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Baseline deficiency of the anti-inflammatory eicosapentaenoic acid in cell membranes worsens lean body mass wasting induced by inactivity

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SUMMARY

Background & aims: Arachidonic (AA) and eicosapentaenoic (EPA) polyunsaturated fatty acids can play respectively a pro- and an anti-inflammatory role. We hypothesized that, at the end of 5-week experimental bed rest, baseline AA/EPA in red blood cells (RBC) membranes, considered the result of dietary fat intake over the previous month, could influence lean body mass wasting in twenty-six healthy volunteers (age: 23.5 ± 0.5 years; body mass index: 22.9 ± 0.5 kg/m^2).

Methods: We measured AA and EPA content in RBC membranes at baseline ambulatory conditions and at the end of the study protocol, to verify the PUFA concentrations stability. We assessed changes, between beginning and end of bed, in lean body mass (bioimpedance), insulin resistance (homeostasis model assessment), systemic inflammation (C-reactive protein) and oxidative stress (thiobarbituric acid reactive substances). Volunteers were divided in two groups according to the AA/EPA ratio median value (i.e. AA/EPA = 44): High AA/EPA group (60 ± 3; $n = 13$) and Low AA/EPA group (37 ± 1; $n = 13$).

Results: At baseline, all analyzed anthropometrical and biochemical indices were similar in the two groups. Bed rest induced a major decrease in lean body mass in High AA/EPA group (−5.2 ± 0.5%), when compared to Low AA/EPA group (−3.7 ± 0.5%; $p = 0.03$; ANOVA). Bed rest mediated-changes of insulin resistance, fat mass, systemic inflammation and oxidative stress, failed to

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show significant interaction with baseline AA/EPA (ANOVA). In pooled data, baseline AA/EPA ratio and percent lean body mass delta changes showed a significant inverse correlation ($n = 26$; $R = -0.50$; $p < 0.01$).

Conclusions: Results suggest that baseline AA/EPA, in RBC membranes, can independently predict lean body mass wasting in immobilized subjects during long term disuse.

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1. Introduction

Sarcopenia affects quality of life and outcome in elderly subjects and in chronically ill patients [1]. Reliable clinical markers able to predict the level of muscle atrophy progression are presently lacking. Physical inactivity and inflammatory response are key factors inducing muscle wasting in different conditions [2]. Inflammation, in particular, is known to trigger muscle wasting in many acute and chronic diseases, as well as during aging and physical inactivity [3,4]. Onset, progression and amplification of inflammatory response rely on metabolites derived either from the proinflammatory n-6 polyunsaturated fatty acid (PUFA) (arachidonic acid, AA), or from the anti-inflammatory n-3 PUFA (docosahexaenoic acid and eicosapentaenoic acid, EPA) [5]. Therefore, the ratio between n-3 PUFA and n-6 PUFA is considered to influence the inflammatory response, in several clinical conditions [6]. An elevated availability of n-6 PUFA in cell membrane has been shown to correlate to inflammatory diseases [6] and to activation of Nuclear Factor-Kappa B, a transcriptional factor controlling protein degradation, through the proteasome-system [7]. On the other side, EPA is known to efficiently inhibit eicosanoids production, thus reducing inflammation and its consequences [8]. PUFA availability and type depends is diet-dependent [9]. Some dietary fats, such as vegetable oils and margarines, contain elevate fractions of n-6 PUFA, while foods such as fatty fish, fish oil and nuts are sources of n-3 PUFA [9]. Recently it has been suggested that n-3 PUFA can improve the protein anabolism in many physiological and pathological conditions [10]. We hypothesized that skeletal muscle catabolism, induced by inactivity or by other conditions associated with sarcopenia, could also be influenced by the AA/EPA ratio. Experimental bed rest is a suitable model to investigate muscle atrophy progression in controlled conditions [11–13]. We measured the body composition changes (bioimpedance analysis) in healthy volunteers before and after 35 days of experimental bed rest and compared them to the AA/EPA ratios, measured on red blood cells (RBC) membranes. Diet composition of the month before the study protocol, assessed in each subject at enrollment, was maintained throughout the study. Insulin resistance (homeostasis model assessment, HOMA), systemic inflammation (high sensible C-reactive protein, high-CRP) and oxidative stress (thiobarbituric acid reactive substances, TBARS) were also investigated.

2. Methods

Thirty healthy young male volunteers were enrolled to participate to three separated 35-day bed rest studies, each including 10 participants. The experiments were conducted at the Valdoltra Hospital (University of Primorska, Ankaran-Capodistria, Slovenia) in the periods of July–August 2006, 2007 and 2008. The project was approved by the ethical committee of the University of Ljubljana and the experimental protocol is in accordance to the Declaration of Helsinki and its amendments (2002). A written informed consent was obtained by each volunteer upon enrollment. All subjects were physically active before admission to the hospital, none of them was under medication and their body weight and diets had been stable during the previous month.

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