**Beat: Health**

# The Psoriasis Theory

### A Hidden Virus Inside Immune Cells

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**USPA NEWS -** Why did researchers fail to analyze the immune cells themselves?
A critical gap in psoriasis research and the case for a viral pathogenesis paradigm
I. The theoretical bias: a paradigm that precluded the right questions
Over time, we've noticed a significant gap and confusion in defining psoriasis. It has become necessary to search for the true cause behind the disease's outbreak and to consider another scientific approach that puts viruses under scrutiny.

Theoretical bias: Because psoriasis is a complex disease and because scientific evidence only implicates immune cells, scientists have adopted the same strategies, considering psoriasis to be primarily an autoimmune disease caused by a genetic defect or mutation that distorts genetic information, thus creating an immune environment hostile to the body itself.
This scientific bias among researchers obscured the true cause and led to the dismissal of the hypothesis of an external pathogen, such as a virus embedded within the immune cell.
Consequently, researchers neglected to ask crucial questions that would have taken a significant step toward the truth: since the immune system dysfunction is not widespread and does not destroy all the body's immune cells, what caused the malfunction in some immune cells, triggering their reprogramming to become confused and attack the body itself?

Since researchers have already assumed that the cause of psoriasis is purely internal and rooted in a genetic defect, it is obvious that financial resources will not be allocated to study how to uncover an external cause such as the integrated virus theory. The flaw in the research path is logical but biased and systematic, since the research system is what determines the hypotheses that can be funded, the scientific experiments that can be applied, and even the questions that are considered scientifically legitimate.

II. Methodological inadequacy: the wrong tools for the right question
At the technical level, the methodologies employed were fundamentally inadequate for detecting viral DNA embedded within immune cells. Polymerase chain reaction (PCR) homogenizes the nucleic acid content of thousands of cells simultaneously, rendering the viral signal practically undetectable when the percentage of virus-carrying cells does not exceed 5–10%, as the embedded sequences dilute below the detection threshold. Immunohistochemistry (IHC) is a specialized immunoassay technique that detects specific antigens (proteins) in cells within a tissue section by utilizing the binding affinity of antibodies.This technique completely fails to detect latent, transcriptionally inactive embedded viral genomes, since the virus is hidden within the host cells. Flow cytometry measures immune cell surface markers and clusters without detecting intracellular embedded sequences or aberrant genetic modifications.

At the same time, more appropriate techniques were conspicuously absent from psoriasis research. Whole-genome sequencing (WGS), the most comprehensive tool for detecting viral sequences embedded within host cell genomes, including silent and latent integrations, was not used on psoriasis-infected immune cells isolated for this purpose. Other techniques have also been overlooked, such as single-cell genome sequencing, which can identify immune cells carrying the embedded viral sequences; laser capture microdissection( LCM )is used to isolate specific cells like immune cells from infected tissues;it is very rapid , non-destructive , and provide high –purity samples for RNA and DNA ,or protein extraction.Also, single-cell RNA sequencing (scRNA-seq), which can reveal aberrant gene expression patterns in individually reprogrammed cells. These tools exist and are available, but they have never been directed toward the right question due to the theoretical bias underlying psoriasis, which always points the finger at the immune system.

The complementary roles of these techniques in uncovering the whole truth about this virus are crucial: whole-genome sequencing reveals the full range of viral integration events, including latent sequences that cannot be detected using transcription analysis methods, while single-cell RNA sequencing reveals the subsequent functional consequences of this integration in actively reprogrammed cells.Comprehensive research requires the use of both.

Importantly, the most recent applications of single-cell RNA sequencing (scRNA-seq) in psoriasis research—including a study by

Nielsen et al. (Frontiers in Immunology, 2025), which analyzed peripheral lymphocytes from 70 patients to compare psoriatic arthritis with cutaneous psoriasis, and another study published in Frontiers in Genetics in 2025 that combined scRNA-seq with Mendelian randomization across 174 skin samples—have used this technique exclusively to characterize subsequent inflammatory cell populations and identify therapeutic biomarkers. None of these studies explored immune cells for embedded viral sequences, applied whole genomic sequencing (WGS) technology to look for viral genomic fusions, or questioned what originally reprogrammed these cells.

III. The streetlight effect: measuring what is visible, not what is causal
Research has focused exclusively on what is easily measurable and visible: elevated levels of cytokines such as TNF-?, IL-17, and IL-23; infiltration of immune cells into the dermis; and gene expression disturbances identified by comprehensive RNA-seq sequencing—without investigating the root cause of these phenomena, even though researchers know that the dysfunction doesn't affect the entire immune system but rather a localized part of it, primarily located in the periphery of the skin. This reflects what philosophers of science call the "streetlight effect": the methodological tendency to search where the tools are available rather than to search for the truth.

Researchers described the smoke—the cytokine storm, the T-cell infiltration, and the excessive proliferation of keratinocytes—without investigating the underlying cause. Wolk et al. (Science Translational Medicine, 2013) demonstrated that psoriatic skin exhibits a robust antiviral state, characterized by significantly elevated antiviral proteins including MX1, BST2, ISG15, and OAS2, mediated by IL-29 produced by Th17 cells. Rather than interpreting this persistent antiviral activation as evidence of ongoing viral pressure within the tissue, researchers framed it solely as a defense mechanism — failing to ask what the immune system was defending against.

Similarly, studies by Molès et al. (2005) and Lättekivi et al. (2018) detected elevated expression of Human Endogenous Retroviruses (HERVs) in psoriatic skin, while a UCSF study (Journal of Translational Medicine, 2014) examined serological responses against HERV-K proteins in psoriatic patients and found a significant decrease in IgM antibodies compared to healthy controls. Rather than interpreting this as evidence that the virus is invisible to the humoral immune system — precisely because it is not circulating freely in the bloodstream but hiding within the cell nucleus — researchers filed it within the existing autoimmune framework and moved on. This finding, however, is entirely consistent with a virus that has already integrated into host immune cell DNA: a hidden pathogen produces no circulating antigens, triggers no CRP elevation, evades the primary immune response, and leaves no trace in the blood — which is exactly why it has never been found there. Thus, while Molès et al.

(2005) detected HERV expression in psoriatic skin, UCSF (2014) found decreased IgM antibodies in the blood — confirming that the virus is present in the tissue while remaining invisible to the humoral immune system. The data pointed toward a new direction; the paradigm prevented the field from following it.

IV. The documented research gap
The lack of research into the underlying immune dysfunction in psoriasis is not merely a conclusion; it is explicitly acknowledged in the scientific literature itself.A 2024 review published in the Journal of Allergy and Clinical Immunology_ ScienceDirect explicitly acknowledged that there are no curative treatments for psoriasis, and that patients continue immunomodulatory therapies indefinitely despite achieving clinical remission — reinforcing the existence of an "inflammatory memory" retained in tissue even after symptoms resolve. The review focused on tissue-resident memory (TRM) cells, Langerhans cells, and dermal dendritic cells as potential drivers of recurrence, yet failed to ask the most fundamental question: what originally programmed this inflammatory memory, and why does it persist even when the visible disease disappears?

A 2024 study published in Frontiers in Genetics also acknowledged that "the etiology of psoriasis is not fully understood," and that gaps in understanding the disease mechanism necessitate a deeper understanding of complex molecular mechanisms. A 2025 review published in PubMed confirmed that while single-cell techniques have revealed cellular diversity in psoriasis, the focus has remained on keratinocytes, fibroblasts, and T cells as therapeutic targets, without any investigation into the original trigger for this diversity.
...To date, no published study has applied whole-genome or single-cell genome sequencing to the immune cells responsible for psoriasis with the explicit aim of detecting integrated viral sequences, identifying virally reprogrammed immune populations, or testing the hypothesis that the virus integrated with the host stimulates the release of aberrant cytokines that target neighboring immune cells and ultimately healthy keratinocytes.

V. The absence of comprehensive thinking that encompasses several disciplines.

These structural obstacles to discovering the truth are exacerbated by the systematic absence of holistic thinking that encompasses

all scientific disciplines. Immunologists, virologists, dermatologists, and gastroenterologists operate in isolated disciplines, each producing insights that remain separate from one another. Currently, there is no integrated framework linking virology to immune cell reprogramming, immune disorders to the gut-skin axis, or microbiome disruption to systemic autoimmunity activation. What is needed is a multidisciplinary consortium working on the same patient samples within a shared research framework—one that does not presuppose answers before the experiment is designed and the results are observed.

VI. Institutional conflicts of interest
At the institutional level, most psoriasis research is funded by pharmaceutical companies with well-established financial interests in biological therapies, including anti-TNF and anti-IL-17 drugs. These companies provide drugs available to every patient annually without achieving a definitive cure. Identifying an integrated virus as the pathogen could open the door to a therapeutic approach, fundamentally altering this economic model. Consequently, research funding continues to be directed toward improving existing treatments rather than uncovering the root cause of the disease—a structural barrier that systematically downplays hypotheses that challenge prevailing paradigms.

VII. The logic of scientific revolutions
As Thomas Kuhn explained in his book *The Structure of Scientific Revolutions* (1962), scientists work within a dominant paradigm and an established scientific path, rarely challenging its fundamental assumptions or updating the established methodology until anomalies accumulate to the point where continuing along the same lines becomes difficult. Within the dominant paradigm—psoriasis as a genetic autoimmune disease—researchers ask only the questions the paradigm allows, use only the tools it supports, and interpret only the results it can accommodate.
The hypothesis presented here represents such a paradigm shift: from “autoimmunity + genetics” to “integrated virus + dysfunctional host environment”—a paradigm in which a viral pathogen integrates into host immune cells, reprograms them, and directs their inflammatory output against neighboring immune cells and, ultimately, against healthy keratinocytes. This hypothesis has never been tested. The tools for testing it are now available. What was lacking was the intellectual readiness, based on an updated understanding, to ask the question.

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