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Prevention of Damage to the Cholinergic Pathway in Bone Marrow Cells After Short-term Exposure to High Fat Diet: the Effect of Supplementation with Omega-3 Fatty Acid (EPA and DHA).

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Abstract

Data from our group show that animals that consumed a short-term high fat diet (HFD) showed reduction of α7nAChR receptor expression in bone marrow cells, so our objective is to evaluate if supplementation with omega3 polyunsaturated fatty acids can prevent the damage caused by the consumption of high fat diet in bone-marrow cells. Swiss male mice were randomly assigned to be previously supplemented with omega3 fatty acid for 17 days and consumed HFD for 3 days or HFD for 3 days without supplementation or normal chow diet, whithout supplementation. The protein content of α7nAChR was decreased in animals fed with HFD, and there was a tendency to increase when supplemented with omega3. However, there were no significant differences between the groups. Although we do not find significant results in the basal model, omega3 supplementation may be promising in models of induced sepsis.

Key words: *bone-marrow, omega-3, α7nAChR .*

Introduction

Activation of α7nAChR receptor reduces the production of proinflammatory cytokines leading to a reduction in inflammation and an increase in survival in a mouse model of sepsis[2,3]. Data from our group show that animals that consumed a short-term high fat diet (HFD) showed reduction of α7nAChR receptor expression in bone marrow cells. Omega3 polyunsaturated fatty acids have an important anti-inflammatory role through the interaction with GPR-120 type receptors located in the membrane of cells.

Our objective is to evaluate if supplementation with Omega3 polyunsaturated fatty acids can prevent the damage caused by the consumption of high fat diet in bone-marrow cells.

Results and Discussion

Swiss male mice were randomly assigned to be previously supplemented with omega3 fatty acid for 17 days and consumed HFD for 3 days or HFD for 3 days without supplementation or normal chow diet, whithout supplementation. Body weight gain was evaluated during the supplementation period. After the isolation of bone marrow cells, the number of cells obtained was counted and the expression of α7nAChR and proinflammatory cytokines was evaluated.

There was no difference in body weight gain and in the total number of cells isolated from the bone-marrow, This result shows that the HFD for 3 days was not able to change the body composition of the mices.

The protein content of α7nAChR was decreased in animals fed with HFD, and there was a tendency to increase when supplemented with omega3, however, there were no significant differences between the groups. This may be explained by the low rate of inflammation characteristic of the short time exposure of the diet, which would not be able to modulate inflammatory markers significantly[1].The protein content of p-NF-κB was descreased in animals fed with HFD, and in animals fed with HFD and supplemented with omega3, suggesting that omega3 was not able to reverse NF-κB activation.

Regarding the content of mRNA for the nicotinic receptor α7, the group that consumed HFD showed a significant reduction in the amount of mRNA for the receptor when compared to the control group. The supplementation with omega-3 fatty acid was no able to prevent the reduction in receptor expression induced by the consumption of HFD. We also did not observe a significant difference in the amount of mRNA for the chemokine CCL2 between the groups, this can be explained because the inflammation caused by the HFD for three days is low grade[1].

To obtain more information on the influence of omega3 fatty acid supplementation, we used the Immunofluorescence technique and analyzed the total number of cells expressing the α7nAChR receptor. We note that although we did not find statistically significant differences, a decrease in the receptor expression was observed in the cells of the animals that consumed the HFD, when compared to the control, and a tendency of recovery of this disease in animals that consumed the HFD, but which were supplemented with omega 3 fatty acid. We believe that the result was not statistically significant due to the low characteristic degree of dietary exposure, which would not be able to modulate inflammatory markers significantly[1].

Conclusions

Although we do not find significant results in the basal model, omega3 supplementation may be promising in models of induced sepsis.

Acknowledgement

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