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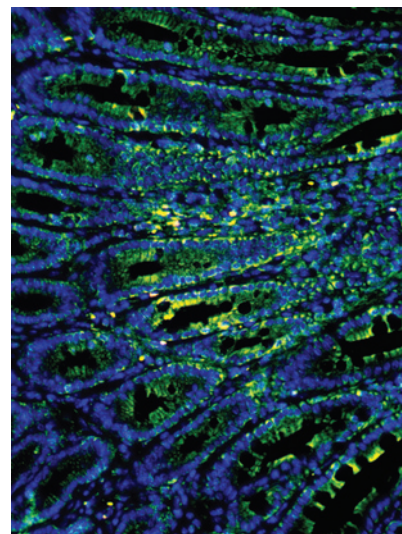
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ABOUT THE COVER

Infection of macrophages by the human intestinal commensal *Enterococcus faecalis* generates DNA damage and chromosomal instability in mammalian cells and is mediated, in part, by *trans*-4-hydroxy-2-nonenal (4-HNE). In this study, the role of cyclooxygenase (COX) and lipoxygenase (LOX) in producing this reactive aldehyde was explored using *E. faecalis*-infected macrophages and interleukin-10 knockout mice colonized with this commensal. The cover micrograph shows immunofluorescence staining of colon sections from *Il10*^{-/-} mice colonized with *E. faecalis*. There is focal 4-HNE-protein adduct (green) staining in macrophages (merged: yellow) and diffuse staining on colonic crypts (green). This was associated with increased staining for COX-2 in macrophages when compared to sham-colonized mice (not shown). DNA is counter-stained using DAPI (blue). These data show that *E. faecalis* can trigger macrophages to produce 4-HNE through COX-2 reinforcing the concept of COX-2 as a procarcinogenic enzyme capable of damaging DNA in target cells through bystander effects that contribute to colorectal carcinogenesis. See the article by Wang et al. (beginning on page 206) for more information.



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