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Role of Interleukin-15 in Vitiligo

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Abstract
Vitiligo relapses remain a significant clinical challenge, with nearly 40% of patients experiencing recurrence due to autoimmune memory perseverance within the skin. A growing body of evidence identifies $CD8^+$ tissue-resident memory T cells (TRM) as central drivers of this relapse. Overexpression of IL-15 in vitiligo skin is likely driven by oxidative stress and chronic inflammation enhances IFN-γ production, promotes CXCR3-mediated T-cell trafficking, and strengthens memory CTL responses, all of which contribute to disease persistence and relapse. Clinical correlations, such as the positive association between IL-15 levels and VASI scores, further reinforce its role in disease severity. IL-15 signaling represents a promising therapeutic target; blocking this pathway such as through anti-CD122 antibodies may offer durable disease control by disrupting the survival of autoreactive TRM cells. Targeting IL-15 could therefore provide a critical strategy for preventing relapses and achieving long-term stability in vitiligo patients.

Keywords: Interleukin-15, Vitiligo

Introduction
Vitiligo is a condition someone develops over time, and it shows up as white, smooth areas on skin. he precise etiology remains unclear, but it's thought to happen because of several different factors that eventually damage the melanocytes [1].
The most well-known idea is that the immunological system outbreaks melanocytes, the cells that make skin color. This happens when the immune system's cells, like T cells as well as B cells, along with certain proteins called cytokines, work together. Skin samples taken from areas near vitiligo patches showed a lot of CD8+ T cells, being a type of immune cell that can damage melanocytes. Also, there was more of two important proteins, TNF-alpha and IFN-gamma, which are linked to a type of immune response called Th1, where the body fights infections by attacking cells directly [2].
In the past few years, studies has centered on the tissue resident memory T cells (TRM) as well as their function in vitiligo reactivation following cessation of treatment. The stability of vitiligo denotes times of no activity of the illness, and it is significant in deciding the path of treatment of the patient; instability is considered as a contraindication for surgical treatment of vitiligo. Unfortunately, the definition of stability is dependent on the patient reporting of no new lesions appearance or preexisting lesions enlargement for 1 year, which is not reliable, as it depends on the memory of the patient. Vitiligo disease activity (VIDA) score (which is utilized to score the activity) depends mainly on the patients' words, so the need for reliable serum markers to detect the activity in vitiligo is mandatory [3].
The cytokine interleukin-15 produced by many different types of cells. This cytokine bridges the gap among the innate as well as adaptive immune systems and has several targets and functions. Numerous cell types as well as tissues, including monocytes, macrophages, keratinocytes, fibroblasts, myocytes, dendritic cells (DCs), and nerve cells, express it constantly [4]

Vitiligo
It considers a prevalent acquired autoimmune skin disease characterized by melanocytes progressive loss, resulting in disfiguring patches or macules of white depigmented skin .This disfiguring disorder usually affects the face and other visible regions of the body that cause severe psychological suffering among sufferers [5].

Prevalence of vitiligo

Vitiligo is the most prevalent depigmentation reason .Its occurrence worldwide has a prevalence of roughly 0.5% to 2% in the general population, adults included as well as children worldwide and it affects all races equally [7]. vitiligo prevalence in egypt ranged about 0.18% [8].

This condition can happen to all ages, but it is most common in the second and third decades of life. Both males and females are equally affected, but women and girls are more likely to seek medical help. The age when symptoms first appear can be different for boys and girls [7].

Clinical picture

Bright white or milky white patches or macules are characteristic for the disease. These lesions lack clinical signs of inflammation, are usually well demarcated and variable in size, often affect the hands , It commonly involves the perioral skin, feet, and scalp, as well as areas that are more susceptible to friction and trauma—such as the elbows and knees—and may occur at any body site, including mucosal surfaces [9].

Clinical symptoms help identify disease activity, as confetti-like depigmentation, trichrome lesions, as well as the Koebner phenomenon. Confetti-like depigmentation looks like small, light-colored spots that are 1 millimeter to 5 millimeters in size, and they appear in groups. These spots usually form around the edges of existing skin lesions [10].

People who have these symptoms often end up losing all color in the affected areas. Trichrome lesions show a patch of lighter skin, sometimes of different widths, between normal skin and completely colorless skin, which creates three different color areas on the skin [10].

The Koebner phenomenon emerges as depigmentation at locations of trauma, generally in a linear configuration. These indicators may be present in people with fresh onset vitiligo as well as those with persistent illness. They suggest continuous CD8+ T-cell-mediated melanocyte death beneath the surface and fast progressing depigmentation [11].

Role of Resident Memory T Cells and IL-15 in Vitiligo relapse

The vitiligo estimated risk relapse after successful repigmentation considers about 40% within the first year [12]. This relapse frequently happens in precisely the same places, which was previously afflicted, indicating that autoimmune memory developed at these sites [13, 14].

The functional CD8 tissue-resident memory T cells (TRM) consider to be responsible for vitiligo reactivation. They consider a T cells long-lasting population that stay inside most nonlymphoid tissues after T cell-driven inflammation and are identified by the expression of surface markers CD69, CD103 and CD49a. After activation, they also express CXCR3, IFN-γ and TNF-α, that attract effector T cells from the circulation [15].

In people with vitiligo, TRM cells in the blood and in the affected skin show a lot of the CD122 part of the IL-15 receptor [16]. Therefore, Using an antibody that targets CD122 suggested as a possible highly effective as well as long-lasting treatment for vitiligo [17].

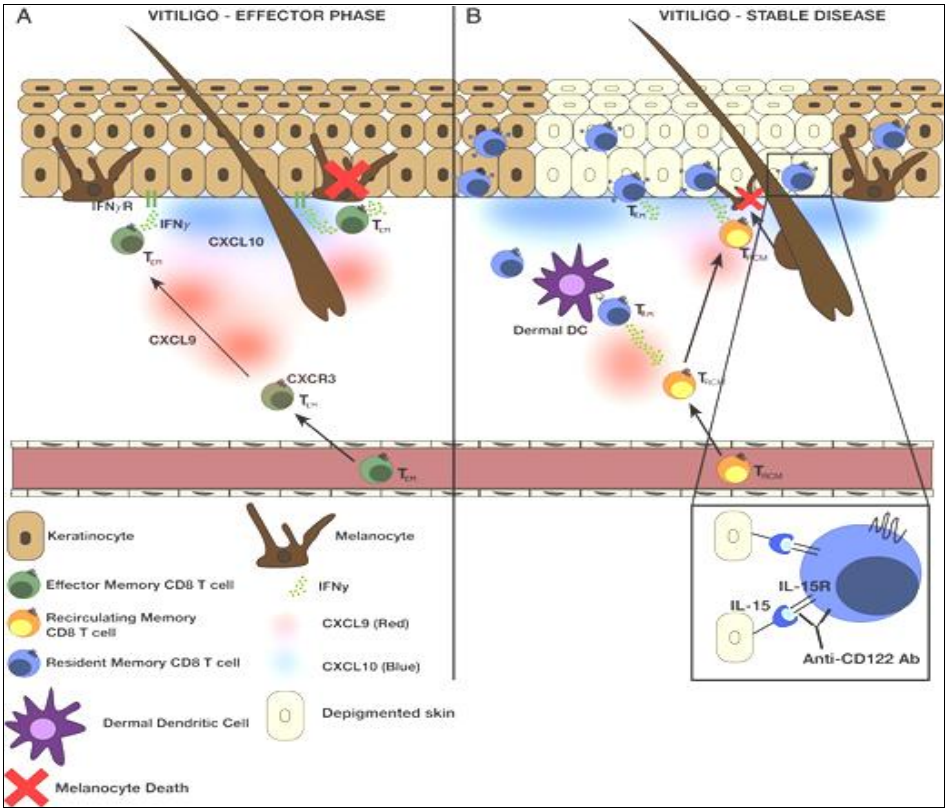

Fig 1: Overview of vitiligo development during both the effector phase as well as persistent vitiligo. (A) In active vitiligo or early lesion development, IFN-γ signaling on keratinocytes stimulates the IFN-γ-dependent chemokines creation, CXCL9 as well as CXCL10, which supports melanocyte-specific CD8+ TEM continued immigration through the dermis to the dermal-epidermal junction (DEJ) where melanocytes reside leading to melanocyte death. Activated CD8+ TEM express IFN-γ, which stimulates recruiting of additional CD8+ TEM to the skin as well as further melanocyte death. (B) Over the disease duration, a subset of CD8+ TEM differentiates into TRM. TRM cells seed the skin tissue, and kept in the tissue due of trans-presentation of IL-15 by keratinocytes. The TRM stimulation induces the inflammatory cytokines IFN-γ as well as CXCL9 production that serve to transport recirculating melanocyte-specific memory T cells (TRCM) to the skin, where they can destroy the repopulating melanocytes. This feed-forward loop and cooperation between TRM and TRCM leads to illness maintenance. In vitiligo, IL-15R activation is enriched on melanocyte-specific CD8+ TRM [18]

Interleukin-15

Interleukin-15 is cytokine, a type of signaling molecule that serves a crucial function in the body's defenses. It enhances growth, staying alive, and working of different immune cells, in addition to NK cells, memory phenotype CD8+ T cells, as well as intraepithelial lymphocytes [19].

Structure

IL-15 considers a cytokine sort that has a structure similar to other cytokines in the four-alpha-helix bundle family. This structure is made up of four long alpha-helices that are arranged in a specific pattern called up-up-down-down. These helices are connected by loops that can be of different lengths and shapes. The loops help determine how well the cytokine binds to its receptor and how strong its biological effects are. IL-15 is first made as a larger protein that includes a signal peptide. This signal peptide is later cut off, leaving behind the mature, active form of the cytokine. [20, 21].

There are two IL-15 mRNA insomnia, one with a shorter signal peptide (SSP) of 21 amino acids (AA) as well as the other with a larger of 48 amino acids (AA) long signal peptide (LSP). Both IL-15 isoforms encode an identical mature IL-15 protein consisting of 114 amino acids, differing only in their signal peptide sequences (279,280).

IL-15 shares the β chain with IL-2 and the γ chain with cytokines IL-2, IL-4, IL-7, IL-9, and IL-21 that together with IL-15 form the common γ chain family of cytokines[22, 23], While IL-15 specificity is provided by high affinity binding to the unique IL-15Rα protein [24].

IL-15 and IL-15Rα can exist bound together on the cell surface or be secreted as a complex, known as trans-presentation, which is fundamental for IL-15's *in vivo* action, particularly in stimulating immune cells like NK cells as well as T cells [25].

This complex arrangement enhances the stability of IL-15 and is crucial for its biological activity, directing its interaction with the receptor complex beta and gamma chains on target cells, facilitating signal transduction and functional responses in immune regulation[26, 27].

IL-15 Physiologic expression:

IL-15 considers a crucial cytokine for immune system function, notably influencing innate and adaptive immunity. It is widely expressed in immune cells like monocytes, macrophages, and dendritic cells, as well as in various non-immune cells under inflammatory conditions [28].

IL-15 expression considers intricately regulated through transcription, translation, as well as its trans-presentation unique mechanism, involving its presentation complexed with IL-15 receptor alpha. This precise regulation helps localize its action, crucial for maintaining immune homeostasis and preventing excessive inflammation or autoimmunity [28].

IL-15 signaling

IL-15 signaling involves a unique trans-presentation mechanism where IL-15 is surface-presented by antigen-presenting cells via binding to IL-15Rα. This complex interacts with IL-2/IL-15R beta (CD122) in addition to the common gamma chain (γc, CD132) on neighboring cells, primarily NK and T cells. This engagement triggers the JAK/STAT signaling pathway, prominently activating JAK1 and JAK3, which then phosphorylate STAT5 [29].

The phosphorylated STAT5 translocates to the nucleus to modulate the genes expression involved in cell survival, proliferation, and differentiation. This signaling cascade is crucial for the development, function, and NK and memory CD8+ T cells survival, playing a significant role in the immune system's response to infections and tumors. Additionally, IL-15 can activate other pathways such as the PI3K/Akt in addition to the MAPK pathway, enhancing cellular responses and functionality further [30].

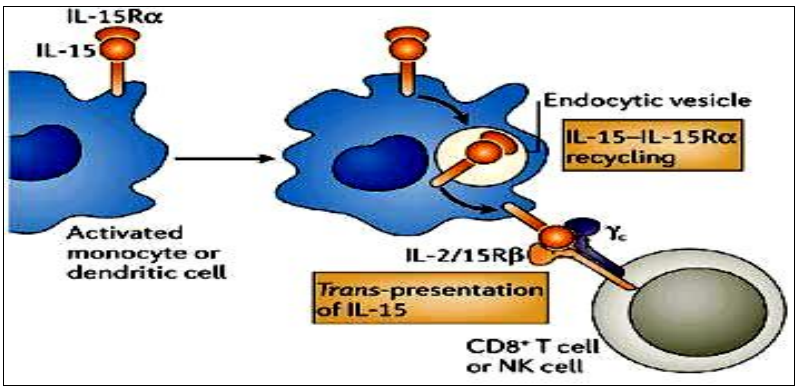

Fig 2: Transpresenting mechanism [29]

Medical implications of IL-15

Autoimmune Diseases: IL-15 helps activate T cells as well as NK cells, which can make autoimmune conditions worse. IL-15 is constantly increased in many different autoimmune diseases, where these immune cells end up attacking the body's own tissues [31].

IL-15 plays a great function in the advancement initiation, as well as natural killer (NK survival) cells as well as T cells, especially enhancing the cytotoxic activities as well as NK and CD8+ T cells which are vital for antiviral and antitumor defenses. Additionally, it supports the maintenance and turnover of memory T cells, essential for long-lasting immune memory [32, 33].

IL-15 overexpression has also been observed in several inflammatory and immune-mediated skin disorders, including alopecia areata, psoriasis, Stevens-Johnson syndrome, toxic epidermal necrolysis, and atopic dermatitis [34-37].

Using monoclonal antibodies to block IL-15 in a human dermatitis model reduced skin cell overgrowth, thickening of the outer layer, and the buildup of immune cells in the skin. This suggests that IL-15 plays a vital part in the immune process which causes psoriasis and could be a good target for new treatments [38].

IL-15 and Vitiligo

It was shown disruption in pro-inflammatory in addition to anti-inflammatory cytokines in the skin as well as blood of people with vitiligo such as IL-15, which considers a pro-inflammatory type. Its production increases during inflammation and it plays an important role in both the body's initial as well as long-term immune responses [39].

Here's how IL-15 might relate to vitiligo [17]:

Activation of Immune Cells: IL-15 may be helpful to the initiation and autoreactive T cells chemoattraction involving cytotoxic T lymphocytes (CTLs). In vitiligo, CTLs have been found to target melanocytes[40].

Promotion of Cytokine Environment: IL-15 can help create a cytokine environment that promotes melanocyte-specific CTLs survival and activity, leading to continued destruction of melanocytes [41].

IL-15 helps CT cells become more effective at killing harmful cells, making both CT cells and Th cells less likely to be stopped from working by regulatory T cells [42].

IL-15 promotes the survival and memory CT cells development which are responsible for vitiligo relapse [41].

Potential Treatment Target: Given its role in immune stimulation, targeting IL-15 signaling pathways could potentially help in modulating immune responses in vitiligo. This could involve using IL-15 inhibitors to reduce T cell activation and prevent further loss of melanocytes [43].

IL-15 considers a potent cytokine involved in the immune system activation, with implications in various diseases including vitiligo. Its role in the enhancement of immune responses implies that IL-15 could indirectly contribute to the autoimmune destruction observed in vitiligo. Future therapies might look at ways to inhibit IL-15 as a means to reduce or halt the progression of such autoimmune disorders [17, 44].

Kassab *et al*. (2023) [45] A study was done to check different markers of oxidative stress, along with related inflammation and immunity markers, in people with stable and active vitiligo, reported a positive correlation between IL-15 as well as VASI score. These results aligned with IL-15 aforementioned role in disease progression through maintaining autoreactive CT cells and tissue-resident memory T cells (TRM) in the skin, being directly involved in melanocyte destruction [17]. These cells accumulate in lesional skin and are maintained by IL-15 signaling, even in the active inflammation absence [46]. In patients with more extensive disease, a larger area of the skin is infiltrated by these cells, potentially leading to higher systemic IL-15 levels. This may explain why IL-15 correlates with the extent and severity of vitiligo rather than its activity.

Moreover, IL-15 has been shown to be implicated and overexpressed in several inflammatory as well as immune-mediated skin disorders like alopecia areata, psoriasis, Stevens-Johnson syndrome and severe epidermal necrolysis, as well as atopic dermatitis [38, 47-51].

All these findings shed the light on IL-15 role in the vitiligo pathogenesis and strengthen its role in disease progression and severity through its ability to maintain and activate TRM cells, which are now recognized as central players in autoimmune melanocyte destruction. Keratinocytes and dendritic cells in lesional skin may overexpress IL-15, particularly in response to oxidative stress and inflammatory stimuli. This overexpression leads to enhanced survival, proliferation, and cytotoxic function of TRM cells via IL-15 trans-presentation, resulting in sustained immune activity even in clinically stable lesions [52].

Furthermore, IL-15 enhances IFN-γ production in addition to other proinflammatory cytokines by TRM and NK cells, CT cells recruitment, enhancing their cytotoxicity as well as promoting maturation as well as memory CT cells survival. IL-15 also upregulates CXCR3 expression on T cells, promoting their migration to the skin where melanocyte-specific antigens are presented, perpetuating the autoimmune response [53]. These mechanisms collectively highlight IL-15 not only as a marker of inflammation but also as a driver of disease chronicity and recurrence, making it a promising target for immunomodulation in vitiligo.

Conflict of Interest

Not available.

Financial Support

Not available.

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