Oral administration of EPA-rich oil induces anti-inflammatory effects in initial stages of wound healing in mice.

Beatriz Burger*, Carolina M. C. Khul, Mariah B. P. dos Anjos, Thamiris Candreva, Hosana G. Rodrigues

Abstract

The aim of this study was to determine the effects of EPA-rich oil ingestion on wound healing in mice.

Key words: fatty acids, cytokines, macrophages

Introduction

Ômega-3 (ω -3) fatty acids modulate the immune system¹. It is known that in tissue repair, inflammation plays an important role. In inflammatory state, macrophages recognize and phagocyte pathogens; interact with T cells; secrete cytokines and recruit other immune cells². The aim of this study was investigate the effects of oral administration of EPA-rich oil on wound healing and, on macrophage isolated from mice.

Results and Discussion

1. Methodology

2. Results and Discussion

2.1. EPA-rich oil impairs the inflammatory and proliferative phases of wound healing process

Figure 1. Macroscopic wound closure of control mice (C, black line) or EPA treated mice (EPA, green line). Values are expressed as mean $\pm$ SEM. (*) $p < 0,05$ indicate significant differences in relation to control as indicated by two way analysis of variance (ANOVA) and Bonferroni test.

2.2. EPA-treated mice presented a biphasic effect on cytokines production

Figure 2. Profile of cytokines in wound tissue isolated from EPA supplemented animals.

Oral supplementation with EPA-rich oil significantly reduced TNF- α , increased IL-10 and sIL-6R α production by macrophages (**Figure 3**). No significant difference in KC, IL-1 β , IL-6, VEGF and MIP-2 concentrations were observed between control and EPA groups.

Figure 3. Concentrations of cytokines in supernatant of macrophages isolated from control mice (C, black bar) and EPA treated mice (EPA, green bar). Values are expressed as mean $\pm$ SEM. (*) $p < 0,05$ indicates significant differences in relation to control as indicated by test t and Mann Whitney test.

2.3. EPA increased nitric oxide (NO) and reactive oxygen species (ROS) production by macrophages

Figure 4. Nitric oxide and superoxide anion (O $_2^{\bullet-}$) production by macrophages isolated from control mice (C, black bar) and mice supplemented daily with oil rich in EPA (EPA, green bar). Values are expressed as mean $\pm$ SEM. (*) $p < 0,05$ versus control as indicated by two way analysis of variance (ANOVA) and Bonferroni test.

Conclusions

Thereby, in mice, the oral administration of EPA-rich oil impaired the inflammatory and proliferative phases of wound healing and the closing of lesion, due to anti-inflammatory effect of EPA on macrophage functions.

Acknowledgement

FAPESP, CNPq, CAPES and FAEPEX/PRP/UNICAMP

References:

- Calder PC. Biochim Biophys Acta., v. 1851, n. 4, p. 469-84, 2015.
- Norling LV et al. J Immunol. v. 186, n. 10, p. 5543-7, 2011.