Revealing the damage being done in AD and PN$^{1-3}$

Learn more about the role of a neuroimmune cytokine in atopic dermatitis (AD) and prurigo nodularis (PN) by clicking below.
IL-31 is a central mediator of neuroimmune skin dysfunction

IL-31 plays a role in many inflammatory skin diseases, including AD and PN.\textsuperscript{1-3} IL-31 bridges the peripheral nervous system and immune system and targets structural skin cells, driving itch, inflammation, and skin abnormalities.\textsuperscript{1}

The center of neuroimmune skin dysfunction\textsuperscript{1}

Ready to dive deeper? Visit IL31role.com to watch videos that give you a closer look at the role of IL-31 in AD and PN.
In AD, there may be more damage than you think

Learn more about a central mediator of neuroimmune dysfunction that ignites and fuels a pathologic process characterized by itch, inflammation, and skin barrier dysfunction in patients with AD.¹,²

DISEASE BURDEN >
The signs and symptoms of AD are disrupting the lives of patients

AD is a common, chronic, inflammatory skin disease that is characterized by persistent itch, embarrassing eczematous lesions, and frequent skin infections.4,5

Persistent itch is much more than just an irritating sensation—it is the most burdensome symptom for patients with AD and can significantly disrupt their daily activities, sleep, and psychological well-being.5,6

79% of patients with AD and their caregivers reported that itch was among their top 3 most problematic symptoms5,*

AD is driven by both inflammatory and neural mechanisms

Current understanding of AD pathophysiology reveals that it is a neuroimmune-mediated disease, driven by2,7,8:

- Itch
- Inflammation
- Skin barrier dysfunction

*Results were based on an online 32-item survey of 1508 patients with AD and their caregivers from the United States and 57 other countries. Survey respondents were asked which 3 symptoms have been the most problematic for the patient with AD.5
IL-31 is at the center of AD pathogenesis

IL-31, a neuroimmune cytokine expressed in AD skin, is a central mediator that ignites and fuels the 3 key pathophysiologic drivers of the disease: itch, inflammation, and skin barrier dysfunction.\(^1,2,9\)

There are many cytokines involved in the pathogenesis of AD, including IL-4, IL-5, IL-13, and IL-31.\(^10,11\)

The effects of IL-31 in AD

IL-31 bridges the peripheral nervous system and immune system\(^1\)
- Stimulating peripheral sensory neurons to cause itch and dermal nerve elongation and branching
- Activating immune cells to release proinflammatory cytokines

Overexpressed IL-31 also targets structural skin cells\(^1\)
- Disrupting keratinocyte differentiation and decreasing the expression of key proteins, leading to skin barrier dysfunction

There are still unmet needs in AD

AD is a chronic and extremely heterogeneous disease. Despite currently available treatment options, some patients with AD are still experiencing adverse events and a lack of disease control.\(^12,13\)
In PN, there may be more damage than you think

Learn more about a central mediator of neuroimmune dysfunction that ignites and fuels a pathologic process characterized by itch, inflammation, and the formation of fibrotic tissue in the skin of patients with PN.¹²

Not an actual patient.
PN is a distinct, damaging skin disease

PN is a distinct, underrecognized, and chronic inflammatory skin disease that is characterized by disfiguring, often excoriated nodules and intractable itch.\textsuperscript{3,14,15} Although PN is sometimes associated with AD, most patients with PN do not have a history of AD.\textsuperscript{16-18}

PN affects an estimated 72 per 100,000 adults aged 18 to 64 years in the United States, primarily middle-aged women and disproportionately people of African descent.\textsuperscript{3,16}

Common diseases associated with PN

PN is associated with a variety of diseases, including\textsuperscript{3}:

- Hypertension
- Chronic kidney disease
- Type 2 diabetes
- HIV infection
- Obesity
The burden of PN is oppressive

PN severely impacts many aspects of patients’ lives—it leaves some suffering with intractable itch, disfiguring nodules, debilitating sleep disturbance, and impacts on their psychological well-being.19

Studies have shown that patients with PN experience a higher burden of disease than patients with several other chronic pruritic skin diseases. This may be due to the itch of PN being more severe than that of the other skin diseases.20

What’s driving PN?

A growing understanding of PN pathophysiology reveals that it is driven by neuroimmune dysfunction, which manifests as:

- Itch
- Inflammation
- Fibrotic tissue in the skin

49% of adults with PN reported that itch was the most burdensome symptom of their skin disease.17

*Results were based on a multicenter, cross-sectional European study of 509 adults with PN. This prospective, questionnaire-based study assessed the clinical profile of PN, as well as its associated burdens.17
IL-31 is at the center of PN pathogenesis

IL-31, a neuroimmune cytokine expressed in PN nodules, is a central mediator that ignites and fuels the 3 key pathophysiologic drivers of the disease: itch, inflammation, and the formation of fibrotic tissue.\(^1\),\(^3\)

There are many cytokines involved in the pathogenesis of PN, including IL-4, IL-13, IL-22, and IL-31.\(^1\),\(^3\),\(^21\),\(^22\)

The role of IL-31 in PN

IL-31 bridges the peripheral nervous system and immune system\(^1\)
- Stimulating peripheral sensory neurons to cause itch\(^1\),\(^3\),\(^23\)
- Activating immune cells to release proinflammatory cytokines\(^1\),\(^3\),\(^23\)

Overexpressed IL-31 also targets structural skin cells, including dermal fibroblasts\(^1\),\(^3\),\(^24\)
- Leading to inhibited cell proliferation and fibrotic remodeling

The unmet need in PN

It is difficult to treat itch in PN.\(^25\) Since there are currently no FDA- or EMA-approved therapies for PN, there is a need to relieve itch in affected patients.\(^3\),\(^25\),\(^26\) Many adults with PN identified the improvement of itch, nodules, and sleep as their top 3 most important treatment goals.\(^27\)*

*Results were based on a multicenter, cross-sectional European study of 509 patients with PN. This prospective, questionnaire-based study assessed patient perception of therapeutic goals, as well as previously used therapies, overall satisfaction with therapy, the efficacy of available therapeutic regimens, and out-of-pocket costs.\(^27\)
Consider the damage being done in AD and PN

The images in this material are not of actual patients with AD or PN. They were created based on informed Galderma insights.

AD and PN can manifest in individuals differently.

References:


©2022 Galderma Laboratories, L.P. All Rights Reserved.
All trademarks are the property of their respective owners.

US-NAD-2100001 March 2022