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Basolateral Secretion of CXC Chemokines by Human Intestinal Epithelial Cells in Response to *Bacteroides fragilis* Enterotoxin Via NF-κB Pathway

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Enterotoxigenic *B. fragilis*, which produces a 20 kDa heat-labile toxin (BFT), has been associated with diarrheal diseases and mucosal inflammation. To test whether epithelial cells are involved in BFT-induced inflammation, we assessed the expression of CXC chemokines by BFT-stimulated human intestinal epithelial cells. BFT stimulation increased the expression of the neutrophil chemoattractant and activators ENA-78, GRO-a, and IL-8. Up-regulated mRNA expression of chemokines was paralleled by increased protein levels. Although lactate dehydrogenase used to monitor cell lysis was released predominantly from the apical surface, CXC chemokines were predominantly secreted from the basolateral surface of BFT-treated polarized Caco-2 epithelial cells. Moreover, BFT stimulation activated NF-κB in HT-29 epithelial cells. The activation of IL-8 and NF-κB transcriptional reporters was inhibited in the HT-29 cells cotransfected with the IκB kinase a and IκBa superrepressor plasmids. Our results indicate that CXC chemokines can be basolaterally secreted from BFT-stimulated colon epithelial cells via NF-κB pathway, suggesting that these chemokines might contribute to the infiltration of inflammatory cells into the underlying intestinal mucosa.