Oral administration of EPA-rich oil delayed the initial stages of wound healing due to anti-inflammatory effect of Interleukin-10.

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Abstract

The aim of this study was investigate the effects of EPA-rich oil on wound healing and on macrophage cell functions, isolated from mice.

Key words:

Wound healing, Eicosapentaenoic fatty acids, Immune cells

Introduction

Poor wound healing affects over six million people around the world and lead to suffer and high costs of care¹. Considering the immune system as one actor in wound healing we investigated the influence of omega-3 fatty acid (EPA) in this process.

Results and Discussion

Statistical analyses: Values were express as mean±SD and (***) p< 0,001; (**) p< 0,01; (*) p< 0,05 indicates significant differences in relation to control.

Figure 1. EPA-rich oil impaired the inflammatory and proliferative phases of wound healing process. (a) Wound area percentages of control mice (●) or mice supplemented with 2g/Kg of EPA-rich oil (▲). (b) Representative photos. Used 5 to 12 animals per group. Statistical analyses by two-way analysis of variance (ANOVA) and Bonferroni posttest

Figure 2. EPA-rich oil decreased lymphocytes and M1 macrophages while increased M2 macrophages 3 days after wounding, however increased lymphocytes 10 days after wounding. (a) Percentage of markers in relation to control, 3 days after wound. (b) Representative dot plots, 3 days after wound. (c) Percentage of markers in relation to control, 10 days after wound. Used 4 to 8 animals per group. Statistical analyses by t test and Mann Whitney posttest.

Figure 3. EPA treated mice presented a biphasic effect on cytokines production. Concentrations of cytokines in scar tissue harvested 3, 7 and 10 days after wounding. Used 5 to 12 animals per group. Statistical analyses by t test and Mann Whitney posttest.

Figure 4. IL-10 delayed de initial stages of wound healing on mice supplemented with EPA-rich oil (a) Wound area percentages 3 days after lesion. (b) Representative photos. Used 4 to 6 animals per group. Statistical analyses by one-way analysis of variance (ANOVA) and Bonferroni posttest.

Figure 5. EPA induced anti-inflammatory effect on cytokines production by peritoneal macrophages. Concentrations of cytokines in supernatant of macrophages. Used 4 to 6 animals per group. Statistical analyses by two-way analysis of variance (ANOVA) and Bonferroni posttests.

Conclusions

In conclusion, in mice, the oral administration of EPA-rich oil impaired the inflammatory phases of wound healing due to anti-inflammatory effect of EPA.

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