



# **Transcript Details**

This is a transcript of an educational program. Details about the program and additional media formats for the program are accessible by visiting: https://reachmd.com/programs/dermconsult/breaking-down-vitiligo-pathophysiology/13975/

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Breaking Down Vitiligo Pathophysiology

## Announcer:

Welcome to *DermConsult* on ReachMD. On this episode, sponsored by Incyte, we'll examine the pathophysiology of vitiligo with Dr. David Rosmarin, who's a clinical investigator and Vice-Chair for Research and Education in the Department of Dermatology at Tufts Medical Center in Boston. Here's Dr. Rosmarin now.

## Dr. Rosmarin:

Vitiligo is a chronic autoimmune disease that destroys the pigment cell or melanocyte in patients leading to white spots often around the orifices in the body, such as around the eyes, mouth, nose, hands are commonly affected, and really anywhere on the body.

There are two main types: Nonsegmental vitiligo, which we usually just refer to as vitiligo, and also segmental type, which is a lot less common, but more frequent in kids. And that usually rapidly progresses and then stops in a localized area.

While it only takes an overactive immune system to cause vitiligo and white spots, to repigment patients, it's really a two-step process. One is we have to tell the immune system to calm down. And two, we have to try to stimulate those pigment cells to come back. Now there are two main places where the pigment cells can migrate from to go back to those white patches. One is the sides of the vitiligo patches, and second more commonly from the hair follicle. And there are certain pre-melanocytes that can come from the follicle and help repopulate those white patches.

In vitiligo, we find that it is the TH1 arm of the immune system that is too active: High levels of interferon gamma, CXCL9, and 10. Whereas in other diseases like psoriasis, it's the TH17 arm that's too active. And in atopic dermatitis or eczema, it's the TH2 pathway that's too active. Many of our medications that tell the immune system to calm down work quite well at the TH2 and TH17 arm, but we have more limited options for the TH1 arm of the immune system.

Hence, JAK inhibitors have really good activity against interferon gamma in that TH1 arm. Interferon gamma signals through the JAK1, JAK2 pathway. Hence, JAK inhibitors have been shown to be very helpful at improving patients who have had vitiligo.

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