LP is a papulosquamous dermatosis which has linked to infection with hepatitis C virus.

There has been increasing evidence that LP is significantly associated with liver disease, especially viral hepatitis.

The aetiology of LP is unknown but there are several theories involved in the pathogenesis, including immunological theory, lymphocytes particularly T-cells play a major role, other factors include antigen presenting cells, adhesion molecules and inflammatory cytokines, while most cases are idiopathic, some are linked to medication use or HCV infection.

HCV is known to replicate in hepatocytes and peripheral blood mononuclear cells. It is unclear whether this virus induces LP and if so, whether these lesions are produced by direct cutaneous infection or by perturbation of immune system systemically or focally.

The aim of this work is to insure whether or not viral antigen of HCV may be present in cutaneous lesions of LP.
This study was carried out on thirty patients diagnosed clinically as having cutaneous and/or oral LP. Serum analysis for HCV antibody had been done to all patients.

Cutaneous biopsies were taken and subjected to hematoxylin-eosin staining and immunohistochemical staining using immunoperoxidase.

Cytoplasmic staining brown granules in dermal histiocytes considered positive for HCV antigen. It was present in ten/thirty patients with LP and HCV-positive, but was absent from other eighteen/thirty patients with LP and HCV-negative.

Detection of HCV antigen in skin of patients with LP either cutaneous and/or oral, as evidence by cytoplasmic staining of dermal histiocytes using immunoperoxidase, is an indication of the role of HCV in the pathogenesis of some cases of LP and that, the infected cells were the target for, the host immune response as was evident by the lymphocytic infiltrate surrounding the infected cells.

OLP is the most common variety of LP associated with HCV infections.

Still the exact aetiology of LP is unknown and there are many factors involved in the pathogenesis of LP.