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A mycotoxin citrinin produced by *Penicillium citrinum* and *P. verrucosum*, mainly contaminates cereals. Previous studies demonstrated that citrinin exposure mainly exerted kidney toxicity but other adverse effects particularly against the immune system have to be examined. Therefore, this study was aimed to investigate the novel toxic effect of citrinin using a mouse model of psoriasis. A mouse model of psoriasis was generated by repetitive topical application of 5% imiquimod cream in female BALB/c mice. Standard rodent diet and rice samples with 3 ppm of citrinin were mixed to obtain a final citrinin concentration of 0.3 ppm, and citrinin contaminated diet was fed to mice daily. Skin thickness, scratching behavior, and trans epidermal water loss (TEWL) were monitored continuously during the imiquimod application. Immediately after the final imiquimod application, ear skin and auricular lymph node (LN) were sampled for further analysis. Only a slight increase was seen in skin thickness in the citrinin exposure group, however, citrinin exposure significantly exacerbated hyperkeratinization and inflammatory cell infiltration in histological evaluation. TEWL, which represents a condition of cutaneous barrier function, was significantly increased by citrinin exposure. In terms of immune function, the number of immune cells in LN (T cells and dendritic cells) and gene expression of IL-17 in skin tissue were significantly increased by citrinin exposure. Taken together, our results imply that oral exposure to citrinin exacerbates the symptoms of a mouse model of psoriasis.

P-109S **Influence of oral exposure
to citrinin on the
pathophysiology in an
imiquimod-induced mouse
model of psoriasis**

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