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Pyoderma gangrenosum (PG) is a painful cutaneous ulceration, causing diagnostic and therapeutic challenges. Although immunosuppression is the first-line of treatment, the effect is disappointing; not even 50% of the patients have healed within 6 months. Traditionally, it is believed to be a neutrophilic dermatosis, and recent studies have also hypothesized that neutrophilic dermatosis could be assigned to the family of autoinflammatory diseases. However, the pathophysiology of PG is only loosely described and the trigger of the autoinflammation is unknown. Because the wound surface are often sterile and antibiotic treatments are ineffective, infection has in general been ruled out as the primary cause. We hypothesize that PG is 1. An infectious-autoinflammatory disease, where difficult-to-eradicate biofilms/bacteria creates an immunological overshoot. 2. A disease of the hair follicle (due to the lack of involvement of PG in certain regions: the nipple-areolar-complex, the palmoplantar skin and PG-scar tissue; sites lacking pilosebaceous units). The objective of the study is to characterize the microbiome and inflammation (on a molecular, cellular and clinical level), in patients with PG compared to venous leg ulcers. This is a prospective, hypothesis-generating descriptive investigation were wound fluid and biopsies are sampled for PNA-FISH, NGS, H&E-staining and multiplex assays.