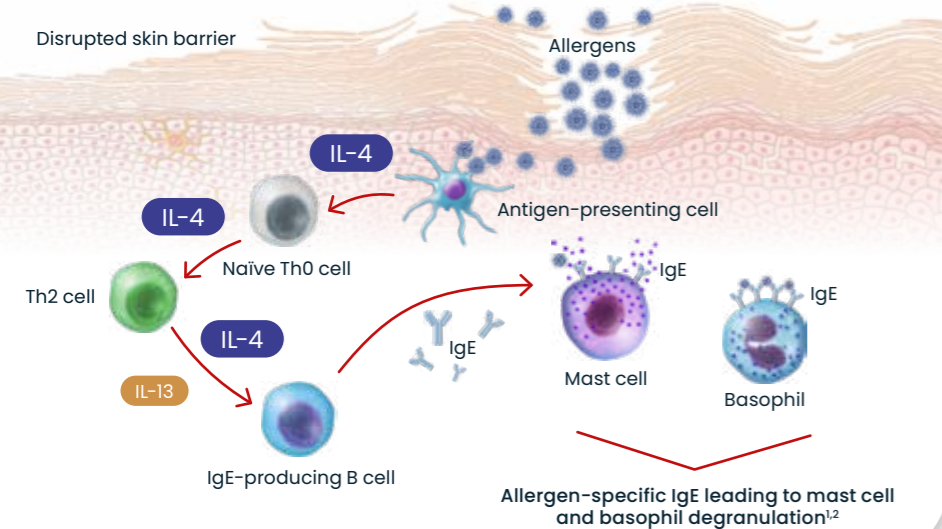




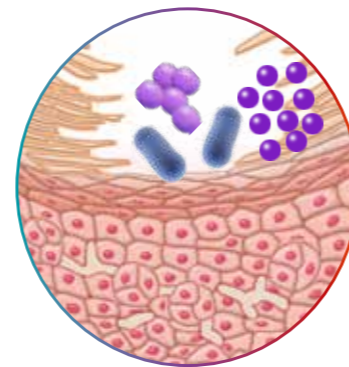
Skin Barrier Dysfunction and Increased Risk of Allergen Sensitization May Impact Atopic March in Atopic Dermatitis (AD)¹⁻⁴

Skin barrier dysfunction in Atopic Dermatitis increases the risk of allergen sensitization, dysbiosis, infection, and other type 2 inflammatory diseases¹⁻³

The dysfunctional skin barrier may allow transcutaneous entry of allergens and subsequent potential sensitization¹



Skin dysbiosis and increased risk of infections³



- Decreased microbial diversity
- Colonization with *S. aureus*

Increased risk of other type 2 inflammatory diseases⁴

Atopic dermatitis can follow multiple trajectories from childhood through to adulthood⁵

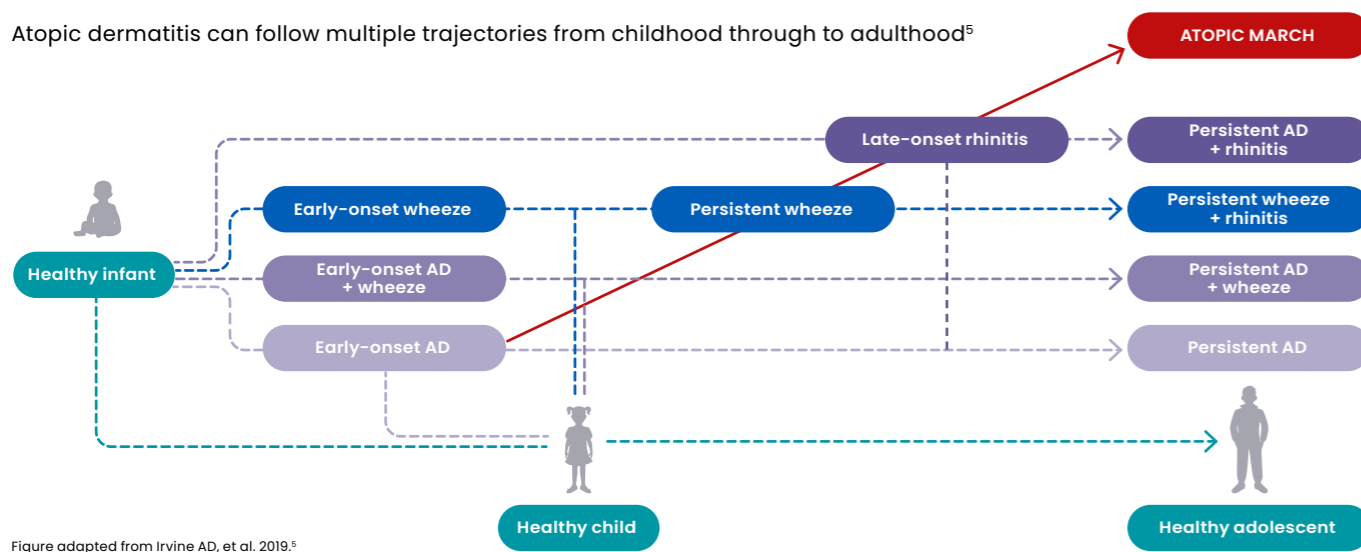


Figure adapted from Irvine AD, et al. 2019.⁵

Limiting exposure to allergens through the skin and controlling type 2 inflammation at an early age may help to prevent or reduce progression to coexisting type 2 inflammatory diseases in children with AD⁶



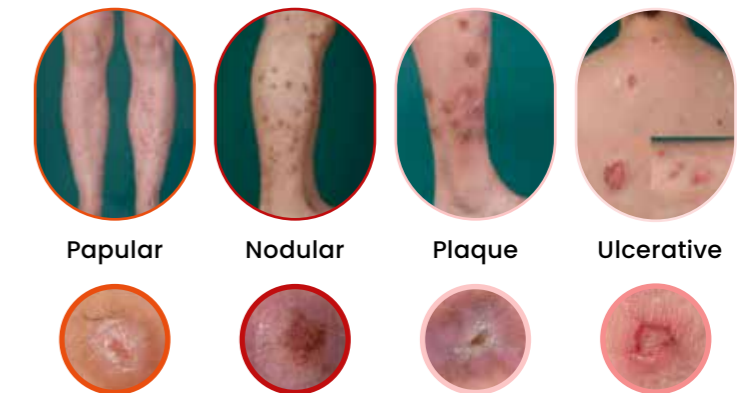
The Evolving Understanding of Prurigo Nodularis (PN) Has Implications for Patient Management

Prurigo Nodularis is a heterogenous, chronic inflammatory, pruritic disease with unique and varied patient characteristics, burden and clinical manifestations¹⁻⁶

PN patients may differ in their:¹⁻³

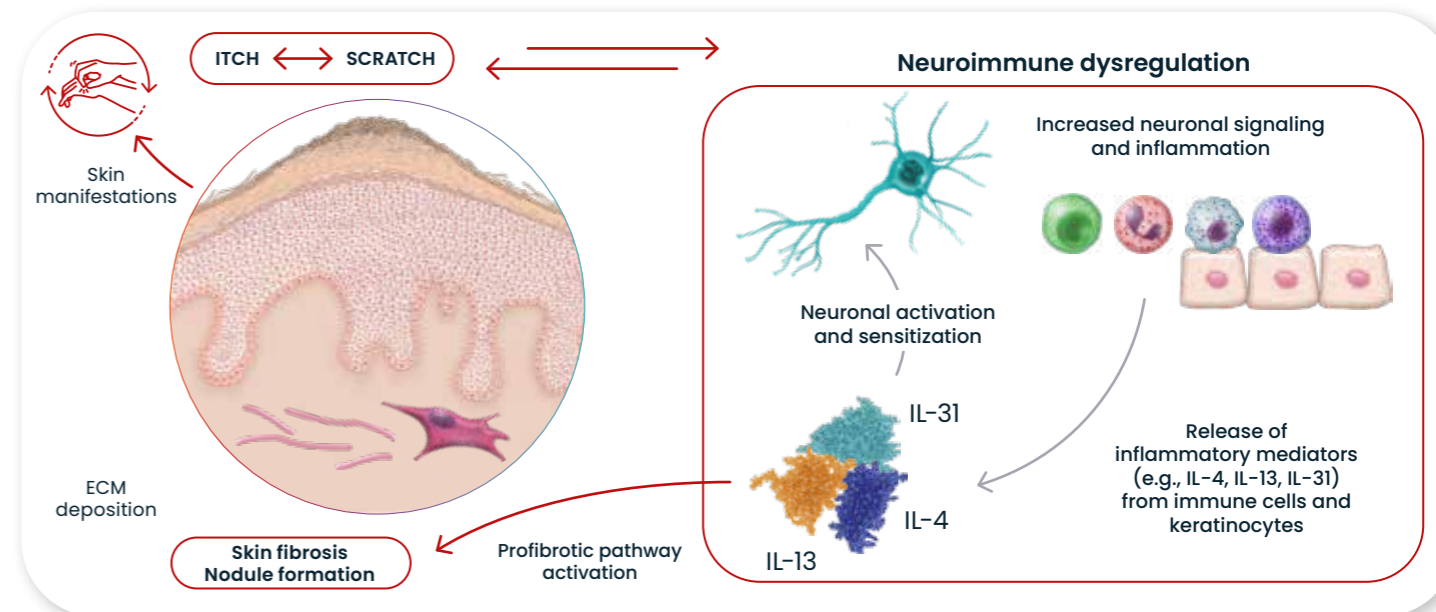
- Disease Etiology**
e.g., primary, idiopathic, neurological
- Treatment History**
e.g., topicals, gabapentinoids
- Lesion Size and Number**
A few mm to 2–3 cm, numbers can vary from a few to hundreds
- Comorbidities**
e.g., atopic, liver and kidney diseases

Varied clinical manifestations in PN include:⁴⁻⁶



Nodular is the most common manifestation, but any skin type can present with any variation

Continuous scratching in PN leads to **neuroimmune dysregulation** and **type 2 inflammation**, resulting in increased neuronal signaling, further pruritus, skin fibrosis and nodule formation⁷⁻⁹



Emerging targeted therapies for PN have been shown to alleviate patient burden by reducing itch and nodules, and by improving sleep and quality of life^{10,11}

AD, atopic dermatitis; Ig, immunoglobulin; IL, interleukin; Th, helper T cell.
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4. Geba GP, et al. *J Allergy Clin Immunol.* 2022;S0091-6749(22):01176-9. 5. Irvine AD, et al. *Br J Dermatol.* 2019;181:895-906. 6. Pallier AS, et al. *J Allergy Clin Immunol.* 2019;143(1):46-55.
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ECM, extracellular matrix; IL, interleukin; PN, prurigo nodularis.
1. Huang AH, et al. *J Invest Dermatol.* 2020;140:480-483.e4. 2. Pereira MP, et al. *J Eur Acad Dermatol Venereol.* 2020;34(10):2373-2383. 3. Zeidler C, et al. *Acta Derm Venereol.* 2018;98(2):173-179. 4. Kwon C, et al. *Medicines (Basel).* 2019;6(4):97. 5. Schedel F, et al. *Hautarzt.* 2014;65:684-690. 6. Zeidler C, et al. *Front Med (Lausanne).* 2021;29:8:649332.
7. Garcovich S, et al. *Vaccines (Basel).* 2021;9:30. 8. Nguyen JK, et al. *Arch Dermatologic Res.* 2020;312(2):81-92. 9. Weigelt N, et al. *J Cutan Pathol.* 2010;37:578-586.
10. Kwatra G, et al. Presentation at AAD 2023 New Orleans, LA, USA, 17-21 March 2023. Abstract #45996. 11. Yosipovitch G, et al. *Nat Med.* 2023;29(5):1180-1190.
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