## P-52 SKIN AND ORAL VIROME IN PSORIASIS PATIENTS ON BIOLOGIC THERAPY

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The viral communities represent the most abundant microbiota population in humans. Viruses, together with other microbiota such as bacteria and fungi, form a dynamic ecosystem that influences the health of the host. The human microbiota is involved in the pathogenesis of psoriasis, a chronic skin inflammatory disorder that affect 2–4% population. Various not yet well-known environmental triggers, including infections, can initiate psoriasis; however, the molecular mechanisms of host-microbe interaction are still unknown (1). Psoriasis is now treated with newly discovered biologic drugs that have shown excellent efficacy. However, patients on biologics are at increased risk of infections (2). Patients using interleukin (IL)-17 blockers are at increased risk of mycotic and respiratory infections, patients treated with TNF-α inhibitors are at risk of reactivation of latent tuberculosis. However, information about the influence of other viruses and viral communities is limited or completely lacking. In our preliminary studies, we found higher prevalence of human papillomaviruses (HPV) in the oral cavity of patients treated with biologic therapy (anti-TNF-alpha, anti-IL-12/23, and anti-IL-17), compared to patients on topical treatment suggesting that the long-term immunosuppression increases the risk of oral HPV acquisition (3). The prevalence of Merkel cell polyomavirus was statistically significantly higher in the genital area of these patients (4). The following study will focus on a) virome analysis of lesional and non-lesional skin of psoriasis patients, b) oral virome analysis, c) influence of long-term treatment with biologics on virome changes. To conclude, our preliminary data show that even when traditional methods, that can detect only known sequences of a small number of targets, are used some alterations in the viral communities of patients on biologic therapy can be detected. The nextgeneration sequencing methods recently optimized in our laboratory will expand our knowledge, helping to identify patients at increased risk of viral reactivation and improve patients' management and therapeutic approaches.

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