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NOD-2 like receptor expression in psoriatic skin

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Abstract
Psoriasis is a long-lasting, widespread disease characterized by inflammation throughout the body, caused by the immune system, and it affects approximately 2% of the population. The disease known as psoriasis is a complicated condition that is caused by a number of genes and is affected by both genetic and environmental factor factors. Inflammatory responses to bacterial psoriasis are influenced by NOD2, which is a member of the NOD1/APAF1 family of apoptosis genes and plays a role in producing these responses. The activation of the transcription factor NFκB is responsible for the regulation of these responses. The NOD2 receptor is an intracellular receptor that is capable of recognizing microbial components that are derived from peptidoglycan found in bacteria. Furthermore, it has the potential to cause inflammation in the mucosal tissues, in addition to its role in maintaining the equilibrium of the mucosal environment where it is found.

Keywords: NOD-2 like receptor expression, psoriatic skin, psoriasis

Introduction
Psoriasis, also known as PS, is a chronic inflammatory condition that is brought on by the immune system. It is characterized by the excessive growth of skin cells, the infiltration of white blood cells into the skin, and inflammation. This contributes to the formation of distinct red and scaly patches on the skin [1].
Plaque PS is the predominant form of PS, constituting over 80% of all cases. Plaque PS is distinguished by red and scaling spots or plaques on the outer surfaces of the body, On the other hand, it can also have an effect on the folds of the skin, as well as the palms, soles, and nails [2].
NOD2, which stands for nucleotide binding and oligomerization domain-containing protein 2, is a member of the NLRC subfamily of NRLs. It was the 2nd member of the family to be recognized, following NOD1. The protein that makes up the NOD2 receptor is made up of 1040 amino acids and has a molecular weight of 110 kilodaltons. In humans, the NOD2/CARD15 gene, which is located on chromosome 16 q 12, is responsible for its production [3].

Genetic basis of psoriasis
The human leukocyte antigen (HLA) types HLA-Cw6, B13, B17, B27, Bw57, and HLADR4 have been linked to the development of psoriasis. Individuals carrying the HLACw6 gene variant exhibited a risk for developing PS that was ten times higher than that of the general population. Furthermore, there is a distinct correlation between guttate PS in children, erythrodermic PS, and the expression of HLA-B13 and HLA-B17 [4].
One-third of patients reported positive family history of the disease. Twins' studies suggest that the affection of one monozygotic twin with PS increases the chance for the other twin more than 55% to have PS, while it is around 20% for dizygotic twins. It was found that if both parents were affected, the risk for their descendants to develop PS was 41%, while the risk was 14% if one parent is psoriatic [4].
Researchers discovered 24 genetic loci linked to PS, many of which have been named PS susceptibility loci (PSORS) (PSORS-1 through PSORS-9). These are hypothesized to be underlying PS pathogenesis pathways. The chromosomal regions that are associated with skin barrier function, IL-23 signaling, nuclear factor kappa β (NF-kB) and interferon (IFN) signaling, and IL-17 cell responses have been discovered to contain genetic variants.

However, specific genes that are associated with susceptibility have not yet been identified [5].

Genetic loci associated with Psoriasis

PSORS-1, the major genetic determinant of psoriasis:

The PSORS1 gene is a component of the Major Histocompatibility Complex (MHC), which can be found on chromosome 6p21. PS was found to have a significant correlation with three genes that are encoded, specifically HLA-C, CCHCR1, and CDSN. A receptor that is a member of the MHC class I, HLA-C is involved in immunological responses by presenting antigens to CD8+ T lymphocytes. This is how it contributes to the immune system. One of the proteins that can be found in keratinocytes is called CCHCR1, and it plays a role in the process of skin cell shedding. Psoriasis is characterized by a disruption in this process. Psoriasis is characterized by the shedding of skin cells, which is also affected by CDSN, which is another protein that is found in keratinocytes and plays a role in the cell turnover process [6].It is believed that HLA-Cw6 has a strong affinity for various PS autoantigens, possibly even more than one. When HLA-Cw6 interacts with CD8+ T lymphocytes, it is able to display a specific auto-antigen known as ADAMTS-like protein 5, according to Arakawa *et al*. [7]. Moreover, additional investigation has shown that HLA-Cw6 has a high affinity for LL-37 [8].

PSORS-2 locus

The initial identification of the PSORS2 region was accomplished through the use of linkage analysis on a multi-generational pedigree from North America, which indicated that the disorder was inherited in an autosomal dominant manner [4]. Later on, extended Asian kindred were found to have linked to the same region. Thanks to advances in next-generation sequencing, we now know that these two families' disease segregation is due to mutations in the CARD14 gene. It is worth mentioning that pityriasis rubra pilaris and generalized pustular PS, two disorders phenotypically similar to plaque PS, have both been found to encompass CARD14 mutations. CARD14 mediates TRAF2-dependent NF-κB signal transduction and is highly expressed in keratinocytes, where it encodes an adaptor protein. Mutations in CARD14 increase cytokine production, which in turn promotes inflammation [9].

PSORS-4 locus

Based on the fact that two EDC genes (LCE3B and LCE3C) encode two late cornified envelope proteins, the PSORS4 region is located on chromosome 1q21 and spans the Epidermal Differentiation Cluster (EDC). There is evidence to suggest that disruption of skin barrier function plays a pathogenic role in PS [10].

Nucleotide binding and oligomerization domain-containing protein 2 (NOD2)

The second member of the NLRC subfamily of NLRs to be discovered, following NOD1, is NOD2. In humans, the NOD2/CARD15 gene on chromosome 16q12 encodes the NOD2 receptor, a protein containing 1040 amino acids and a molecular weight of 110 kDa [3].

Structure

The C-terminal part of the NOD 2-like receptor molecule includes a leucine-rich repeat domain, which is recognized to be involved in protein-protein interactions. The protein's central region contains a NOD domain, a crucial component for self-oligomerization. Apoptosis and NF-kB activation pathways are known to be impacted by two CARD domains located in the N-terminal region [3].

Expression

Despite the fact that it is present in monocytes, macrophages, dendritic cells, hepatocytes, preadipocytes, oral cavity, lung, and intestinal epithelial cells, it is present in intestinal stem cells and ileal Paneth cells to a greater extent than it is in any other type of cell. As is the case with NOD1, NOD2 is found in the cytoplasm; however, it is able to detect bacterial invasion at the entrance site by being recruited to the plasmatic membrane [11]. Additionally, their expression in psoriatic skin was recently verified [12].

NOD2 signaling

To eliminate infections and protect tissues from injury, the innate immune system must be activated. NOD2, which is an intracellular receptor for microbial components and is made from bacterial peptidoglycan, is responsible for regulating mucosal homeostasis as well asinflammatory responses. The effector domain of the NOD2 protein is made up of two tandem N-terminal CARDs. These CARDs are responsible for facilitating distinct homophilic interactions with molecules that contain CARDs further downstream. NOD2 self-oligomerizes and recruits the downstream adaptor molecule, RIP2, through homophilic CARD-CARD interaction in response to the activation signal that is provided by muramyl dipeptide, which is a component of the cell wall of some Gram-positive bacteria as well as some Gram-negative bacteria [13].

The ubiquitination of the essential modulator NF-kB α occurs when RIP2 is an active protein. The stimulation of the IKK complex takes place, which then leads to the phosphorylation of the inhibitor of transcription factor NF-kB, which is known as IKBα. Following this, NF-kB travels to the nucleus, where it begins the process of transcription of genes that promote inflammation, such as cytokines, growth factors, and various factors that stimulate immune cells. The IKK complex is accountable for the activation of MAP kinases and the transcription factor Activator Protein 1, both of which play a role in the processes of cell proliferation, differentiation, and death. TGF-β activated kinase 1 is the target of RIP2, which in turn activates the factors that are being discussed [13].

Function of NOD2

An amino NH2-terminal CARD domain is possessed by NOD 1 and 2, which are prominent members of the NLRC subgroup. These proteins are involved in the process of identifying bacterial pathogens that are present in the cytoplasm. These receptors, which are found inside of cells, have been shown to be capable of detecting fragments of bacterial peptidoglycan and initiating proinflammatory pathways such as NF-kB, according to research [14, 15].

According to the findings of this discovery, the significance of peptidoglycan specificity in the activation of these receptors to produce an immune response has been revealed. D-glutamyl-meso-diaminopimelic acid (iE-DAP) is a peptidoglycan pattern that is present in both gram-positive and gram-negative bacteria. In response to this pattern, NOD1 causes the activation of NF-kB, which promotes the

growth of the bacterial population. Muramyl dipeptide, on the other hand, is a common peptidoglycan motif that can be found in both gram-positive and gram-negative bacteria. It is responsible for activating NOD2. Both NOD1 and NOD2 are able to provide an additional layer of defense for the host, which allows them to coordinate an immune response by searching the cytosol for microorganisms that have invaded the body [16].

NOD 1 and 2, inflammatory mediators and immune response:

There is a wide variety of genes that are activated as a result of the recognition of ligands by NOD1 and NOD2, and a significant number of these genes are dependent on the activation of NF-kB. In dendritic cells, macrophages, and monocytes, the activation of NOD1 and NOD2 occurs, resulting in the production of pro-inflammatory cytokines such as IL-1β, IL-6, TNFα, CXCL8/IL-8, KC, IL-10, IL-18, IL-12p40, and IL-12p70. Additionally, the manifestation of co-stimulatory molecules and adhesion molecules is observed. One of the outcomes of inducing the NOD pathway in epithelial cell lines is the release of pro-inflammatory substances, such as TNF, IL-6, CXCL8/IL-8, CCL2, and CXCL5, in addition to the release of antimicrobial peptides, specifically β-defensin 2. For the recruitment and stimulation of effector cells, as well as the inflammatory processes that lead to the establishment of an appropriate immune response, each and every one of these components is absolutely necessary [17].

The impact of NOD2 activation on the adaptive immune responses

Research has shown that NLRs play a role in both initiating the innate immune response and promoting adaptive immunity. The capacity of NOD2 to facilitate adjuvant action has been proven, as the simultaneous injection of human serum albumin (HSA) and MDP leads to the production of IgG1 antibodies against this particular T cell-dependent antigen [18].

NOD 2 and autophagy

Innate and adaptive immunity, adaptation to starvation, degradation of misfolded or aggregated proteins or damaged organelles, and removal of intracellular pathogens are all examples of the physiological activities that autophagy engages in. Autophagy is a cellular stress response that enhances cellular survival by engaging in these physiological activities such as these. One can argue either way about the significance of NOD1 and NOD2 in the process of autophagy [19].

Through the actions of NOD1 and NOD2, the autophagy protein ATG16L1, which is an essential component of the autophagic machinery, was brought to the plasma membrane at the point where bacteria entered the cell. A mechanism that did not involve the adaptor RIP2 or the transcription factor NF-kB was responsible for this recruitment, which took place [18].

Additional research has uncovered evidence of a mechanism that activates autophagy through NOD2, without the need for RIP2. The activation of NOD2 by MDP induces autophagy in dendritic cells, and this process requires the presence of RIP2. Researchers have found that the activity of RIP2 tyrosine kinase has two effects on NOD2-dependent autophagy. Firstly, it activates p38 MAPK, which promotes autophagy. Secondly, it counteracts the suppression of autophagy caused by the phosphatase PP2A [20].

Role of NOD2 in PS:

Tervaniemi *et al.* [21] conducted a study in which the transcriptomes of split-thickness skin grafts were analyzed in order to investigate the characteristics of psoriatic epidermis, with a specific emphasis on epidermal thickening, which is a prominent characteristic of psoriatic therapy (PS). For this particular purpose, they decided to use RNA 5'-end sequencing. The significance of NLR signaling pathways was brought to light by the fact that the psoriatic epidermis exhibited elevated levels of expression of the NLR signaling genes NOD2, PYCARD, CARD6, and IFI16 [22]. Integrative analysis of the methylome and transcriptome in the Chinese Han population demonstrated that NLR signaling is a prominent molecular mechanism involved in PS [23].

PS vulgaris and pustular PS, two types of psoriasis, have been associated with mutations in the CARD14 gene, a member of the CARD family. Upon comparing psoriasis patients to healthy controls, researchers identified an additional 15 rare missense variations in the CARD14 gene [24]. CARD14 gain-of-function mutations were discovered to induce spontaneous skin inflammation resembling psoriasis by enhancing keratinocyte reactions to IL-17A. Hyperactivation of CARD14 in living organisms was sufficient to induce psoriasis mediated by IL-23/IL-17 [25]. Moreover, there is a broad consensus that inflammasomes play a significant role in the pathogenesis of psoriasis, as well as in the activation of CARD 15 in the epidermis affected by psoriasis [12].

Role of NOD2 in psoriasis treatment

NOD-like receptors, including NOD2, play a crucial role in the innate immune response by identifying bacterial peptidoglycan. This recognition triggers the activation of NF-κB and the synthesis of pro-inflammatory cytokines like IL-1β, IL-6, and TNF-α. These cytokines play a role in creating the inflammatory conditions in psoriasis by encouraging excessive growth of keratinocytes and maintaining long-term inflammation [26].

The activation of NOD2 can worsen the inflammatory response by interacting with other pattern recognition receptors (PRRs) and increasing the production of cytokines. The heightened inflammatory state plays a crucial role in the development of psoriasis, which is characterized by an increase in the growth and a decrease in the proper maturation of keratinocytes [27].

Genetic variations in the NOD2 gene have been associated with a higher vulnerability to psoriasis, suggesting a genetic predisposition related to this receptor. These genetic variations can result in abnormal functioning of the NOD2 protein, which in turn contributes to the inflammatory processes seen in psoriatic skin lesions [28].

Therapeutic Implications

Targeting the NOD2 pathway represents a promising therapeutic strategy for psoriasis. Inhibitors that specifically target NOD2 or its downstream signaling pathways could potentially attenuate the inflammatory response. Blocking NOD2 activation or inhibiting key molecules in it signaling cascade, such as NF-κB, may reduce cytokine production and alleviate psoriasis symptoms [29].

Current biologic therapies targeting TNF-α, IL-17, and IL-23 may also influence pathways involving NOD2. Understanding the interactions between these pathways can enhance the efficacy of existing treatments or lead to new combination therapies that more effectively address the underlying inflammation in psoriasis [30].

Modulating the skin microbiome presents another potential therapeutic approach, as NOD2 recognizes bacterial components. Adjusting the microbiome to reduce pro-inflammatory microbial patterns through probiotics or antibiotics could decrease NOD2-mediated inflammation and improve psoriasis outcomes [31].

Gene therapy offers a novel intervention for patients with specific NOD2 mutations associated with severe psoriasis. Techniques such as CRISPR/Cas9 could be used to correct these mutations, potentially reducing disease severity and improving patient quality of life [32].

Research into small molecule inhibitors of NOD2 activation is ongoing. These inhibitors could provide a less invasive option compared to biologics, offering an alternative treatment for patients with moderate to severe psoriasis. Such small molecules could directly inhibit NOD2 or its downstream signaling pathways, providing targeted therapeutic effects [33].

Combining NOD2 inhibitors with other anti-inflammatory agents or immune modulators might provide a synergistic effect. This approach could enhance overall treatment efficacy, reduce disease severity, and offer new therapeutic options for patients with challenging cases of psoriasis [34].

Conclusions

NOD2-like receptors play a significant role in the inflammatory pathways involved in psoriasis. Targeting these receptors and their downstream effects offers a promising avenue for developing new treatments. By reducing NOD2 activity, it may be possible to alleviate the chronic inflammation and keratinocyte hyperproliferation that characterize psoriasis. Continued research into the specific mechanisms and interactions of NOD2 will be vital for translating these insights into clinical practice.

Conflict of Interest

Not available

Financial Support

Not available

References

1. Rendon A, Schäkel K. Psoriasis pathogenesis and treatment. International Journal of Molecular Sciences. 2019;20. doi:10.3390/ijms20010014
2. Armstrong AW, Read C. Pathophysiology, clinical presentation, and treatment of *psoriasis*: A review. Journal of the American Medical Association. 2020;323:1945-60. doi:10.1001/jama.2020.4006
3. Domínguez-Martínez DA, Núñez-Avellaneda D, Castañón-Sánchez CA, Salazar MI. NOD2: Activation during bacterial and viral infections, polymorphisms and potential as therapeutic target. Revista de Investigación Clínica. 2018;70:18-28. doi:10.24875/RIC.18002477
4. Capon F. The genetic basis of *psoriasis*. International Journal of Molecular Sciences. 2017;18(12). doi:10.3390/ijms18122684
5. Wang Z, Zheng H, Zhou H, Huang N, Wei X, Liu X, *et al*. Systematic screening and identification of novel psoriasis-specific genes from the transcriptome of psoriasis-like keratinocytes. Molecular Medicine Reports. 2019;19:1529-1542. doi:10.3892/mmr.2019.9835
6. Uppala R, Tsoi LC, Harms PW, Wang B, Billi AC, Maverakis E, *et al*. "Autoinflammatory psoriasis"-genetics and biology of pustular psoriasis. Cellular & Molecular Immunology. 2021;18:307-317. doi:10.1038/s41423-020-00499-5
7. Arakawa A, Siewert K, Stöhr J, Besgen P, Kim SM, Rühl G, *et al*. Melanocyte antigen triggers autoimmunity in human psoriasis. Journal of Experimental Medicine. 2015;212:2203-2212. doi:10.1084/jem.20151093
8. Mabuchi T, Hirayama N. Binding affinity and interaction of LL-37 with HLA-C*06:02 in *psoriasis*. Journal of Investigative Dermatology. 2016;136:1901-1903. doi:10.1016/j.jid.2016.05.097
9. Berki DM, Liu L, Choon SE, David Burden A, Griffiths CEM, Navarini AA, *et al*. Activating CARD14 mutations are associated with generalized pustular psoriasis but rarely account for familial recurrence in psoriasis vulgaris. Journal of Investigative Dermatology. 2015;135:2964-2970. doi:10.1038/jid.2015.251
10. Dand N, Mahil SK, Capon F, Smith CH, Simpson MA, Barker JN. Psoriasis and genetics. Acta Dermato-Venereologica. 2020;100. doi:10.2340/00015555-3431
11. Sidiq T, Yoshihama S, Downs I, Kobayashi KS. Nod2: A critical regulator of ileal microbiota and Crohn's disease. Frontiers in Immunology. 2016;7:367. doi:10.3389/fimmu.2016.00367
12. Shehata WA, Shoeib M, Shoeib MM, Shokhba H, Shams A. Nucleotide binding and oligomerization domain 2 in psoriasis: A clinical and immunohistochemical study. Journal of Immunoassay and Immunochemistry. 2022;43:43-53. doi:10.1080/15321819.2021.1978666
13. Trindade BC, Chen GY. NOD1 and NOD2 in inflammatory and infectious diseases. Immunological Reviews. 2020;297:139-61. doi:10.1111/imr.12953
14. Al Nabhani Z, Dietrich G, Hugot JP, Barreau F. Nod2: The intestinal gate keeper. PLoS Pathogens. 2017;13. doi:10.1371/journal.ppat.1006177
15. Liu P, Lu Z, Liu L, Li R, Liang Z, Shen M, *et al*. NOD-like receptor signaling in inflammation-associated cancers: From functions to targeted therapies. Phytomedicine. 2019;64:152925. doi:10.1016/j.phymed.2019.152925
16. Keestra-Gounder AM, Tsolis RM. NOD1 and NOD2: Beyond peptidoglycan sensing. Trends in Immunology. 2017;38:758-67. doi:10.1016/j.it.2017.07.004
17. Barnabei L, Laplantine E, Mbongo W, Rieux-Laucat F, Weil R. NF-κB: At the borders of autoimmunity and inflammation. Frontiers in Immunology. 2021;12:716469. doi:10.3389/fimmu.2021.716469
18. Negroni A, Pierdomenico M, Cucchiara S, Stronati L. NOD2 and inflammation: Current insights. Journal of Inflammation Research. 2018;11:49-60. doi:10.2147/JIR.S135155
19. Cui B, Lin H, Yu J, Yu J, Hu Z. Autophagy and the immune response. In: Qin Z-H, editor. Autophagy:

Biology and diseases: Basic science. Singapore: Springer Singapore; c2019. p. 595-634. doi:10.1007/978-981-13-2895-8_23
20. Lassen KG, Xavier RJ. Mechanisms and function of autophagy in intestinal disease. Autophagy. 2018;14:216-20. doi:10.1080/15548627.2017.1382625
21. Tervaniemi MH, Katayama S, Skoog T, Siitonen HA, Vuola J, Nuutila K, *et al*. NOD-like receptor signaling and inflammasome-related pathways are highlighted in *psoriatic* epidermis. Scientific Reports. 2016;6:22745. doi:10.1038/srep22745
22. Ferrari D, Casciano F, Secchiero P, Reali E. Purinergic signaling and inflammasome activation in psoriasis pathogenesis. International Journal of Molecular Sciences. 2021;22. doi:10.3390/ijms22147718
23. Tang L, Cheng Y, Zhu C, Yang C, Liu L, Zhang Y, *et al*. Integrative methylome and transcriptome analysis to dissect key biological pathways for psoriasis in Chinese Han population. Journal of Dermatological Science. 2018;91:285-91. doi:10.1016/j.jdermsci.2018.04.004
24. Chen L, Cao SQ, Lin ZM, He SJ, Zuo JP. NOD-like receptors in autoimmune diseases. Acta Pharmacologica Sinica. 2021;42:1742-1756. doi:10.1038/s41401-020-0468-y
25. Wang M, Zhang S, Zheng G, Huang J, Songyang Z, Zhao X, *et al*. Gain-of-function mutation of CARD14 leads to spontaneous *psoriasis*-like skin inflammation through enhanced keratinocyte response to IL-17A. Immunity. 2018;49:66-79.e5. doi:10.1016/j.immuni.2018.06.006
26. Strober W, Murray PJ, Kitani A, Watanabe T. Signalling pathways and molecular interactions of NOD1 and NOD2. Nature Reviews Immunology. 2006;6:9-20. doi:10.1038/nri1747
27. Kaser A, Zeissig S, Blumberg RS. Inflammatory bowel disease. Annual Review of Immunology. 2010;28:573-621. doi:10.1146/annurev-immunol-030409-101225
28. Lesage S, Zouali H, Cézard JP, Colombel JF, Belaiche J, Almer S, *et al*. CARD15/NOD2 mutational analysis and genotype-phenotype correlation in 612 patients with inflammatory bowel disease. American Journal of Human Genetics. 2002;70:845-57. doi:10.1086/339432
29. Girardin SE, Travassos LH, Hervé M, Blanot D, Boneca IG, Philpott DJ, *et al*. Peptidoglycan molecular requirements allowing detection by Nod1 and Nod2. Journal of Biological Chemistry. 2003;278:41702-8. doi:10.1074/jbc.M307198200
30. Blauvelt A, Lebwohl MG, Bissonnette R. IL-23/IL-17A dysfunction phenotypes inform possible clinical effects from anti-IL-17A therapies. Journal of Investigative Dermatology. 2015;135:1946-1953. doi:10.1038/jid.2015.126
31. Scher JU, Ubeda C, Artacho A, Attur M, Isaac S, Reddy SM, *et al*. Decreased bacterial diversity characterizes the altered gut microbiota in patients with psoriatic arthritis, resembling dysbiosis in inflammatory bowel disease. Arthritis & Rheumatology. 2015;67:128-139. doi:10.1002/art.38975
32. Hsu PD, Lander ES, Zhang F. Development and applications of CRISPR-Cas9 for genome engineering. Cell. 2014;157:1262-78. doi:10.1016/j.cell.2014.05.010
33. Becker C, Dornhoff H, Neufert C, Fantini MC, Wirtz S, Huebner S, *et al*. Cutting edge: IL-23 cross-regulates IL-12 production in T cell-dependent experimental colitis. Journal of Immunology. 2006;177:2760-2764. doi:10.4049/jimmunol.177.5.2760
34. Gisondi P, Girolomoni G. Psoriasis and atherothrombotic diseases: disease-specific and non-disease-specific risk factors. Seminars in Thrombosis and Hemostasis. 2009;35:313-324. doi:10.1055/s-0029-1225761.

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