Comment on "The Bacterial Fermentation Product Butyrate Influences Epithelial Signaling via Reactive Oxygen Species-Mediated Changes in Cullin-1 Neddylation"

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We refer to the important article by Kumar et al. (1) This report links the inhibition of ubiquitin ligase and the suppression of NF-κB in intestinal epithelial cells to fatty acid (FA)/reactive oxygen species (ROS) production, but it is unclear whether this process is sufficient to cause clinical disease. We found that an analogous E3 suppression mechanism, possibly FA/ROS induced, plays a role in human epithelial inflammation in vivo in a similar mucus-producing, goblet cell-containing, stratified squamous epithelium (2).

In sun-exposed people, a fibrovascular proliferative condition called pterygium frequently occurs (3). Our studies showed that the ubiquitin ligases UbE3C and UbE3B and the ubiquitin conjugase UbE2K were down-regulated in pterygium epithelia compared with uninvolved conjunctiva epithelia from the same conjugase UbE2K were down-regulated in pterygium epithelia the ubiquitin ligases UbE3C and UbE3B and the ubiquitin

FIGURE 1. A, Immunohistochemistry on conjunctiva and pterygium tissues using primary Abs against various ubiquitin-modifying enzymes (green) and nuclear staining with 4,6-diamidino-2-phenylindole (blue). B, Western blots against selected E3 ligases. Scale bar, 50 μm.

References