Influence of microencapsulated probiotic intake on myeloperoxidase activity in TNBS-induced colitis in rats

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Introduction: The hypothesis that the intestinal bacterial flora contributes to the pathogenesis of inflammatory bowel disease (IBD) has been supported by experimental and clinical evidence [1]. The dysbiosis present in this condition is related to dysregulation of mucosal immune response. One of the indicators of leukocyte infiltration at the sites of inflammation is the activity of myeloperoxidase (MPO) [2]. Numerous studies have been conducted in order to examine the effects of probiotic intake in IBD. However, during ingestion of probiotics, the harsh conditions which are present in the gastrointestinal (GI) tract often impair the delivery of viable microorganisms in the lower intestine. For this reason, probiotic (*Lactobacillus casei 01*) was incorporated in Ca-alginate-microparticles coated with whey protein [3] and the effects of the formulation were examined in rat model of TNBS (trinitrobenzene sulfonic acid) - derived colitis.
The objective of this work was to examine the lower intestine MPO activity after induction of TNBS colitis in rats, and to compare the effects of ingestion of microparticulate probiotic formulation *vs* non-encapsulated probiotic.

Materials and methods: The effect on MPO activity was assessed after oral administration of the microparticulate *L. casei* formulation (once daily during 21 days; probiotic viability 8,7 $\log_{10}$cfu/g) to Wistar rats in which inflammation was induced by intrarectal administration of TNBS (10 mg in 0.25 ml 50% ethanol). For comparison, a group of Wistar rats received the same amount of non-encapsulated *L.casei* (8,7 $\log_{10}$cfu/g). At the same time, a negative and a positive (TNBS) control group were also tested. The MPO activity was measured as described by Peran et al., 2007 [4]..

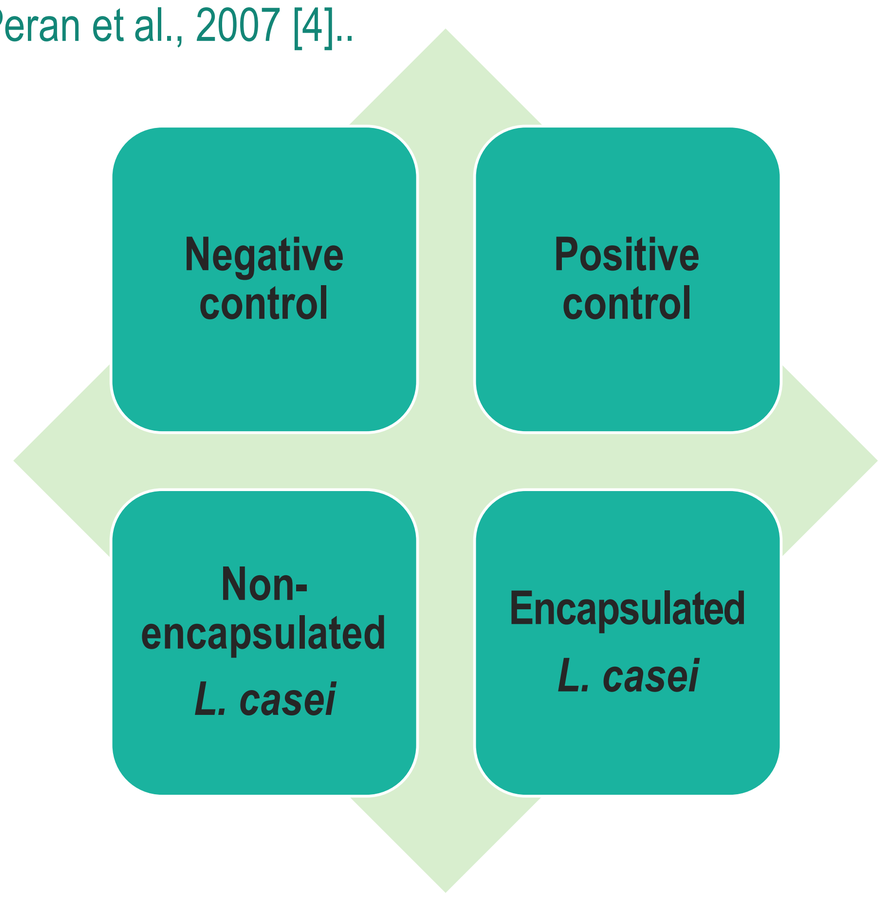

Fig. 1: Groups of treated Wistar rats that were examined for MPO activity.

Results and discussion: The obtained values of MPO confirmed the presence of inflammation in TNBS rat model of colitis, with the highest activity noted in the positive (TNBS) control group as was initially expected, due to the known effect of this hapten-induced inflammation. The activity of MPO was found to be lower in the group of rats that were administered a microparticulate probiotic formulation, in comparison to the group that was administered non-encapsulated probiotic. These results suggest that encapsulation of *L. casei* efficiently protects the probiotic during the GI transit, therefore resulting in better colonization of the lower intestine, which subsequently results in lower MPO activity.

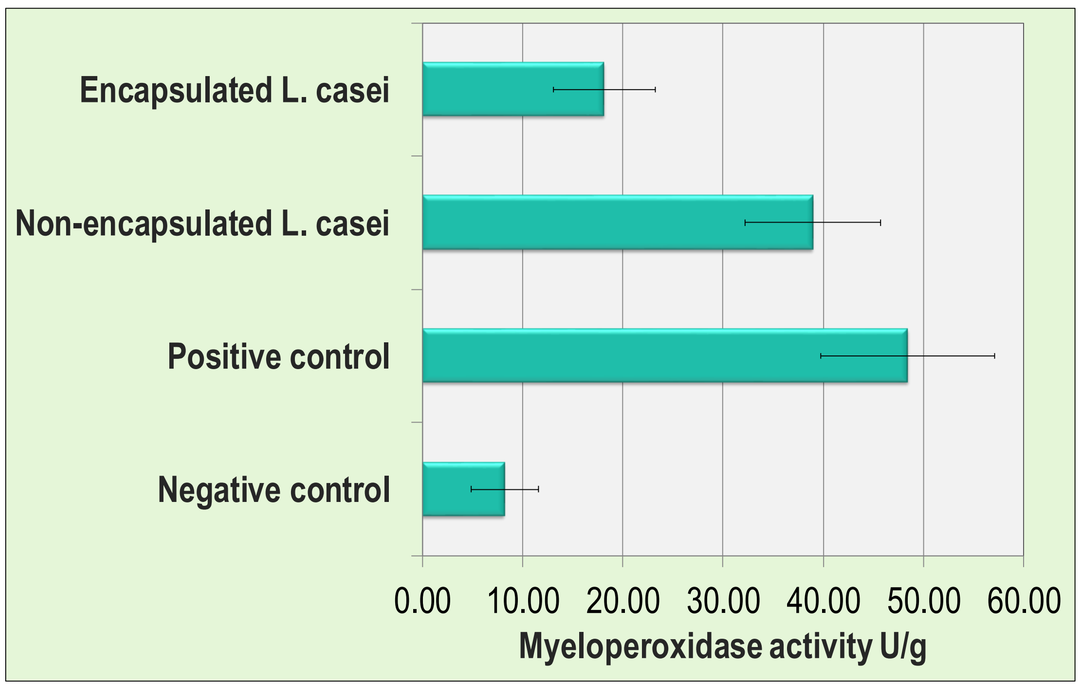

Fig. 2: Levels of MPO (U/g tissue) in the examined groups of rats.

CONCLUSION

Although the antioxidative influence of probiotics to cells is still to be explored, the role of probiotic molecules to reactive oxygen species is rather unraveled. Having the survival of probiotics a major issue to address, our results showed that encapsulation might be a strategy that leads to better outcome of probiotic action, since we observed lowered MPO activity in our encapusulated probiotic formulation in this model of colitis in rats. As other object of further research, other indicators of gut wall immune response should be examined in order to confirm and support the current finding that microencapsulated probiotic confer better effects than non-encapsulated one.

References

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