Journal Abstract

Bifenthrin activates homotypic aggregation in human T-cell lines.

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Background: Here, we addressed the concern that, despite the lack of overttoxicity, exposure to low levels of the common household pyrethroid pesticide, bifenthrin, could causeharm to the immune system. To do this, we measure the effect of bifenthrin on phytohemagglutinin (PHA)activation of homotypic aggregation in human T-cell lines. Material/Methods: The human CD4+ H9, and Jurkatcell lines and the human promonocyte U937 cell line, were exposed to varying concentrations of bifenthrin.Cell viability was determined using the AlmarBlue Toxicity Assay. Concentrations of bifenthrin whichdid not reduce cell viability were determined and these concentrations were tested for the effect ofbifenthrin on PHA-mediated homotypic aggregation. Blocking antibodies to ICAM and LFA-1 were used todisrupt aggregation and a nonspecific IgG was used as a control. Results: Bifenthrin was found to benontoxic at concentrations ranging from 10^{-4} to 10^{-13} M. Bifenthrin did not inhibit PHA induced cellaggregation in all cell lines tested. However, at 10^{-4} M, bifenthrin to form aggregates stimulatedhomotypic aggregation in the H9 and Jurkat T-cell lines. The bifenthrin-induced aggregate formation,like that seen with PHA, was blocked by treating the cells with antibodies to either LFA-1 or ICAM. Conclusions:The results here show that bifenthrin activates T-cell function by stimulating ICAM/LFA-1 mediated homotypicaggregation. This data suggests that exposure to bifenthrin, even at "acceptable" limits, can increasethe risk for and frequency of inflammatory responses and diseases such as asthma.

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