



Artery Research

ISSN (Online): 1876-4401

ISSN (Print): 1872-9312

Journal Home Page: <https://www.atlantis-press.com/journals/artres>

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To cite this article: Salome Kristensen, Erik Berg Schmidt, Annette Schlemmer, Claus Rasmussen, Esther Lindgreen, Martin Berg Johansen, Jeppe Hagstrup Christensen (2016) 7.7: THE EFFECT OF MARINE N-3 POLYUNSATURATED FATTY ACIDS ON CARDIAC AUTONOMIC AND HEMODYNAMIC FUNCTION IN PATIENTS WITH PSORIATIC ARTHRITIS: A RANDOMISED, DOUBLE-BLIND, PLACEBO-CONTROLLED TRIAL, Artery Research 16:C, 64–64, DOI: <https://doi.org/10.1016/j.artres.2016.10.053>

To link to this article: <https://doi.org/10.1016/j.artres.2016.10.053>

Published online: 7 December 2019

7.7

THE EFFECT OF MARINE N-3 POLYUNSATURATED FATTY ACIDS ON CARDIAC AUTONOMIC AND HEMODYNAMIC FUNCTION IN PATIENTS WITH PSORIATIC ARTHRITIS: A RANDOMISED, DOUBLE-BLIND, PLACEBO-CONTROLLED TRIAL

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Background: Patients with psoriatic arthritis are at high cardiovascular risk. Marine n-3 polyunsaturated fatty acids (PUFA) may reduce the incidence of cardiovascular disease. The aim of this study was to investigate the effect of marine n-3 PUFA on cardiac autonomic function and vascular function in patients with psoriatic arthritis.

Methods: The study was conducted as a randomized, double-blind, placebo-controlled trial, where 145 patients with psoriatic arthritis were supplemented with 3 g of n-3 PUFA or olive oil (control) daily for 24 weeks. Blood pressure, heart rate, HRV, central blood pressure, pulse wave velocity (PWV) and fatty acid composition of granulocytes, were determined.

Results: At baseline we found a significant difference in the HRV parameter RR when comparing subjects with the highest vs the lowest fish intake ($p = 0.03$). After supplementation for 24 weeks there was a trend towards an increase in RR ($p = 0.13$) and decrease in heart rate ($p = 0.12$) comparing the n-3 PUFA group with the control group. However, per-protocol analysis (performed on participants who completed the trial with a good compliance) showed significantly increased RR ($p = 0.01$) and lowered heart rate ($p = 0.01$) in the n-3 PUFA supplemented patients compared to controls. Blood pressure, PWV and central blood pressure did not change after supplementation with n-3 PUFA.

Conclusions: Marine n-3 PUFA increased HRV in patients with psoriatic arthritis which may suggest a protective effect of n-3 PUFA against cardiovascular disease in this population.

7.8

ARTERIAL STIFFNESS AND SYSTEMIC INFLAMMATION IN COPD PATIENTS

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Background: COPD is one of the leading causes of mortality worldwide. Systemic low-grade inflammation is a common finding in COPD. Soluble urokinase-type plasminogen activator receptor (suPAR) indicates an inflammatory state and it has an association with atherosclerosis and cardiovascular disease (CVD). ThesuPAR reflects different aspects of inflammation as high sensitive C-reactive protein (hsCRP) and IL-6. Elevated CVD risk is observed in COPD. However the correlation between COPD and arterial stiffness is rarely investigated in the literature.

We investigated the association between some inflammatory biomarkers (suPAR, IL-6, hsCRP) and arterial stiffness in COPD and control patients.

Methods: We measured 45 middle aged individuals (25 COPD and 20 control normotensive patients) without diabetes and cardiovascular disease. IL-6, hsCRP, suPAR were determined in fasting blood samples. Whole body plethysmography, assessment tests and aortic pulse wave velocity (aoPWV), augmentation index (Aix), central systolic blood pressure (cSBP) were determined. COPD patients were categorized according to GOLD-classification.

Results: Patients with COPD have a higher level of IL-6 (5.38 vs 3.63 pg/ml $p = 0.022$), suPAR (2.84 vs 2.41 ng/ml $p = 0.036$), and hsCRP (2.99 vs 1.91 mg/L $p = 0.068$). The patients with COPD have a significant higher aoPWV ($p = 0.002$), and cSBP ($p = 0.022$).

Conclusion: In this study we found elevated inflammatory markers and aoPWV in COPD patients, both of them indicate the presence of earlier atherosclerosis than in controls without COPD.

7.9

CAROTID ARTERY STIFFNESS IS ASSOCIATED WITH CT-MEASURED LUNG AIR-TRAPPING IN COPD PATIENTS AND CONTROLS INDEPENDENT OF AGE, BLOOD PRESSURE AND SMOKING HISTORY

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Background: Early stages of chronic obstructive pulmonary disease (COPD) are characterized by loss of the terminal bronchioles and 'air trapping' often before overt emphysema manifests (1). COPD patients are also at risk for cardiovascular disease (CVD), therefore, we hypothesized that the degree of air trapping on computed tomography (CT) (2) would be associated with higher aortic (carotid femoral pulse wave velocity, CFPWV) and carotid artery stiffness (β -stiffness), biomarkers of CVD risk.

Methods: Ten adults with COPD but little emphysema (age 66 ± 8 yrs, 5F/5M, GOLD stage 1-3) and 9 adults without COPD (age 59 ± 13 yrs, 5F/4M) that had a research chest CT were recruited.

Results: COPD patients had greater smoking history (45.9 ± 21 vs. 6.4 ± 12.9 pack-years, $P < 0.001$) and air trapping (0.85 ± 0.07 vs. 0.78 ± 0.05 Expiration/Inspiration attenuation ratio, $p < 0.05$) (2) compared with non-COPD subjects, but did not differ by age, BMI, SaO₂%, brachial BP or % emphysema (all $p > 0.05$). COPD patients had significantly higher CFPWV (999 ± 293 vs. 760 ± 147 cm/sec, $p < 0.05$) but not carotid β -stiffness (13.3 ± 5.1 vs. 10.6 ± 4.7 U, $p = 0.26$). In the entire cohort ($n = 19$), air trapping was associated with higher CFPWV ($r = 0.60$, $p < 0.01$) and carotid β -stiffness ($r = 0.75$, $p < 0.001$). After adjustment for age, mean BP and pack-years, the correlation between carotid β -stiffness and air-trapping remained significant ($r = 0.68$, $p < 0.01$).

Conclusions: Carotid artery stiffness is significantly associated with air trapping in COPD patients and controls, independent of age, smoking history and BP. This suggests a link between high CVD risk in COPD patients with small airway disease without predominant emphysema.

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7.10

AORTIC STIFFNESS AND BODY MASS INDEX (BMI) IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)

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Background: Patients with COPD have increased Cardiovascular (CV) risk and commonly present with altered body composition. Patients with COPD and a low BMI have poorer health outcomes¹, while obesity may increase CV risk². The aim of this analysis was to explore BMI, CV risk, exercise capacity and systemic inflammation in COPD.

Methods: This analysis included 524 stable patients with COPD (confirmed with spirometry) from the ARCADE (Assessment of Risk in Chronic Airways Disease Evaluation) study. Assessments included lung function (forced expiratory volume in 1 second (FEV₁)), smoking history, BMI, aortic pulse wave velocity (PWV) (SphygmoCor device), blood pressure (BP), 6-minute walking distance (6MWD). Inflammation was measured by high sensitivity C-reactive protein (hsCRP) and fibrinogen. Patients were classified by BMI as follows: low (< 19.9 Kg/m²), healthy (20-24.9 Kg/m²), overweight (25-29.9 Kg/m²) obese (> 30 Kg/m²).

Results: There was no difference in gender, age, lung function or smoking history between patients grouped according to BMI. However, there was a difference in PWV, systolic BP, 6MWD and inflammation between the groups ($p < 0.05$). The difference in PWV remained after adjustment for age and mean BP (Table 1). Overweight and obese patients (BMI < 25) had greater PWV and inflammation, while obese patients had the poorest 6MWD.

Conclusions: The findings suggest obese patients with COPD have greater CV risk which may be a result of poorer physical capacity and greater inflammation. Optimisation of BMI in COPD may improve outcomes further follow-up of this cohort will evaluate the prognostic value of arterial stiffness and possible therapeutic targets.