
Abstract
Probiotics are widely used in the treatment and prevention of gastrointestinal problems. However, in some immunocompromised populations, the administration of live microorganisms may not be appropriate. A potential alternative to live microorganisms is to inactivate them as long as the beneficial function is retained. We hypothesized that UV-inactivated Lactobacillus rhamnosus GG (LGG) could downregulate interleukin-8 (IL-8) production in intestinal epithelial cells stimulated by the pathogenic ligand, flagellin, using similar mechanisms as live LGG. Caco-2 cells were pretreated with live or UV-inactivated LGG at 10^11 colony-forming units/L and stimulated by flagellin at a dose of 500 mg/L. IL-8 production was measured by ELISA, inhibitor of kB (IkB) and ubiquitinated-IkB (Ub-IkB) expression by immunoblotting and nuclear factor (NF) kB localization by immunofluorescence staining. Flagellin induced a 17-fold increase in IL-8 production compared with control (P < 0.05), whereas pretreatment with either live LGG or UV-inactivated LGG resulted in 66 and 59% decreases, respectively, compared with the flagellin group (P < 0.05). Flagellin-induced NFkB nuclear translocation was prevented by both live and UV-inactivated LGG. Flagellin decreased IkB, which was reversed by either live or UV-inactivated LGG (P < 0.05). UV-inactivated LGG decreased Ub-IkB expression (P < 0.05), although live LGG had no effect. This study supports the concept that UV-inactivated and live LGG are equally effective in decreasing IL-8 production in the intestinal epithelium. Although the mechanism involves different pathways, both alter cytoplasmic IkB, thereby inhibiting NFkB nuclear translocation.