1. No.
   2024Y006

2. Title
   Elucidation of the Immunological Mechanism of Smoking in the Exacerbation of Psoriasis, an Inflammatory Keratotic Disease

3. Period
   2024-2025

4. Summary
   During the grant period, we investigated the immunological impact of smoking on the pathogenesis of psoriasis, an inflammatory keratotic skin disease. In the first year, we examined the effects of smoking-related substances using an imiquimod-induced murine psoriasis model. Two experimental smoking models were employed: a benzo[a]pyrene exposure model and a nicotine administration model. In these experiments, benzo[a]pyrene exposure did not exacerbate psoriatic skin inflammation. Similarly, nicotine administration did not increase the severity of skin thickening. Interestingly, high-dose nicotine administration was associated with a reduction in epidermal hyperplasia, suggesting that nicotine itself may not be the primary factor responsible for smoking-associated exacerbation of psoriasis. These findings indicate that other components of cigarette smoke may contribute to disease progression.

   Based on these findings, the second year of the study focused on lifestyle-related factors associated with psoriasis. Using an obesity-associated psoriasis model, we investigated the effects of aerobic exercise. The results suggested that aerobic exercise partially attenuated epidermal hyperplasia under obese conditions. Collectively, our findings indicate that psoriasis pathogenesis is influenced by multiple lifestyle-related factors, including smoking, obesity, and physical activity. Further studies are required to clarify how smoking-related components interact with metabolic and immunological pathways in psoriasis.