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Dear Colleagues,

In this issue we look at several aspects linked to the possible role of vitamin D in gastroenterology, with the usual contributions of experts. The question of a potential interaction between vitamin D and intestinal microbiota is complex and still for the most part uncertain – and therefore fascinating – especially in cases of qualitative and quantitative alterations of the latter.

In the context of the physiology of the intestinal absorption of vitamin D, we must recall that any anatomical or functional alteration impacting the digestive tract can have an effect on microbiota and vitamin D status. On the other hand, given vitamin D's recognised immunomodulatory role, we cannot exclude that the role attributed to microbiota in the pathogenesis of many inflammatory bowel diseases (IBDs) is at least in part caused by a changed local availability of vitamin D.

As we shall see, many studies have assessed the effects of vitamin D on intestinal microbiota, in particular – though not exclusively – in connection with IBDs: in this case, an association between vitamin D deficiency and disease activity, risk of relapse and failure of therapy has been documented.

At the same time, significant effects of microbiota on vitamin D have been predicted and described. We need only recall the possible consequences of qualitative and/or quantitative modifications of intestinal microbiota on vitamin D absorption, which are secondary to, for example, hypochylia, alterations in intestinal motility or the administration of probiotics. Intestinal dysbiosis also seems to be involved in the pathogenesis of non-alcoholic fatty liver disease, (NAFLD). In the last few years, many epidemiological studies have reported that patients affected by NAFLD have significantly lower circulating 25(OH)D levels compared to control populations.

Low vitamin D₃ levels have also been linked to increased histological severity of NAFLD. Although the aetiopathogenesis of the mechanisms that can account for this association are still not clear, it has been proposed that vitamin D₃ can have important hepatoprotective effects. In particular, *in vitro* studies have shown that vitamin D₃ is able to positively modulate insulin signalling (by improving insulin resistance at the hepatic level as well) and reduce the proliferation of fibroblasts and collagen production. To date, however, the literature has not provided us with broad prospective cohort studies or ample randomised clinical studies which have assessed a possible correlation between circulating vitamin D₃ levels and the risk of developing or aggravating NAFLD. Such studies are necessary in order to confirm the biological plausibility and possible causal role of vitamin D₃ in the development and progression of NAFLD.

Nonetheless, as you will read below, the recently published results of prospective cohort and Mendelian randomization studies effectively suggest that maintaining sufficient 25(OH)D levels may constitute an efficient approach in the primary and secondary prevention of NAFLD.

In addition, let me point out that we should begin considering NAFLD as one of the pathologies that causes secondary osteoporosis. This finding has emerged from a meta-analysis that was recently published in *Osteoporosis International*¹, which reports a significant correlation between

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NAFLD and the prevalence and risk of osteoporosis and fractures, especially in males. This represents another reason to consider administering vitamin D to these patients. What do you think?

Bibliography

- ¹ Pan B, Cai J, Zhao P, Liu J, Fu S, Jing G, Niu Q, Li Q. Relationship between prevalence and risk of osteoporosis or osteoporotic fracture with non-alcoholic fatty

liver disease: A systematic review and meta-analysis. *Osteoporos Int.* 2022 Nov;33(11):2275-2286. <https://doi.org/10.1007/s00198-022-06459-y>