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Antimicrobial Triclosan (TCS) Increases Production of Interleukin 1 beta (IL-1β) in Human Immune Cells

ABSTRACT

Triclosan (TCS) is an antimicrobial compound that is widely used in personal hygiene products such as mouthwash and toothpaste. TCS can be ingested or absorbed through the skin and has been found in human blood, breast milk, and urine. Interleukin-1 beta (IL-1β) is an important pro- inflammatory cytokine produced by immune cells such as monocytes and lymphocytes, and plays a critical role in immune response regulation, tissue repair, and cellular growth. Overproduction of IL-1β can contribute to chronic inflammation and inflammatory diseases such as rheumatoid arthritis and multiple sclerosis. IL-1β has also been shown to stimulate tumor development. In a previous study, we showed that TCS at concentrations between 0.05-5 μM increased the secretion of IL-1β from immune cells within 24 h. Here we will examine whether the increase in IL-1 β secretion stimulated by TCS is due to an increase in the cellular production of IL-1β or only due to release of existing stores of the cytokine. Human peripheral blood mononuclear cells (PBMCs) were exposed to TCS at concentrations of 0-5 μM. The cellular production (combination of secreted and intracellular levels) of IL-1β was measured at 10 minutes, 30 minutes, 6 hours, and 24 hours. Secreted levels were measured in supernatants from exposed cells using enzyme-linked immunosorbent assay (ELISA) and intracellular levels were measured by lysing the exposed cell pellet followed by Western Blot. Results indicate the production of IL-1β was increased by exposure to one or more concentration of TCS at each length of exposure. The greatest increase in IL-1β production was seen at 6 h, where all TCS exposures caused quite substantial increases in IL-1β production in all donors. The magnitude of these increases varied dependent on the donor. These results indicate that TCS has the capacity to increase cellular production of the important pro-inflammatory cytokine IL-1β from immune cells. TCS-induced increases in IL-1 β production may serve to produce unwarranted inflammation and the pathologies associated with it