observed in the FO group between the change in salivary cortisol concentration and any of the blood pressure variables (Table 4).

**Subject Compliance and Side effects**

No subject in either group returned any unused pills, suggesting 100% compliance. It should be noted, however, that all of the subjects were friends, colleagues, or students of the researchers. This could have given them an increased incentive to follow the study guidelines, or alternatively it may have served to decrease the likelihood that the subjects would admit to missing doses. Both treatments were very well tolerated, with no major side effects noted for either treatment. Of particular note, no one in the fish oil group reported experiencing “fishy” reflux, which is a common side effect noted in studies that have used a non-enteric coated fish oil capsule.

**DISCUSSION**

The main finding of this study was a significant decrease in systolic blood pressure following 6wks of consumption of a fish oil that is rich in long chain omega 3 fatty acids (Table 2). While this finding is in agreement with previous studies (Flaten et al., 1990; Mortensen, Schmidt, Nielsen, & Dyerberg, 1983), other studies have failed to observe a significant effect of fish oil on blood pressure in normotensive subjects (Bruckner, et al., 1987; Prisco, et al., 1998;
Ryu, et al., 1990). Unfortunately, differences in study design, such as the dose of fish oil used and the duration of treatment, limits direct comparison between the different studies that have examined the effects of fish oil on blood pressure in normotensive subjects. More work is needed to clearly define the effects of fish oil supplementation on blood pressure in healthy normotensive subjects.

The clinical significance of the hypotensive effect observed in this study is also unknown. Previous work has shown that decreasing blood pressure in normotensive individuals with known coronary disease results in a reduction in negative cardiovascular events (Nissen et al., 2004). Although no study to date has directly determined if a similar protective effect exists when blood pressure is reduced in normotensive subjects without coronary disease, epidemiologic data reveal a decreased risk of cardiovascular events and all cause mortality in individuals who have a resting systolic blood pressure below the accepted value of 120mm Hg (Stamler, Stamler, & Neaton, 1993). Therefore the results of the present study indicate that the increased consumption of fish oil may serve as a prophylactic means of reducing the risk of cardiovascular disease in healthy normotensive individuals, but more work is needed to clearly establish this.

The repeated measures ANOVA showed a tendency for salivary cortisol concentrations to decrease in the fish oil group (SO = 0.005±0.129 μg/dL; FO = -0.068±0.148 μg/dL; p=0.072). However, when a two tailed dependent t test was performed on the Pre and Post scores of each group independently, the SO change was not significant (p=0.93), but the Post score was significantly lower than the Pre score in the FO group (p=0.05). It is very likely that the reduced statistical power of the omnibus F used in the repeated measures ANOVA resulted in a type II error, and the reduction in salivary cortisol concentrations following fish oil supplementation is a real effect. In support of this, the 95% confidence interval of the Pre-Post difference in salivary cortisol concentration for the fish oil group (Table 2) does not contain the null value of zero, but rather contains only negative values (-0.132 to -0.004 μg/dL). In contrast, the 95% confidence interval for the safflower oil group is centered on a mean difference value of essentially the null value of zero (-0.124 to 0.134 μg/dL). Taken together, these additional statistics suggest a reduction in salivary cortisol concentration in the subjects taking the fish oil. Although there is a very limited amount of data examining the effects of dietary fish oil on cortisol levels, this finding is in agreement with other studies that have shown a reduction in cortisol levels following fish oil treatment (Delarue, et al., 2003; Eguchi, et al., 2011; Michaeli, et al., 2007). However, it should be pointed out that these studies measured cortisol concentration following a stressor, whereas the present study measured morning cortisol concentration in a rested state.

An increased consumption of fish oil resulted in a reduction in blood pressure as well as a reduction in morning salivary cortisol concentrations.
Despite this, there was no significant correlation observed in the fish oil group between the change in blood pressure and the change in salivary cortisol concentrations following treatment (Table 4). This would suggest that the reduction in blood pressure observed following fish oil treatment in normotensive subjects is not a result of a reduction in cortisol levels. In the present study, the subjects had normal values for morning salivary cortisol concentrations, as well as normal resting supine blood pressure values. Therefore, it still remains a possibility that a reduction in cortisol may relate to a reduction in blood pressure in individuals that have an elevated blood pressure as a result of elevated levels of cortisol. In support of this, it has been shown that the administration of the cortisol suppressing drug dexamethasone lowers blood pressure in hypertensive, but not normotensive subjects (Whitworth, Gordon, McLachlan-Troup, Scoggins, & Moulds, 1989). Hence, dietary fish oil may have a more pronounced effect in individuals who have an elevated blood pressure as a result of increased cortisol levels since the hypotensive effect from a reduction in cortisol may be additive to the direct hypotensive effects of fish oil consumption.

Since the reduction in cortisol concentration does not appear to explain the observed reduction in blood pressure in the present study, the mechanism(s) responsible for the reduction in blood pressure remain unclear. Other studies have suggested a variety of potential mechanisms by which fish oil consumption can decrease blood pressure, including an increased large artery vasodilation (Engler & Engler, 2000; Johansen, Seljeflot, Hostmark, & Arnesen, 1999), a reduced reactivity of vessel vascular smooth muscle (Chu, Yin, & Beilin, 1992), an inhibition of hemostasis and platelet aggregation (Rogers et al., 1987; Vericel, Calzada, Chapuy, & Lagarde, 1999), and an enhanced endothelium dependent vasodilation (Shah et al., 2007; Shimokawa & Vanhoutte, 1989). It is likely then, that one, or a combination, of the factors listed was responsible for the reduction in blood pressure following fish oil treatment observed in the present study.

In addition to the modest sample size used, there are other limitations to the present study. The main limitation of this study was the collection of data at only one time point pre and post treatment. It is generally recognized that blood pressure measurements should be made several times over the course of the day to accurately determine a true average blood pressure (Powers et al., 2011). Given the dynamic nature of blood pressure control, it remains a possibility that fish oil treatment only lowers systolic blood pressure under the specific conditions used for measurement in the present study, and the effect would be lost if 24h ambulatory measurements were used. Similarly, cortisol excretion has a pronounced diurnal rhythm, and a series of measurements over 24h would be more likely to capture the true effect of dietary fish oil on cortisol excretion. There are a multitude of lifestyle choices that can influence resting blood pressure. Although the subjects were instructed not to change their diet or activity
level during the course of the study, no attempts were made to control for these variables.

The present study is the first to examine the relationship between morning cortisol levels and blood pressure following fish oil treatment in healthy normotensive adults. Although there was a significant reduction in systolic blood pressure following fish oil treatment, this was not correlated with the reduction in salivary cortisol concentrations observed. However, it remains a possibility that a reduction in cortisol following fish oil treatment may be related to a reduction in blood pressure in individuals that have hypertension as a result of elevated cortisol levels. Future research should evaluate the effects of fish oil treatment on individuals with elevated cortisol levels and hypertension. The clinical significance of lowering systolic blood pressure in already normotensive adults is unclear; more studies are needed to determine if this change results in a decrease in long-term morbidity and mortality rates.
Table 1
Subject Characteristics

<table>
<thead>
<tr>
<th></th>
<th>Safflower Oil</th>
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<td></td>
<td>Pre</td>
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<td>Post-Pre Difference</td>
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<td>Female (n)</td>
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<td>14</td>
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<tr>
<td>Age (y)</td>
<td>36±14y</td>
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<td>35±13y</td>
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<td>(29,41)</td>
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<td>(29,41)</td>
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<td>Weight (kg)</td>
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<td>71.5±16.0</td>
<td>0.3±0.8</td>
<td>72.3±14.8</td>
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<td></td>
<td>(63.8,77.5)</td>
<td>(64.0,79.0)</td>
<td>(-0.1,0.7)</td>
<td>(65.4,79.2)</td>
</tr>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>72.3±14.1</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>(65.7,78.9)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(-0.4,0.4)</td>
</tr>
</tbody>
</table>

Data are expressed as means ± SD (95% confidence interval).
Table 2
Pre and Post values following 6 weeks of treatment with 4g/d of safflower oil, or 4 g/d of fish oil

<table>
<thead>
<tr>
<th></th>
<th>Safflower Oil</th>
<th>Fish Oil</th>
<th>Post-Pre Difference</th>
<th>Safflower Oil</th>
<th>Fish Oil</th>
<th>Post-Pre Difference</th>
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</thead>
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<td>Post</td>
<td>Difference</td>
<td>Pre</td>
<td>Post</td>
<td>Difference</td>
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<tr>
<td>Salivary Cortisol</td>
<td>0.316±0.242</td>
<td>0.320±0.319</td>
<td>0.005±0.129††</td>
<td>0.275±0.186</td>
<td>0.207±0.137</td>
<td>-0.068±0.148††</td>
</tr>
<tr>
<td>(µg/dL)</td>
<td>(0.203,0.429)</td>
<td>(0.171,0.469)</td>
<td>(-0.124,0.134)</td>
<td>(0.188,0.362)</td>
<td>(0.138,0.276)</td>
<td>(-0.132,-0.004)</td>
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<tr>
<td>Heart Rate</td>
<td>65±10</td>
<td>67±12</td>
<td>2±7</td>
<td>62±12</td>
<td>61±10</td>
<td>-1±9</td>
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<td>(BPM)</td>
<td>(60,70)</td>
<td>(62,72)</td>
<td>(-1,5)</td>
<td>(56,68)</td>
<td>(56,66)</td>
<td>(-3,3)</td>
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<tr>
<td>Systolic BP</td>
<td>108.4±10.9</td>
<td>109.7±12.5</td>
<td>1.3±5.8*</td>
<td>111.3±9.9</td>
<td>104.6±8.9</td>
<td>-6.8±10.2*</td>
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<tr>
<td>(mmHg)</td>
<td>(103,113.5)</td>
<td>(103,8,115.6)</td>
<td>(-1.4,4.0)</td>
<td>(106,7,115.9)</td>
<td>(100,4,108.8)</td>
<td>(-11.6,-2.0)</td>
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<tr>
<td>Diastolic BP</td>
<td>67.2±8.2</td>
<td>68.3±9.0</td>
<td>1.1±6.7</td>
<td>68.8±6</td>
<td>68.5±6.7</td>
<td>-0.4±7.4</td>
</tr>
<tr>
<td>(mmHg)</td>
<td>(63.3,71.1)</td>
<td>(64.1,72.5)</td>
<td>(-2.1,4.3)</td>
<td>(66.0,71.6)</td>
<td>(65.3,71.7)</td>
<td>(-3.8,3.0)</td>
</tr>
<tr>
<td>Pulse Pressure</td>
<td>41.2±6.5</td>
<td>41.4±8.8</td>
<td>0.2±7.8**</td>
<td>42.5±10.3</td>
<td>36.1±6.4</td>
<td>-6.4±8.8**</td>
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<tr>
<td>(SBP-DBP)</td>
<td>(38.1,14.3)</td>
<td>(37.3,45.5)</td>
<td>(-3.5,3.9)</td>
<td>(37.7,47.3)</td>
<td>(33.1,39.1)</td>
<td>(-10.5,-2.3)</td>
</tr>
<tr>
<td>MAP</td>
<td>80.9±8.7</td>
<td>82.1±9.4</td>
<td>1.2±5.3†</td>
<td>83.0±5.8</td>
<td>80.5±6.9</td>
<td>-2.5±7.3†</td>
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<tr>
<td>(mmHg)</td>
<td>(76.8,85)</td>
<td>(77.7,86.5)</td>
<td>(-1.3,3.7)</td>
<td>(80.3,85.7)</td>
<td>(77.3,83.7)</td>
<td>(-5.9,0.9)</td>
</tr>
</tbody>
</table>

Data are expressed as means ± SD (95% confidence interval). Data were analyzed using a treatment X time repeated measures ANOVA. BPM = beats per minute. BP = blood pressure. Pulse pressure = Systolic blood pressure – diastolic blood pressure. MAP=mean arterial pressure (2/3 * DBP + 1/3 * SBP) * significant treatment X time interaction, p=0.004, ** significant treatment X time interaction, p=0.02, † treatment X time interaction, p=0.08, †† treatment X time interaction, p=0.072
Table 3. Relationship between baseline salivary cortisol and blood pressure variables

<table>
<thead>
<tr>
<th>Salivary Cortisol</th>
<th>HR</th>
<th>DBP</th>
<th>SBP</th>
<th>PP</th>
<th>MAP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pearson Correlation</td>
<td>0.046</td>
<td>0.052</td>
<td>0.156</td>
<td>0.146</td>
<td>0.107</td>
</tr>
<tr>
<td>Sig. (2-tailed)</td>
<td>0.780</td>
<td>0.750</td>
<td>0.338</td>
<td>0.369</td>
<td>0.511</td>
</tr>
<tr>
<td>N</td>
<td>40</td>
<td>40</td>
<td>40</td>
<td>40</td>
<td>40</td>
</tr>
</tbody>
</table>

HR = heart rate. DBP = diastolic blood pressure. SBP = systolic blood pressure. PP = pulse pressure. MAP = mean arterial pressure.

Table 4. Relationship between the change in salivary cortisol and the change in blood pressure variables following 6 wk of fish oil treatment

<table>
<thead>
<tr>
<th>Δ Salivary Cortisol</th>
<th>ΔHR</th>
<th>ΔDBP</th>
<th>ΔSBP</th>
<th>ΔPP</th>
<th>ΔMAP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pearson Correlation</td>
<td>-.046</td>
<td>-.067</td>
<td>.021</td>
<td>.081</td>
<td>-.035</td>
</tr>
<tr>
<td>Sig. (2-tailed)</td>
<td>.847</td>
<td>.779</td>
<td>.929</td>
<td>.734</td>
<td>.883</td>
</tr>
<tr>
<td>N</td>
<td>20</td>
<td>20</td>
<td>20</td>
<td>20</td>
<td>20</td>
</tr>
</tbody>
</table>

Δ = change in variable following treatment.
Appendix A. Fatty acid profile of fish oil and safflower oil capsules.

<table>
<thead>
<tr>
<th>Fatty Acid</th>
<th>Fish Oil (%)</th>
<th>Safflower Oil (%)</th>
<th>Fatty Acid</th>
<th>Fish Oil (%)</th>
<th>Safflower Oil (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>14:0</td>
<td>0.00</td>
<td>0.00</td>
<td>20:1w11</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>14:1</td>
<td>0.00</td>
<td>0.00</td>
<td>20:1w9</td>
<td>2.05</td>
<td>0.00</td>
</tr>
<tr>
<td>15:0</td>
<td>0.00</td>
<td>0.00</td>
<td>20:1w7</td>
<td>1.02</td>
<td>0.00</td>
</tr>
<tr>
<td>15:1</td>
<td>0.00</td>
<td>0.00</td>
<td>21:0</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>16:0</td>
<td>0.33</td>
<td>7.00*</td>
<td>20:2w6</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>16:1</td>
<td>0.39</td>
<td>0.00</td>
<td>20:3w6</td>
<td>0.35</td>
<td>0.00</td>
</tr>
<tr>
<td>16:2w6</td>
<td>0.00</td>
<td>0.00</td>
<td>20:4w6</td>
<td>2.74</td>
<td>0.00</td>
</tr>
<tr>
<td>17:0</td>
<td>0.00</td>
<td>0.00</td>
<td>20:3w3</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>17:1</td>
<td>0.00</td>
<td>0.00</td>
<td>20:4w3</td>
<td>1.82</td>
<td>0.00</td>
</tr>
<tr>
<td>16:4w3</td>
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<td>0.00</td>
<td>20:5w3</td>
<td>44.42</td>
<td>0.00</td>
</tr>
<tr>
<td>18:0</td>
<td>1.10</td>
<td>2.00*</td>
<td>21:5w3</td>
<td>2.67</td>
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<tr>
<td>18:1w9</td>
<td>1.84</td>
<td>13.0*</td>
<td>22:0</td>
<td>0.47</td>
<td>0.00</td>
</tr>
<tr>
<td>18:1w7</td>
<td>0.7</td>
<td>0.00</td>
<td>22:1w11</td>
<td>2.61</td>
<td>0.00</td>
</tr>
<tr>
<td>18:2w6</td>
<td>0.00</td>
<td>78.0*</td>
<td>22:1w13</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>18:2w4</td>
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<td>0.00</td>
<td>22:1w7</td>
<td>0.41</td>
<td>0.00</td>
</tr>
<tr>
<td>18:3w6</td>
<td>0.00</td>
<td>0.00</td>
<td>22:1w9</td>
<td>0.74</td>
<td>0.00</td>
</tr>
<tr>
<td>18:3w3</td>
<td>0.00</td>
<td>0.00</td>
<td>22:4w6</td>
<td>0.61</td>
<td>0.00</td>
</tr>
<tr>
<td>18:4w3</td>
<td>0.36</td>
<td>0.00</td>
<td>22:5w6</td>
<td>1.03</td>
<td>0.00</td>
</tr>
<tr>
<td>19:0</td>
<td>0.00</td>
<td>0.00</td>
<td>22:5w3</td>
<td>8.97</td>
<td>0.00</td>
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<tr>
<td>19:1</td>
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<td>0.00</td>
<td>24:0</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>20:0</td>
<td>0.48</td>
<td>0.00</td>
<td>22:6w3</td>
<td>23.69</td>
<td>0.00</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>24:1w9</td>
<td>0.89</td>
<td>0.00</td>
</tr>
</tbody>
</table>

* average value
REFERENCES


