Jia et al.\(^1\) report that dietary fish oil or curcumin, given alone, promotes mucosal inflammation and damage, whereas, given together, resolution of inflammation was promoted and major inflammatory factors, especially NF-κB, were suppressed. These findings are suggested to reflect an interaction of dietary lipids and curcumin in mucosal homeostasis and in the control of colonic inflammation through the suppression of PG release in colonic mucosa by both compounds through the Toll-like receptor 4/cyclo-oxygenase-2/PGE signalling axis. However, these effects might also arise through the actions of these two compounds on vitamin D signalling through vitamin D receptors (VDR), since both bind to the VDR\(^2\). DHA competes with calcitriol for binding to the VDR, as do other PUFA and biliary lithocholic acid, which may tend to reduce VDR signalling activity, while curcumin can have calcitriol-like effects since its binding to the VDR induces some genomic effects of the activated VDR and it can also induce rapid non-genomic VDR-mediated effects such as activation of chloride channels\(^2\). Vitamin D reduces inflammation generally, including in the colonic mucosa, and is activated in the gut as in most target tissues\(^3,4\). Furthermore, pharmacological preparations of fish oil contain small amounts of vitamin D but large amounts of vitamin A, which antagonises many actions of vitamin D. For example, vitamin A reduces vitamin D-induced absorption of dietary Ca\(^5\). It would be of interest to know whether the reported effects and interactions of fish oil and curcumin on colonic inflammation are related to variations in vitamin D intake (or to vitamin D repletion as judged by serum 25-hydroxyvitamin D concentration) or to variations in vitamin A intake, or both.

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