

“Prevalence of Psoriasis Among the Patients in Dhaka and Chittagong and Comparison of Cytokine Profiling and Association of Gut Microbiota”

Submitted by

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partial fulfillment of the requirements for the degree of Bachelor of Science
in Biotechnology

Mathematics and Natural sciences

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It is thus declared that:

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Dedication:

“This work is dedicated to my beloved parents and Nisarg who were always there for me whenever I needed them and to my dearest moynu who never failed to cheer me up whenever I was on the verge of giving up.”

Ranita Saha

“I dedicated this work to my parents for being an unyielding support system and constant motivation throughout this process. To my supporting teachers and mentors at BRAC University for providing me with invaluable guidance, patience and inspiration through my academic career. And finally, to my friends for supporting and loving through all of the ups and downs - your love, laughter and support, at times, made it more bearable! ”

Tufatun Nur Marjan

“ This work is dedicated to my grandmother (Buli), whose unwavering love and support have guided me to where I am today ”

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Background:

In recent times, one of the most acknowledged and common diseases that's seen among almost every person, is dermatological disease. The reasons behind the rapid spread of various skin diseases can be because of the changes in the environment and increased pollutants, changes in lifestyle factors of people that cause significant immune system dysregulation, or expeditious emergence of microbial threats. Psoriasis is one of the accelerating chronic, immune-mediated skin diseases, which is caused by genetic susceptibility, environmental factors, and immune dysregulation. Interleukin-17 is highly responsible as a mediator of inflammation in psoriasis. Interleukin- 6 on the other hand, contributes to inflammation, and immune regulation, interacts with the IL-17 pathway which further signifies the inflammatory response characteristics of psoriasis and most importantly, contributes to cancer and tumor development. One of the recent discoveries has stated the correlation between gut microbiota and skin health: gut-skin axis, which has further highlighted attention to the several aspects of systemic health and dermatological conditions along with how microbial imbalance contributes to inflammation. However, to acknowledge the specific mechanisms, and reasoning of psoriasis and develop more advanced treatments, more discoveries regarding these should be made.

Abstract:

Psoriasis is a non-contagious skin inflammatory condition characterized by erythematous, silvery scaling and itchy plaques which are seen commonly on regions of elbow and knee with bleeding spots at scraped off parts. The early explanatory models of psoriasis pointed to genetic and environmental factors as being able to explain the pathogenesis of the disease especially in familial predisposition or environmental exposures. This research aims to find a multi-dimensional insight of psoriasis to focus on the interaction between immune-transducing molecules, the dynamics of intestinal microbiome and environmental contextual factors which will further help in enhancing global awareness of the disease and covering information gaps.

According to the current evidence, constant inflammation is caused mostly by immunological dysregulation. Interleukin-17 (IL-17) has become the subject of interest to many researchers as it mediates critical immune signaling by influencing the activation process of keratinocytes and recruits immune effectors to the dermal surface to fuel the development of plaque and incite inflammation. IL-6 is locally and systemically active and promotes differentiation of Th17 lineage and upregulation of pathways mediated by IL-17. The gut microbiome further alters cutaneous health through the skin-gut axis. Preliminary findings from our research suggests an association between changes in the gastrointestinal microbial community (both temporary and chronic) and intestinal hyperpermeability, blunted immune stimulation, and systemic inflammatory diseases, which can either aggravate or trigger psoriasis. To detect the mechanism, serum cytokines levels were quantified by ELISA, whereas to detect the presence of *Helicobacter pylori* PCR was done for genomic DNA after isolating from human stool samples. These insights can be used to raise the awareness of its complexity and provide future approaches towards diagnosis and therapeutic intervention.

Keywords: Psoriasis, gut microbes, IL-6, IL-17, ELISA

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1. Introduction:

Psoriasis is a long-lasting, immunologic disease that comes in the form of erythematous, scalp, and usually pruritic plaques on different parts of the body. Though not being infectious, it produces visible symptoms, and has a long-term prognosis, thus becoming a socially and medically important issue for a psoriatic patient (Nestle, Kalpan, & Barker, 2009). The illness has not only been attached to physical pain but also psychological stresses like anxiety, depression, and seclusion resulting mainly because the disorder is characterized by stigma (Parisi et al., 2013). Although psoriasis has a long history in the developed world, recent epidemiological evidence shows that it is gaining momentum in developing countries including Bangladesh where there is a steady inclination in the skin clinics that more patients are now presenting with psoriatic patterns (Bhuiyan et al., 2020).

In the past, psoriasis was believed to be mainly genetically and environmentally induced. Those who have a family history of psoriasis or those who are exposed to various environmental triggers which are related to psoriasis are more susceptible to the disease, especially infections, stress, and trauma on the skin (Griffiths & Barker, 2007). Nevertheless, recent scientific opinion has changed significantly with an increase of references to immunological dysregulation as an essential pathogenic factor in the development and progression of psoriasis (Lowes, Suarez-Farinas, & Krueger, 2014). Particularly, cytokine imbalances have since been identified as a major cause of persistent inflammation that characterizes the disease.

Interleukin-17 (IL-17) is one of the cytokines that have gained a lot of research interest in relation to psoriasis because of its pivotal role in immune signaling. IL-17, a pro-inflammatory cytokine that is mainly produced by Th17 cells, triggers the activation of the keratinocytes and the binding of other immune cells to the surface of the skin. Thus, leading to the formation of the plaques and inflammation that are characteristic of psoriasis (Di Cesare, Di Megalio, & Nestle, 2009). Both the lesions and the psoriatic plaques consistently exhibit elevated levels of IL-17 and therefore are thought to be indicators of the severity of the disease (Prinz, 2019). Conversely, IL-6 is a local and systemic immune mediator that has also been shown to be important in the early differentiation of Th17 cells and therefore acts in an indirect manner to enhance IL-17-signalling pathways (Di Cesare, Di Megalio, & Nestle, 2009). Its role goes beyond psoriasis to other inflammatory and autoimmune diseases, e.g., rheumatoid arthritis and some malignancies, so it is a molecule of great biomedical interest (Campanati et al., 2021). Learning how these cytokines play a role in patients with psoriasis is likely to yield important information about the disease processes and may also help design more specific treatments.

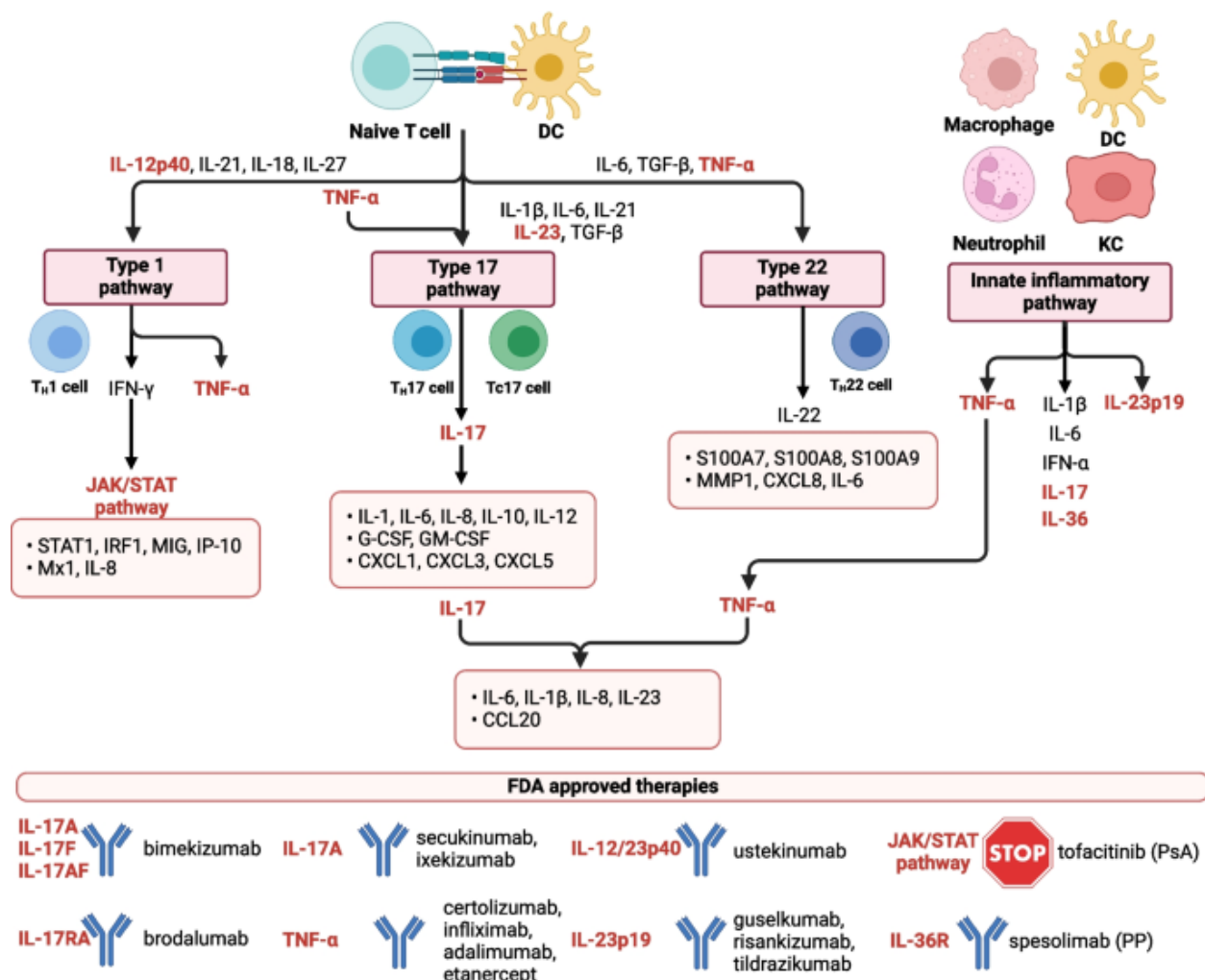


Figure 1: Immunology of Psoriasis; <https://link.springer.com/article/10.1007/s12016-024-08991-7>

Figure 1 shows the immunological pathways involved in psoriasis, highlighting the interaction between dendritic cells, T cells, and keratinocytes. The figure focuses on how pro-inflammatory cytokines such as TNF-α, IL-17, and IL-23 drive chronic skin inflammation and abnormal keratinocyte proliferation.

In recent years, there has been another addition to the study of psoriasis, how the microbiome in the gut can affect the health of the skin. The skin-gut axis sometimes also known as the gut-skin axis is the notion of a concept that suggests the proportion of microorganisms inhabiting the gastrointestinal tract can affect the immunity of the systemic body in a way that influences the physiology of the skin (Salem et al., 2018). The gut is colonized by a large and highly complex microbial community, which is in close contact with the immune system of the host. An imbalance

in this ecosystem, or dysbiosis, can predispose intestinal hyperpermeability, improper immune stimulation, and systemic inflammatory status. These processes can in turn aggravate or even cause inflammatory diseases of the skin such as psoriasis (Szanto et al., 2019).

A specific bacterium that has been raised in this respect is the *Helicobacter pylori*, a pathogen that is normally known to be related to gastric diseases, such as ulcers, and cancer of the stomach. Yet, recent evidence indicates that even extra-gastric pathologies may be influenced by *H. pylori* through immune reaction (Liatsos et al., 2022). Sustained *H. pylori* infection has been associated with an increased level of systemic inflammation and changes in the cytokine profile, which may also explain the immune dysregulation in psoriasis. *H. pylori* infection has been found at higher rates in psoriatic patients in some studies which has led to speculation of its involvement with disease flares (Yan et al., 2024). However, this association is not clearly established especially within people of South Asian origin who have a high burden of both infection and affliction by psoriasis but little is known about the association between the two.

Studying this intersection is a research gap in the Bangladeshi context because it is not apparent anywhere. Previous research has addressed the immunopathogenesis of psoriasis, however, not many studies have tried to combine both factors' cytokines and the role of the gut microbiota in the clinical aspect. In a bid to fill this gap, the present study was proposed to study these interrelated issues among patients in two of the major cities of Bangladesh: Dhaka and Chittagong. 600 members were enrolled in this study (300 members from each city). These people were exposed to thorough data collecting by means of structured questionnaires aimed at gathering information pertaining to demography, lifestyle habits, environmental exposures and medical history. It was expected to examine not only genetic and environmental factors of psoriasis but also social and behavioural factors that can contribute to disease development and the process of the disease. Biological samples such as peripheral blood and stool were obtained in addition to the survey data of selected patients and matched controls. The detection of IL-6 and IL-17 concentrations in blood samples was performed based on enzyme-linked immunosorbent readily forecasted as a sensitive process of revealing body protein biomarkers. These measurements gave this quantitative evaluation of systemic inflammation and immune activation among the psoriatic patients. At the same time, microbiological analysis of stool samples was carried out. Cultures of *H. pylori* were taken and counted using colony-based assays such as colony forming units (for CFU). Polymerase chain reaction (PCR) was used to verify the identity of the bacterial isolates and to evaluate genetic markers.

With such a combination of three layers of immunological profiles, microbial composition, and lifestyle/environmental background, this research will provide a multidimensional insight into psoriasis. The result should also show the interaction between immune signaling molecules such IL-6, IL-17 and the factors of the gut microbial like *H. pylori* colonization and how these interrelations are determined by the contextual factors of people of the Bangladeshi population. Such a strategy will not only assist in globalizing the world to understand more about psoriasis,

but also fill the loophole that greatly demands region specific data, which can be used in clinical practices and in safeguarding the well-being of the population in South Asia.

Long-term, the study is aimed at translating into workable upstream biological sources of intervention that can form the basis of future interventions to tackle the condition rather than treating them symptomatically. A better understanding of the underlying immune/ microbial mechanisms would help to make inroads in personalized medicine; where an individual immune/ microbial-based set of treatment strategies would be used. Besides, the study highlights the relevance of a systems-biology approach to dermatology, with such diseases as psoriasis no longer being perceived as responding to local issues in the skin region but rather as the sign of systemic dysregulation.

2. The Correlation Between Psoriasis and Inflammation: A Focus on IL-6 and IL-17

2.1 Introduction:

Systemic Immune-Mediated Disorder of Psoriasis

As an example, Psoriasis, in the past considered mainly as a skin chronic recurring illness, is later on well endorsed by the medical fraternity as a systemic inflammatory disease that has a multifaceted immunopathological foundation (Griffiths & Barker, 2007). On the one hand, its most recognizable symptoms, erythematous, scaly plaques, can still be considered the most apparent manifestations of the disease; however, on the other hand, the disease is intensely immune-dysregulated and has far-reaching implications beyond the skin (Nestle et al., 2009). Psoriasis is also thought to affect 2-3 percent of the world population, which is now manifested through lesions that appear on the head, knees, legs, lower back, but not restricted within the confines of the skin. In more progressive or mismanaged ones it can include inflammation of the joints (psoriatic arthritis) and is correlated with heightened risk to systematic complications like insulin resistance, cardiovascular pathology, metabolic syndrome (Parisi... This new conception of psoriasis has required a change in clinical and research thought, and it is currently considered as multisystemic with immune properties, as opposed to a cosmetic skin issue.

2.2 Immune Dysregulation in Psoriasis: The Pivotal Role of Cytokines

The central problem of the psoriatic disease is the maladaptive immune response, specifically the abnormal activity and the behaviour of the T-helper (Th) lymphocytes. Out of this, the subset known as Th17 has been of specific interest since it extensively contributes to pro-inflammatory signaling in both systemic and cutaneous scenarios. Th17 produces a distinct set of cytokines one of which is Interleukin-17 (IL-17) which is an important effector cytokine. At the same time, Interleukin-6 (IL-6) is a multifunctional cytokine secreted by immune and non-immune cells, and it is responsible for differentiating and stabilizing the Th17 lineage, forming a loop where elevated and persistent inflammation occurs (Di. Cesare et al.,2009)

IL -6, despite playing roles in many physiological processes, hematopoiesis, host defense, among others, is pathologically elevated in inflammatory diseases, of which psoriasis is one. It contributes to the differentiation of naive T-cells to Th17 cells and an increase in acute-phase proteins which explains its association to more than the localized skin inflammation of the condition such as fatigue and cardiovascular stress (Di. Cesare et al.,2009) . Increased levels of IL-6 have been shown to be consistent in psoriatic patients and tend to be associated with severity of the disease and thus can be used as a potential therapeutic target (Saggini et al., 2014).

The most predominant role of IL-17, which is secreted mostly by Th17 cells, is to initiate and maintain a psoriatic phenotype. It activates proliferation of keratinocytes in an abnormal way and promotes more pro-inflammatory factors, such as IL-1, IL-6, TNF-alpha and other chemokines. This causes a positive-feedback event of inflammation leading in turn to the characteristic scaling, erythema, and thickening of psoriatic lesions (Prinz, 2019). Moreover, IL-17 increases recruitment of neutrophils and promotes chronic immune activation, not only to skin but also to vascular and joint, thus the systemic nature of the disease in so many patients (Prinz,2019).

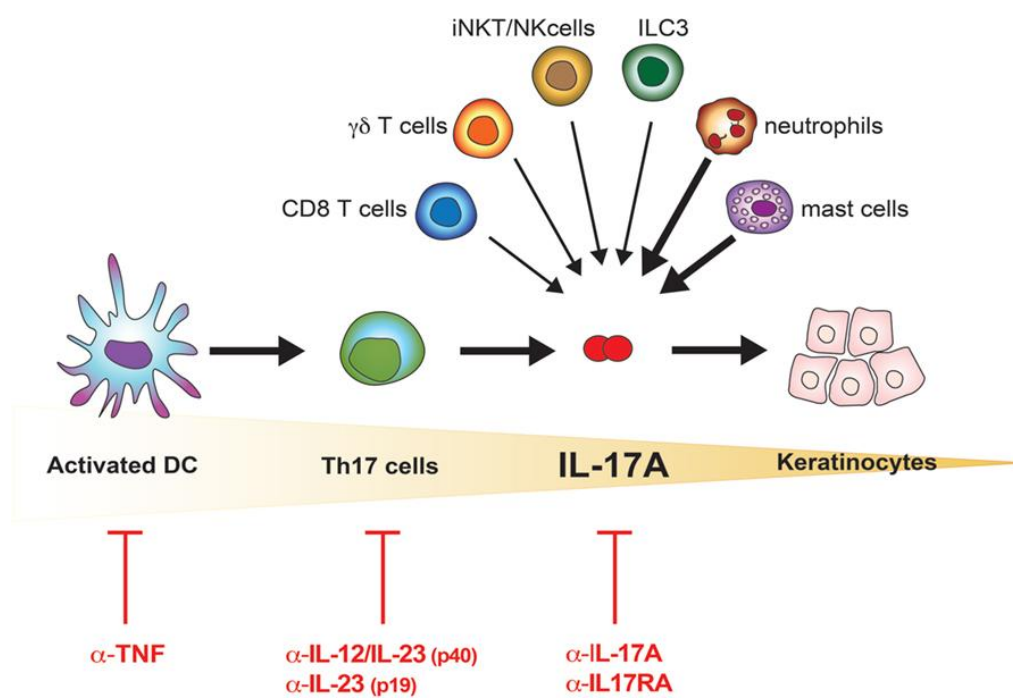


Figure 2: Function of IL-17 family in Psoriasis;

<https://www.frontiersin.org/journals/immunology/articles/10.3389/fimmu.2018.01682/full>

This figure shows the complex immune network in psoriasis, involving innate (iNKT/NK cells, ILC3, neutrophils, mast cells) and adaptive (Th17, CD8, $\gamma\delta$ T cells) immune cells that drive IL-17A-mediated keratinocyte activation. It also shows therapeutic targets such as anti-TNF, anti-IL-12/23, anti-IL-23, and anti-IL-17 agents that disrupt these inflammatory pathways.

2.3 Association of Cytokines with Systemic Disease

An excess production of IL-6 and IL-17 does not just lead to a local damage to the skin. Instead, these cytokines take an active part in psoriatic comorbidities formation. Chronic overexpression of IL-6 is related to endothelial dysfunction, which is the bone of atherosclerosis (Anyfanti et al., 2022). IL-17 has been reported to aggravate insulin resistance and cause metabolic disarrays (Oliveira et al., 2023). . These associations emphasize the importance of treating psoriasis not just on a surface level like skin but also with regard to overall health.

Additionally, it is also possible that the systemic inflammation that is the hallmark of psoriasis might also make the patients predisposed to autoimmune diseases like inflammatory bowel disease and rheumatoid arthritis where the same pattern of cytokine has also been noted (Sikora et al.,

2020). This convergence served to drive the notion that IL-6 and IL-17 are not restricted to psoriatic pathology alone, but are unifying agents of immune dysfunction in a number of disease conditions.

2.4 Therapeutic Implications of Treating IL-6 and IL-17

As they are so involved with the initiation and spread of diseases, IL-6 and IL-17 are now among the favorite targets in the creation of biological therapy. Particularly effective in the treatment of overall patients and decreasing lesions on the skin in individuals who have psoriasis, monoclonal antibodies that specifically bind IL-17 or its receptor, include secukinumab and ixekizumab. The additional advantage of these therapies over the old immunosuppressive agents is that they have less systemic side effects (Schwarz et al., 2020).

Likewise, in the case of IL-6, though therapies where IL-6 is approached directly (e.g., tocilizumab) currently have higher prevalence in such diseases as rheumatoid arthritis, their ability to find application in psoriasis is a major point of research. Given the importance of IL-6 in the differentiation of Th17 cells, these interventions may break early inflammatory-transforming, providing another route of obtaining treatment, especially with the systemic patient or comorbid rate (Parisi et al., 2025).

The development of cytokine specific therapies highlights the fact that psoriasis needs to be seen not only as a skin disease but as an immune mediated disorder but of systemic scope. Such a change of paradigm is reflected in therapeutics against IL-6 and IL-17 that provide tube patients with more accurate and promising management opportunities.

Psoriasis pathogenesis is entrenched in immune dysregulation, with the involvement of IL-6 and IL-17 cytokines being crucial to starting and maintaining an inflammatory response. Such molecules do not work locally in the skin but also make up a systemic inflammatory profile that heightens vulnerability to metabolic and cardiovascular comorbidities (Ridker, 2021). Getting to learn of their biological roles and clinical interpretations offers a very essential basis of coming up with more specific treatment interventions. The increased interest in these cytokines, scientifically and clinically is indicative of the general redefinition of psoriasis as not only a skin disease, but a systemic disorder needing multi disciplinary management.

3. Psoriasis as an Inflammatory Disease



Figure 3: Psoriasis at different sites

Figure 3 shows the clinical presentation of psoriasis at different body sites, including areas such as the scalp, elbows, fingers, feet, hand, knees, and trunk. The images highlight the characteristic erythematous plaques with silvery scales, red pustule-like spots demonstrating the variation in severity and distribution among patients.

3.1 Keratinocyte Hyperproliferation-Immune-Mediated Pathogenesis

Throughout most of the previous century psoriasis was mostly thought of as an overgrowth disorder of the keratinocyte and abnormal epidermal turnover. Cell hyperproliferation of keratinocytes was regarded as the key pathological hallmark, whereas immune response was regarded as secondary response. Novel developments in immunology have revolutionized this way of thinking (Zhou et al., 2022). First, the finding that T-cells are highly infiltrated in the psoriatic plaques and subsequently the identification of the proinflammatory cytokine tumor necrosis factor- α (TNF- α), interleukin-23 (IL-23), interleukin-6 (IL-6), and interleukin-17 (IL-17) has relegated psoriasis into the realm of immune-mediated inflammatory diseases (Fitch et al., 2007). This reclassification was a game changer in terms of research and clinical management and has formed the basis of the present-day immunological based therapeutic interventions that have revolutionized the results of the treatments.

3.2 Dendritic Cells and the Initiation of the Psoriatic Cascade

The pathogenic cycle of psoriasis in most cases starts with the activation of dendritic cells that are antigen presenting cells mediating the transitions between the innate and adaptive immune systems. The formation of these cells is provoked by an intertwining of genetic predispositions and environmental slanders (Schön, 2019). Mechanical trauma (the Koebner phenomenon), microbial infection, and psychological stress are some factors that have been found to play a key role in the initiation of the disease (Rendon & Schäkel, 2019). Once activated, dendritic cells release pro-inflammatory cytokines, notably IL-23. IL-23 plays a pivotal role in expanding and stabilizing the Th17 cell population, thereby amplifying immune activation within psoriatic lesions (Rendon & Schäkel, 2019).

Th17 cells, a distinct subset of CD4⁺ T-helper cells, are central orchestrators of the psoriatic immune response. Through their production of cytokines such as IL-17A, IL-17F, IL-21, and IL-22, Th17 cells initiate and sustain inflammatory processes. Each of these cytokines exerts a unique yet complementary effect—IL-17A and IL-17F directly stimulate keratinocytes and stromal cells to produce chemokines and recruit neutrophils; IL-21 enhances T-cell and B-cell activation; and IL-22 promotes keratinocyte proliferation and epidermal thickening (Korn et al., 2009). The cumulative effect is a self-perpetuating inflammatory environment that underlies both the clinical presentation and chronicity of the disease.

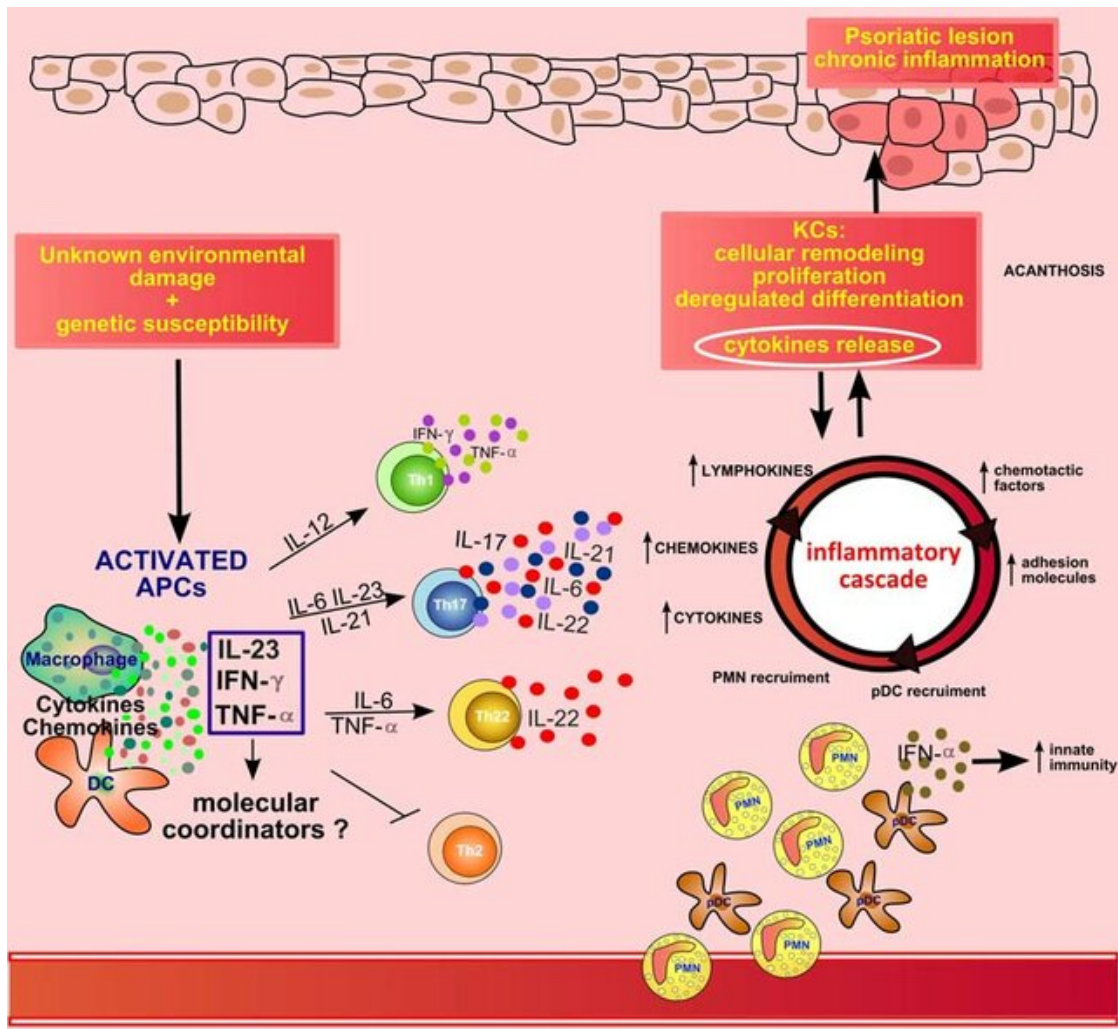


Figure 4: Inflammation cascade in Psoriasis;

https://www.researchgate.net/publication/51592687_A_Pathogenetic_Approach_to_Autoimmune_Skin_Disease_Therapy_Psoriasis_and_Biological_Drugs_Unresolved_Issues_and_Future_Directions/figures?lo=1

Figure 4 shows the inflammatory cascade in psoriasis, starting with genetic susceptibility and activation of antigen-presenting cells (APCs), followed by cytokine release (IL-12, IL-23, TNF- α , IL-6) and recruitment of immune cells. This cascade amplifies IL-17/IL-22-driven keratinocyte activation, neutrophil infiltration, and sustained chronic inflammation.

3.3 Keratinocytes as Active Participants in Inflammation

Traditionally, keratinocytes were viewed as passive targets of immune-mediated damage. In psoriasis, however, they actively participate in the inflammatory cycle, functioning not only as responders but also as amplifiers of immune signaling. Upon stimulation by cytokines IL-17, IL-22, and TNF- α , the keratinocytes secrete antimicrobial peptides (AMPs), including LL-37, which have a dual role of enhancing host protection and further activating the dendritic cells by pushing the formation of an immune complex (Kamata & Tada, 2023). On top of that, keratinocytes secrete chemokines [e.g., CXCL1, CXCL8] that attract neutrophils and other leukocytes to the skin, enhancing the level of inflammation (Piipponen, Li, & Landén, 2020). This communication signaling between keratinocytes and immune cells creates a very strong positive feedback loop that contributes to persistent inflammation and proliferative epidermal hyperproliferation, two of the key characteristics of psoriatic plaques.

3.4 The IL-6 Amplifying Psoriatic Inflammation

In such a system of cell interaction and cytokine signaling, IL-6 vividly plays a key role. In addition to its classical role in acute-phase responses, IL-6 facilitates the formation and maintenance of the Th17 cells by maintaining their differentiation and growth in the presence of IL-23 (Korn et al., 2009). This makes IL-6 a central enhancer to psoriatic inflammation and links the effects of innate immune signaling to adaptive responses (Schön, 2019). High levels of IL-6 in psoriatic patients not only enhance the local cutaneous inflammatory reaction, they also cause systemic symptoms, such as fatigue, malaise, and predisposing them to the rise in cardiovascular risk (Ridker, 2021). IL-6 secures the Th17 cell lineage and continues mechanisms of IL-17-mediated processes, a central path between localized psoriatic disorder and the systemic immune imbalance (Rendon & Schäkel, 2019).

The contemporary view of psoriasis places the disease in a complicated immunologic matrix in which dendritic cells, T-helper subsets, keratinocytes, and cytokines are engaged in a self-perpetuating expression of inflammation (Schön, 2019; Rendon & Schäkel, 2019). What was initially assumed to be a hyperactivity disorder of keratinocyte can be now described as a defect of a heavily impaired immune system. At the core of this, there are Th17-mediated IL-23 responses and the functional roles of the cytokines IL-6 and IL-17, which not only keep themselves in a state of psoriatic phenotype but also create systemic inflammatory mechanisms (Korn et al., 2009; Qu et al., 2013; Ridker, 2021).

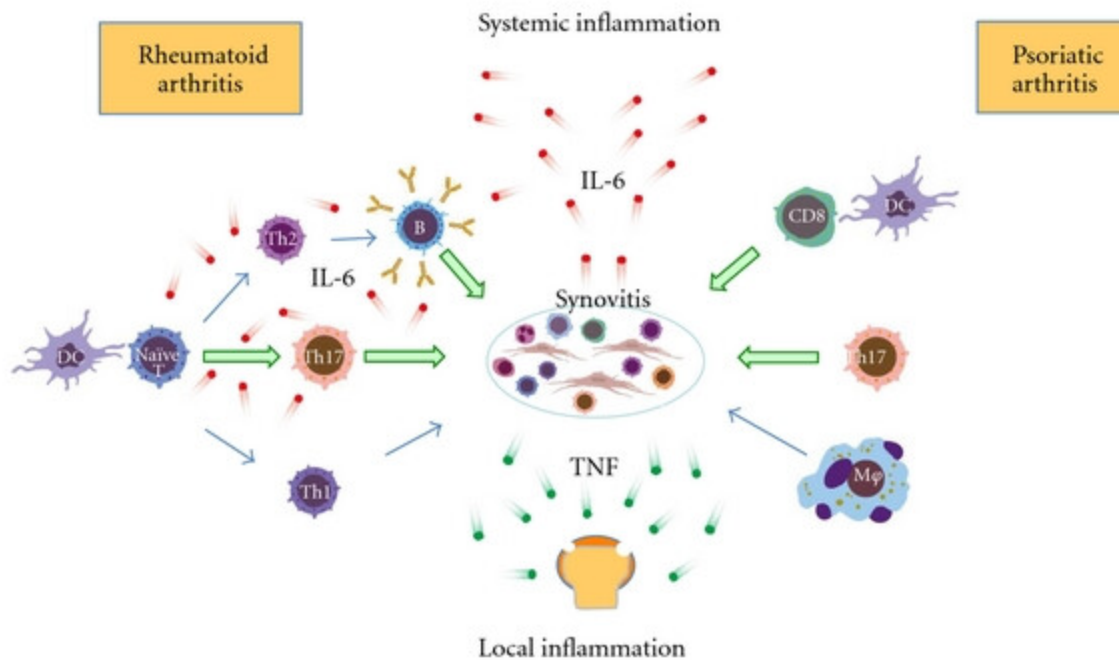


Figure 5: Role of IL-6 in Psoriatic Arthritis;
<https://onlinelibrary.wiley.com/doi/10.1155/2012/713618>

Figure 5 shows the role of IL-6 in psoriatic arthritis, showing how IL-6 promotes synovial inflammation, joint damage, and bone friction. It focuses IL-6 as a key mediator linking skin inflammation to musculoskeletal involvement in psoriasis.

4. Understanding IL-17: A Potent Driver of Cutaneous Inflammation

4.1 IL-17 Biology and Function

IL-17 refers to a family of cytokines (IL-17A to IL-17F), with IL-17A being the most studied member. It is secreted primarily by Th17 cells, though other immune cells such as $\gamma\delta$ T-cells, innate lymphoid cells (ILCs), and neutrophils can also produce it under inflammatory conditions. IL-17A binds to its receptor (IL-17RA/IL-17RC complex) found on a wide range of cells, including keratinocytes, fibroblasts, epithelial cells, and endothelial cells (McGeachy, Cua, & Gaffen, 2019).

Upon binding, IL-17 triggers a downstream signaling cascade that results in the production of pro-inflammatory molecules such as IL-6, TNF- α , IL-1 β , GM-CSF, and several chemokines (e.g., CXCL1, CXCL8). This cascade facilitates the recruitment of neutrophils and other immune cells to the site of inflammation (McGeachy, Cua, & Gaffen, 2019).

4.2 IL-17 in Psoriatic Skin

In psoriatic lesions, elevated levels of IL-17A and IL-17F have been documented in both lesional skin and patient serum. The cytokine acts directly on keratinocytes, inducing their hyperproliferation and causing the abnormal differentiation that results in the scaly plaques characteristic of the disease. The protective function of the skin is further compromised by IL-17, which also lowers the expression of filaggrin and other epidermal barrier proteins. IL-17 may promote the development of psoriasis by increasing expression of vascular endothelial growth factor (VEGF), a factor that leads to angiogenesis, a feature of psoriasis, and IL-17 has direct effects on the skin as well. It also promotes systemic inflammation which is the reason why comorbidities such as cardiovascular disease are common among the psoriasis patients (Mosca et al., 2021).

5. The Liaison Between Innate and Adaptive Immunity: IL-6

Introduction:

Using structural and functional approaches, the role of IL-6 in the body has been gained.

Interleukin-6 (IL-6) is a very versatile cytokine with a wide spectrum of functional diversities, and it is produced by a variety of cells including immune and non-immune cells. They include activated T lymphocyte, B lymphocyte, monocytes/macrophage, dendritic cell, fibroblast, endothelial cells, and even stressed keratinocytes in an inflammatory state. IL-6 is very important to coordinate the immune responses and it is one of the main mediators in connection between the innate and adaptive arms of the immune system (Tanaka, Narazaki, & Kishimoto, 2014). The biological effects of the drug cover a wide range of areas, such as enhancement of hepatocyte acute-phase protein production, control of the hematopoietic process, as well as the regulation of lymphocyte activity (Tanaka, Narazaki, & Kishimoto, 2014).

IL-6 acts in autocrine and paracrine mechanisms, which means that it is capable of influencing the surrounding cells or of strengthening its effects on the cell of its genesis. Its role in the maturation of lymphocytes is one of the most important ones in the given context of immune regulation. Specifically, IL-6 facilitates B-cell differentiation into antibody-producing plasma cells and

supports the expansion of T-cells under inflammatory conditions. Its involvement in T-helper cell lineage specification is especially significant in autoimmune and chronic inflammatory diseases such as psoriasis (Tanaka, Narazaki, & Kishimoto, 2014).

In particular, IL-6, in the presence of transforming growth factor-beta (TGF- β), promotes the differentiation of naïve CD4⁺ T-cells into Th17 cells—a subset known for producing the pro-inflammatory cytokine IL-17. This action not only enhances the inflammatory loop but also links IL-6 indirectly to the IL-17 pathway, thereby positioning IL-6 as an upstream regulator in the immunological architecture of psoriasis (Tanaka, Narazaki, & Kishimoto, 2014).

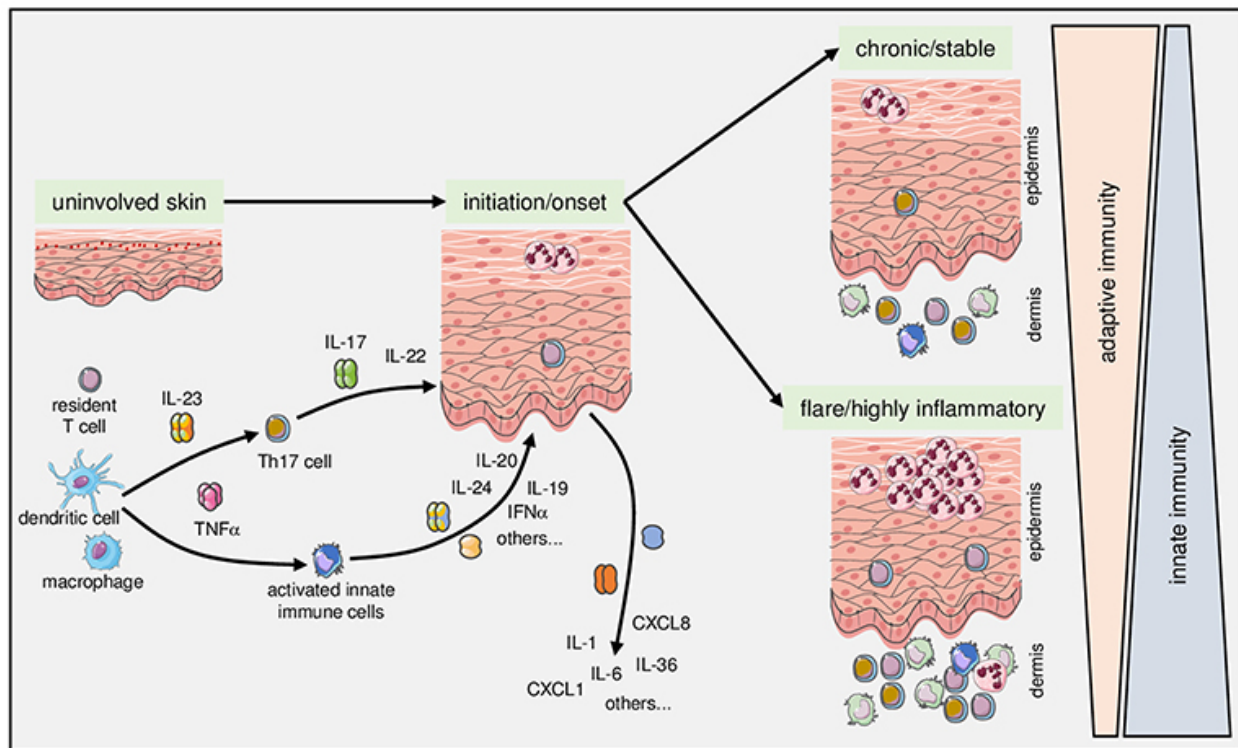


Figure 6: Adaptive and Innate Immunity in Psoriasis;

<https://www.frontiersin.org/journals/immunology/articles/10.3389/fimmu.2019.01764/full>

Figure 6 shows the interaction between adaptive and innate immunity in psoriasis. The diagram shows how resident T cells, dendritic cells, and macrophages initiate inflammation through cytokines such as IL-17, IL-22, IL-23, and TNF- α , while innate immune mediators including IL-1, IL-6, IL-36, and chemokines like CXCL1 and CXCL8 amplify the inflammatory cascade. Together, these processes drive keratinocyte hyperproliferation and sustain chronic psoriatic lesions.

5.1 IL-6 Expression and Function in Psoriatic Disease

Elevated IL-6 concentrations have been consistently reported in the peripheral blood of individuals with psoriasis. These heightened levels are often proportional to clinical indicators of disease severity, such as lesion size, body surface area affected, and patient-reported symptom intensity. Moreover, successful clinical treatment whether through systemic therapies or biological agents often results in a measurable decline in circulating IL-6 levels, reinforcing its role as both a driver and biomarker of psoriatic activity (Pietrzak et al., 2020).

Beyond its effects on the skin, IL-6 contributes significantly to the systemic inflammatory environment associated with moderate to severe forms of psoriasis. It has been linked to emerging insulin resistance, expanded adiposity and endothelial dysfunction upon its involvement. They are important elements of metabolic syndrome and cardiovascular disease, the conditions which often co-exist in patients with chronic inflammatory diseases (Andújar, Esplugues, & García-Martínez, 2022). In this way, IL-6 does not only potentiate cutaneous inflammation but also has a central position in the extra-cutaneous comorbidities that increase the burden of the disease in psoriatic persons.

The implications of IL-6 systemically give credence to targeting IL-6 as a long-term approach to manage skin lesions as well as a methodology that focuses on ameliorative measures against multi-system morbidities as a result of chronic systemic inflammatory responses. Its functionality in numerous tissues and apparatuses reasserts its position as a central molecular link between local skin disorder and multi-tissue involvement in psoriatic disorder.

6. A Synergistic Pathway IL-6 and IL-17

6.1 Molecular Interface and Amplification Circles

The interaction between IL-6 and IL-17 is defined as a synergistic rather than a sequential relationship. IL-6 triggers the process of differentiation and proliferation of Th17 cells, which effectively precondition the production of IL-17. Once released, IL-17 functions to maintain an inflammatory response through direct effects of the cytokine on the keratinocyte, which subsequently releases pro-inflammatory cytokines and enhances the local immune cell

recruitment. This cyclic procedure creates a self-sustaining loop, which feeds the chronic inflammation encountered in the psoriatic skin (Hou, Jin, Kang, & Kim, 2014).

In this loop, IL-6 and IL-17 are not independent of each other because they cannot work independently. The IL-6 stimulates cells (especially stromal and epithelial cells) to become more sensitive to the effects of IL-17 cytokine. Such sensitization leads to increased expressions of several inflammatory mediators, cytokines, such as chemokines (CXCL1 and CXCL8 or IL-8), and enzymes, such as matrix metalloproteinases (MMPs), which are involved in remodeling and degradation of the tissues. The synergistic effect of these cytokines helps not only in epidermal thickening and infiltration of leukocyte but also the increase of tissue damage and reinforces the pathologic circle. This molecular feedback is one reason why chronic psoriasis is resistant to treatment, though targeting individual cytokines, and proposes that their blockade might be therapeutically important, in addition to or alongside the inhibition of individual cytokines (McGeachy, Cua, & Gaffen, 2019).

Based on the results, the IL-6/IL-17 signaling axis is emerging to be a significant circuit within the immunopathogenesis of psoriasis. Treatment regimens targeting to suppress both cytokines simultaneously or prevent the interference of the respective pathways that enhance each of them together can be particularly promising in severe or treatment-resistant individuals. The continued exploration of this synergistic pathway holds promise for refining existing treatment modalities and developing next-generation therapies aimed at achieving durable remission in psoriasis.

6.2 Clinical Evidence

Clinical observations support this interaction. For example:

In psoriatic patients, those with higher IL-6 levels also tend to have higher IL-17 levels and more severe skin symptoms. Inhibition of IL-17 with drugs like secukinumab results in a drop in both IL-17 and IL-6 levels. IL-6-deficient mice fail to develop Th17 responses and show reduced susceptibility to psoriasis-like skin inflammation in experimental models. These findings reinforce the idea that IL-6 and IL-17 are mutually reinforcing agents in psoriatic pathology (Kolbinger et al., 2022).

6.3. Therapeutic Implications

Given the central role of IL-6 and IL-17 in psoriasis, both cytokines have become therapeutic targets. IL-17 Inhibitors: Drugs such as secukinumab, ixekizumab, and brodalumab target IL-17A or its receptor. These biologics have shown remarkable efficacy in clearing psoriatic lesions and reducing inflammation (Kolbinger et al., 2022; Mosca et al., 2021).

IL-6 Blockers: While tocilizumab, an IL-6 receptor blocker, is primarily used in rheumatoid arthritis, it has shown potential in treating psoriasis-related symptoms, especially when coexisting with psoriatic arthritis or metabolic issues (Tanaka, Narazaki, & Kishimoto, 2014; Hou, Jin, Kang, & Kim, 2014). Targeting these cytokines not only relieves skin symptoms but also reduces systemic inflammation, improving overall quality of life and lowering the risk of comorbid conditions.

7. The Broader Perspective: Systemic Inflammation and Comorbidities

7.1 Systemic Implications of IL-6 and IL-17 in Psoriasis

While the cutaneous manifestations of psoriasis are its most visible hallmark, the roles of IL-6 and IL-17 extend far beyond the skin. These cytokines contribute to a pro-inflammatory state that affects multiple organ systems, thus explaining the frequent co-occurrence of systemic comorbidities in psoriatic individuals (Andújar, Esplugues, & García-Martínez, 2022; Pietrzak et al., 2020; Tanaka, Narazaki, & Kishimoto, 2014). . This broader inflammatory burden is increasingly being recognized as an essential component of the disease's pathophysiology.

7.2 Cardiovascular Complications

Cardiovascular disease is one of the most dangerous of chronic psoriasis systemic consequences. Abnormally high levels of IL-6 have been linked to the occurrence of atherosclerotic plaque and endothelial cell dysfunction which are prerequisites of ischemic heart disease (Andújar, Esplugues, & García-Martínez, 2022; Ridker, 2021). Through surface expression of adhesion molecules and mobilization of monocytes into vascular tissues, IL-6 encourages the development of atheromas and leads to inflammatory processes with vessels. The combination of these effects and the chronic low-grade inflammation that is triggered by IL-17 form a microenvironment that favours cardiovascular pathology (Mosca et al., 2021; Hou, Jin, Kang, & Kim, 2014). There is no coincidence that patients with moderate-severe psoriasis are at an augmented risk of myocardial infarction and stroke and this is not conditional on prototypical risk factors of cardiovascular disease (Ridker, 2021) .

7.3 Metabolic Dysregulation and Obesity

IL-6 is also important in developing metabolic conditions such as the metabolic syndrome that includes such conditions as abdominal obesity, dyslipidaemia, high blood pressure and insulin resistance. Adipose tissue, especially visceral fat, is an endocrine active organ and this can secrete some pro-inflammatory cytokines, such as IL-6. With elevated IL-6, there is consequently elevated systemic inflammation that disrupts the insulin signaling pathways and attributes to glucose intolerance (Paniagua, 2016). This may be further worsened by IL-17, which enhances the

inflammatory state of adipose tissue and also seems to partake in reducing insulin sensitivity. The relationship between obesity and psoriasis is therefore a two-way one, i.e., inflammation causes metabolic dysfunction in psoriasis which in turn exacerbates the disease activity of psoriasis.

7.4 Neuroinflammatory Effects: Depression and Fatigue

Chronic systemic inflammation was also found to relate to neuropsychiatric symptoms in psoriatic patients. IL-6 and IL-17 can permeate the blood-brain barrier or indirectly affect it through the neurovascular pathways and immune ones. An over-activity of these cytokines was associated with higher risks of depression, cognitive disturbances, and chronic fatigue, which are common symptoms reported by patients despite the fact that skin lesions are kept under control (Hołodrowicz & Żebrowska, 2025). These results indicate that there is an increasing understanding that neuroinflammation is a significant but rather unrecognized sign of chronic immunologically based conditions, such as psoriasis.

This expanded thinking about the cytokine-mediated inflammatory fabric supports the idea that psoriasis is no longer a skin condition but a multi-system multi-faceted disease. The participation of IL-6 and IL-17 in various body systems indicates the possibility of using them as biomarkers of exposure to the systemic risk and as the targets to implement the comprehensive treatment.

Psoriasis immunopathogenesis has already taken a certain hold due to elaborate cytokine signaling processes, among which IL-6 and IL-17 serve as two of the most key disease progression factors. The presence of IL-6, which stimulates the synthesis of Th17 cells and is a major producer of systemic inflammation, qualifies as an upper-level regulator, whereas IL-17 is one of the strong downstream effector molecules, the contribution of which is to hyperactivate keratinocytes, drive cytokine-mediated feedback mechanisms, and attract more immune cells to the skin (Zhou, Chen, Cui, Shi, & Guo, 2022; Kamata & Tada, 2023; Mosca et al., 2021). . All these cytokines perpetuate the process of ongoing chronic inflammation characteristic of psoriatic disease. Notably, the effect of IL-6 and IL-17 is not limited to the skin. The fact that they manifest themselves in cardiovascular disease, metabolic syndrome and neuroinflammation symptoms has to do with the augmented systemic behavior of psoriasis. This realisation has not only changed the clinical knowledge of the disease but has changed the mode of treatment of the disease in a revolutionary way. Newer biologic treatments of these cytokines have been extremely successful not only alleviating signs and symptoms of dermatologic origin, but possibly preventing longstanding dangers of chronic inflammation.

With ongoing research especially in region specific settings (like Bangladesh), it can be anticipated that there will be more clarity to gather as far as local epidemiological patterns, profile of immune

response and microbiome interactions involved in psoriatic disease are concerned. This will be functioning in discovering better and individualized interventions to be taken on various populations. Conclusively, the improvement of basic knowledge of cytokine circuits such as ones associated with IL-6 and IL-17 holds not only hope of being able to more efficiently manage the disease, but also the potential of being able to make life in general easier, for sufferers of this complex and poorly understood disorder.

8. Gut Microbiota's Indirect Modulation of IL-6 and IL-17 in Psoriasis

Introduction:

The past couple of years have seen the human microbiome, especially the gut microbial ecosystem shaping up to be a key player in determining the regulation of the systemic immune responses. The effects of the intestinal lumen are amplified tremendously beyond it, and it determines host immunity, metabolism, and even neuroendocrine. Within a wide range of host-microbiota interactions, the so-called gut-skin axis is currently a popular idea in immunodermatology. There is growing data suggesting that the intestinal microbiota has the capacity to influence far away organ systems, including the skin, through the modulation of immune pathways that are recognized to be disrupted in the course of chronic inflammatory skin diseases like psoriasis. Specifically, the effect of the microbiota on the cytokines IL-6 and IL-17, which are complicit in the pathogenesis and systemic illnesses of the psoriatic disease, is of pertinence (Jimenez-Sanchez et al., 2025; Memariani & Memariani, 2025).

8.1. IMPORTANCE OF GUT MICROBIOTA IN PSORIASIS:

Gut microbiota has a central role in maintaining homeostasis in the immune system, metabolism, and epithelial barrier integrity. There is an emerging body of evidence to indicate that psoriasis, which has long been considered a skin-based immune-mediated inflammatory condition, is also a product of systemic factors, such as gut microbial composition. Changes in intestinal microbiota, also known as dysbiosis, have been linked to the loss of balance between anti-inflammatory and pro-inflammatory activity. In particular, microbial changes may encourage the activation of Th17 and Th1 cells and suppress the activity of regulatory T cells (Tregs), which leads to exacerbated inflammation typical of psoriasis. Further, the microbial metabolites including short-chain fatty acids (SCFAs) decrease in the dysbiotic condition and further worsen the anti-inflammatory mechanisms and skin protection. The gut microbiota of psoriatic patients offers important information about the pathogenesis of the disease and possible treatment options. Microbial diversity and abundance can be profiled to assist in identifying dysbiotic patterns that have a relationship to disease severity, progression, or response to treatment. These analyses can also

identify microbial biomarkers of susceptibility or flare-ups, which provide the promise of personalized medicine. Moreover, the characterization of microbiota opens the possibility of microbiome-specific therapy, including probiotics, prebiotics, dietary interventions or fecal microbiota transplantation, as a potential adjunctive approach to traditional immunomodulatory therapy. Therefore, the systematic analysis of gut microbiota is not only necessary to learn more about the pathophysiology of psoriasis, but also to develop new diagnostic and therapeutic strategies.

8.2. GUT- SKIN AXIS AND IMMUNE CROSSTALK IN PSORIASIS

The gut-skin axis can be defined as a multidimensional (bidirectional) communications system between the gastrointestinal tract and the skin based on immunological and metabolic mediators. To a great extent, this interaction is staged with the participation of resident microbes with their metabolic products, such as resident microbial surface antigens, that can contribute to local and systemic immunity. Dysbiosis of the intestinal microbiome has been implicated in the development of an array of autoimmune and inflammatory diseases, including that of psoriasis.

Research has established that psoriasis patients usually have diverse microbial alterations and composition. There is a decrease in beneficial genera like *Lactobacillus* and *Bifidobacterium* with anti-inflammatory properties. Specifically, in comparison, an overrepresentation of such pathobionts as *Escherichia coli*, *Clostridium* spp., and other pro-inflammatory species has frequently been reported (Codo\chezcode bl students cake rigid These disruptions among the microbes can stimulate the distortion of the immune signaling, which entails augmented secretion of IL-6 by antigen presenting cells, and consequently maturation of the T helper cell terminology into the Th17 category (Jimenez-Sanchez et al., 2025; Memariani & Memariani, 2025). This type of altered immune reaction leads to the accumulation of large quantities of IL-17, an effect that directly influences keratinocytes in order to maintain the presence of psoriatic plaques and hyperplasia in the epidermis(Martin et al.,2012) .

Widespread facilities of the South Asian population including those that live in urban areas like Dhaka and Chittagong could also influence these microbial imbalance further by local dietary and lifestyle patterns. A recent body of evidence indicates the dietary intake that is high in carbohydrate and low in fiber present in a majority of South Asian households is linked to reduced microbial diversity and this leads to a loss of short-chain fatty acid (SCFA)-producing bacteria (Hasan et al., 2021). Such alterations in the diet may disrupt the gut barrier and increase intestinal permeability, exposing the organism to more microbial constituents including lipopolysaccharides (LPS). It is thought that this phenomenon of a leaky gut can be seen as a priming aspect of both systemic inflammation and cytokine dysregulation.

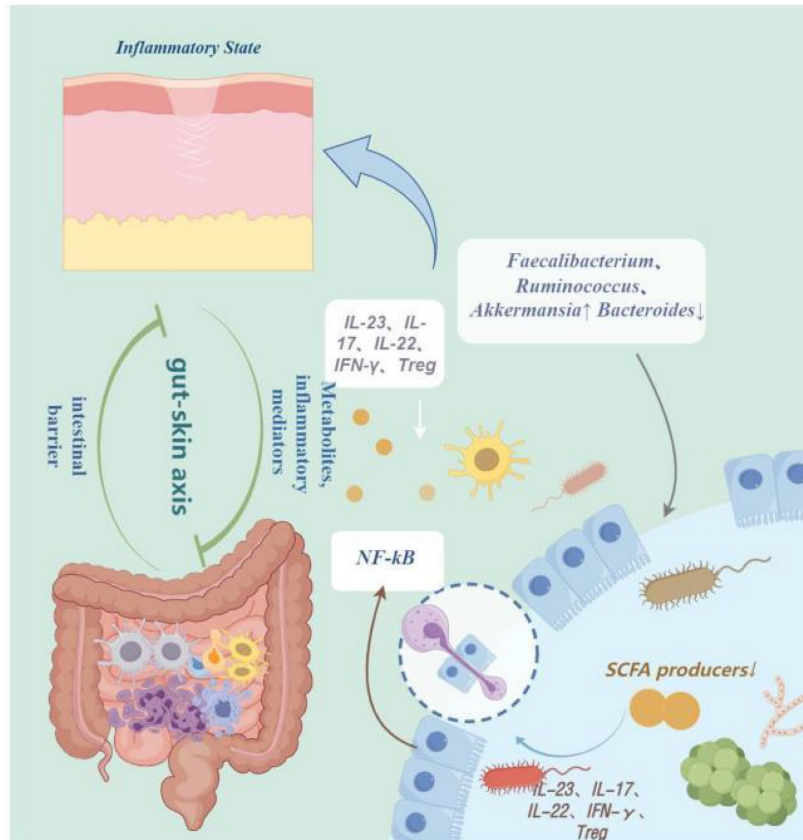


Figure 7: Pathogenesis of Psoriasis

Figure 7 shows the pathogenesis of psoriasis, highlighting the interplay between innate and adaptive immune responses. Activation of dendritic cells and macrophages leads to the release of cytokines such as IL-23, IL-17, IL-22 and TNF- α , which stimulate keratinocyte proliferation and sustain chronic inflammation, resulting in the characteristic psoriatic lesions

8.3. Th17 Cell Differentiation Must Have Occurred to Induce IL-17 by Microbes

The most important cytokine affected by the microbe activity in the gut is IL-17 produced by the Th17 cells that have a significant role in maintaining the psoriatic inflammation. Th17 cell maturation occurs in GALT whereby dendritic cells and macrophages process microbial immunogen and release IL-6, IL-12, IL-23. These cytokines, in turn, instruct naïve CD4⁺ T-cells to adopt a Th17 phenotype (Lee et al., 2020).

Although humans lack certain microbial species such as segmented filamentous bacteria (SFB), which are potent Th17 inducers in mice, analogous effects have been reported with human-

associated commensals. For example, strains of *Bacteroides fragilis*, *Akkermansia muciniphila*, and *Prevotella copri* have all been linked to increased IL-17 expression through direct interactions with immune cells in the intestinal mucosa. The IL-17 produced locally does not remain confined to the gut; instead, it can disseminate via the circulatory system and contribute to systemic inflammatory processes, including those occurring in the skin. This mechanism supports the clinical observation that Th17 cells and IL-17 are frequently upregulated in both intestinal and cutaneous tissues in psoriasis patients (Nogales et al., 2008).

Moreover, the gut microbiota can indirectly amplify IL-17 production by promoting chronic inflammation. Dysbiotic microbial communities may stimulate persistent activation of pattern recognition receptors (PRRs) such as Toll-like receptors (TLRs), which are expressed on dendritic cells. Under constant stimulation of these receptors, a continuous release of IL-6, IL-23 and other pro-inflammatory molecules keeps Th17 cells activated, and inflammation goes unresolved. In such a manner, the gut microbes not only act as the actors of the initiation processes of immune responses but also as the actors of the maintenance of pathological cytokines circuits.

8.4. Role of Microbial Metabolites: microbial short-chain fatty acids and immune homeostasis

In addition to their direct immune stimulatory capability, microbial metabolic products, such as short-chain fatty acids are key regulators of host inflammation. Acetate, propionate, and butyrate are the CFA metabolites produced through the fermentation by anaerobe bacteria of the intestines *Faecalibacterium prausnitzii*, *Eubacterium rectale*, *Roseburia* spp. These metabolites have various effects on the host cells such as epithelial cells, macrophages and T-cells to enhance immune homeostasis (Zhang et al., 2023).

As an example, butyrate has been shown to have anti-inflammatory effects, by promoting regulatory T-cells (Tregs)-mediated inhibition of overactive Th17. It also regulates the expression of the histone deacetylases (HDACs), thus affecting the immune cells gene transcription. As a result, when the SCFA contents decrease, as is the case with most patients who have psoriasis, the immunoregulatory equilibrium goes to disarray. This reduction in Treg ability and Th17 uncontrolled proliferation lead to an environment rich with cytokines which includes IL-17 and IL-6 which are both key to the development of psoriasis (Chen et al., 2019).

This way, the lack or depletion of microbes that form SCFA does not only deny the host the essential supply of anti-inflammatory cues, but also allows the emergence of a pro-inflammatory cytokine signature. Probiotics Conventional approaches used to treat inflammatory diseases include antibiotics and non-steroidal anti-inflammatories. However, studies have recently shown

potential of probiotics to be used as an adjunctive treatment in chronic inflammatory diseases, such as psoriasis. The nature of probiotics to restore SCFA producing bacteria and hence combat inflammation and oxidative stress has been explored widely (Zhang et al., 2023; Chen et al., 2019). The modulation of the systemic immunity via the influence of the gut microbiota, especially the IL-6 and IL-17 pathways, presents a breakthrough in terms of the understanding of the pathology of psoriasis (Jimenez-Sanchez et al., 2025; Memariani & Memariani, 2025).

Instead of the skin alone, there are various factors which affect the inflammatory processes that lead to psoriasis, and it is the dynamics that happen within the gastrointestinal tract. Gut bacteria influence the immune effects of the gut by presenting antigens, signaling via cytokines and metabolites, and either can enhance or suppress psoriatic inflammation.

In people with psoriasis, intestinal dysbiosis with the depletion of SCFA-producing commensals and concomitant overgrowth of pro-inflammatory species leads to the shifting of the immune reaction to the Th17/IL-17-dominated pattern. This is subsequently increased by the metabolites of microbes as well as intestinal permeability enabling the systemic circulation of inflammation signs. The gut microbiota therefore does not just provide background, but becomes a key factor in the pathogenesis of psoriasis as well as the episode (Buhaş et al., 2022; Zakostelska et al., 2016)

This mapping of microbial-immune interactions will be important to conduct future studies, particularly in population-specific situations such as the country of Bangladesh. These studies and the subsequent work may open the path to microbiota-based diagnostics and personalized treatment approaches to supplement the current set of interventions in psoriasis, thus bringing a more comprehensive approach to the management of the condition.

8.5. Gut Microbiota and IL-6 Modulation

IL-6 is a pleiotropic cytokine with both pro- and anti-inflammatory properties. In psoriasis, it primarily functions as a pro-inflammatory mediator that supports Th17 cell survival and contributes to **systemic inflammation**. Gut bacteria influence IL-6 levels directly and indirectly.

Lipopolysaccharides (LPS), components of the outer membrane of gram-negative bacteria like *E. coli*, activate Toll-like receptors (especially TLR4) on macrophages and dendritic cells, triggering the **NF-κB signaling pathway** that upregulates IL-6 production (Kawai & Akira, 2010). In psoriatic patients with gut dysbiosis, the overrepresentation of LPS-producing bacteria can lead to chronically elevated IL-6 levels.

Additionally, increased **intestinal permeability**—often referred to as "leaky gut"—enables microbial fragments like LPS and bacterial DNA to enter systemic circulation. These circulating pathogen-associated molecular patterns (PAMPs) further stimulate innate immune cells in

peripheral tissues to secrete IL-6, contributing to systemic and cutaneous inflammation (Mu et al., 2017). This process may be accelerated by environmental and dietary factors prevalent in Bangladesh, such as frequent antibiotic use, poor sanitation, and high intake of processed carbohydrates.

Conversely, certain bacterial species exert anti-inflammatory effects by **suppressing IL-6 production**. *F. prausnitzii*, for instance, produces butyrate and other anti-inflammatory molecules that inhibit IL-6 transcription via the STAT3 and NF- κ B pathways (Qin et al., 2010). A deficiency of these bacteria, which has been observed in both IBD and psoriasis, further supports the role of gut microbes in regulating IL-6.

8.6. Environmental Factors in Bangladesh and Their Relevance

The combination of low-fiber, high-starch diets, regular exposure to antibiotics (as often as not used without medical prescription), and the lack of ready access to clean drinking water means that in Bangladesh, an environment is created where gut dysbiosis is promoted. Additionally, recent data on tertiary hospitals in Dhaka and Chittagong show that the gastrointestinal symptoms are prevalent in psoriasis patients but they are not addressed during the medical examination (Islam et al., 2023). These results indicate the necessity to evaluate gut health as a method of managing psoriasis in this area.

Besides, the stress that is a reported psoriasis trigger and a regulator of the gut microbiota complicates the role. Stress alters gut barrier integrity and reduces microbial diversity through the hypothalamic-pituitary-adrenal (HPA) axis, indirectly increasing IL-6 and IL-17 production (Gareau et al., 2008). The psychosocial burden of chronic disease, especially in low-resource settings, could therefore feed back into the gut-skin axis and worsen clinical outcomes.

8.7. Therapeutic Implications

Understanding the influence of gut microbiota on IL-6 and IL-17 has opened up new avenues for potential **non-pharmacological interventions**. In pilot studies, **probiotic supplementation** with strains like *Lactobacillus plantarum*, *Lactobacillus rhamnosus*, and *Bifidobacterium infantis* has shown promise in reducing inflammatory markers in both psoriasis and IBD patients (Groeger et al., 2013). These probiotics may work by restoring microbial diversity, strengthening gut barrier function, and lowering cytokine production.

Other emerging strategies include **prebiotic fiber supplementation**, designed to enhance SCFA production and promote the growth of beneficial bacteria. In rural areas of Bangladesh, incorporating **traditional fiber-rich diets** (e.g., lentils, leafy greens, green bananas) may provide a culturally appropriate and cost-effective approach to restoring gut microbial balance.

Lastly, the use of **personalized microbiome analysis** could help tailor treatment approaches based on individual microbial and cytokine profiles. Such precision-based care models, though currently expensive, may in the future become accessible and relevant in the South Asian context as technology becomes more affordable.

9. *Helicobacter pylori* and Its Inflammatory Correlation with Psoriasis: A Complex Interplay

Introduction:

Psoriasis, a chronic, immune-mediated skin condition, manifests with a wide range of clinical phenotypes, the most prominent being erythematous plaques with silvery scales. The pathology of psoriasis extends beyond superficial dermal alterations and deeply involves systemic immunological dysregulation. One underexplored area that has gained recent scientific attention is the potential link between **chronic microbial infections**—notably *Helicobacter pylori*—and the exacerbation of psoriatic symptoms. While *H. pylori* is historically known for its gastrointestinal implications, such as peptic ulcers and gastric carcinomas, mounting evidence suggests it may influence immune and inflammatory pathways in organs beyond the gastrointestinal tract, including the skin.

9.1 Understanding *Helicobacter pylori*: Pathogenicity and Immunological Footprint

Helicobacter pylori, a Gram-negative, helical bacterium, has evolved intricate survival strategies enabling it to inhabit the acidic gastric mucosa with remarkable persistence. This microbe employs a plethora of virulence strategies most importantly its ability to secrete a urease to destroy stomach acid, delivery of cytotoxins like CagA (cytotoxin-associated gene A) and VacA (vacuolating cytotoxin A), and its motility which is provided by flagella to avoid immune clearance and settle in chronic colonization. *H. pylori* is contracted at an early age and typically lives within the body without causing any indications of its own presence throughout several decades without causing any signs and symptoms. Even so, it sets up a long term, quiet inflammatory noise environment which can have downstream implications far beyond the gastric lining.

During the colonization process, *H. pylori* binds to epithelial cells of the stomach leading to a series of immunologic reactions. Neutrophils, monocytes, and several subsets of T- cells are also attracted to the location of infection where they are a part of the cascade of cytokines secreted, many of which are associated with inflammatory cascades. Among these, IL-6 and IL-17 have

emerged as particularly relevant not only to the mucosal inflammation associated with *H. pylori* infection but also to extra-gastric inflammatory conditions, including psoriasis.

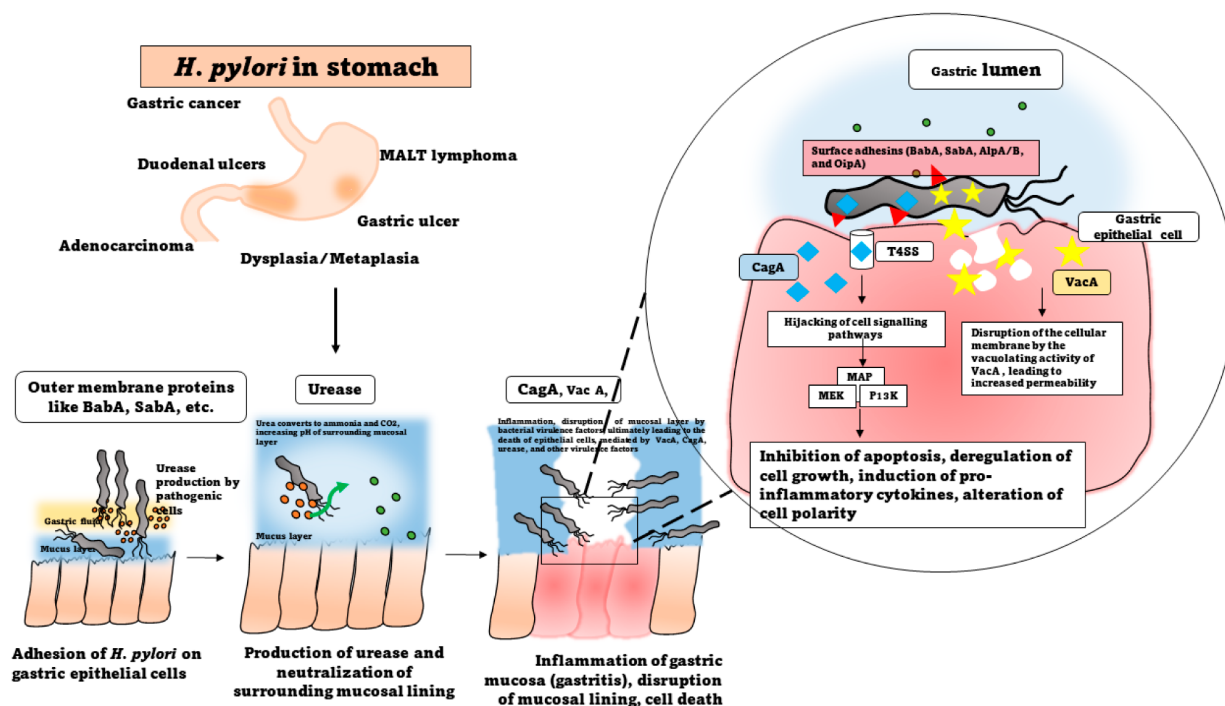


Figure 8: Pathogenesis of *H. pylori*; <https://www.mdpi.com/2076-2607/12/1/222>

Figure 8 shows the pathogenesis of *H. pylori* infection, showing how the bacteria colonize the gastric mucosa, produce virulence factors like CagA and VacA, and trigger an inflammatory response. This can cause epithelial damage, immune activation, and with time it might cause gastritis or ulcers.

9.2 Intersecting Cytokine Profiles: Psoriasis and Beyond

Psoriasis is increasingly recognized as a systemic inflammatory disorder rather than a condition restricted to the skin. The hallmark plaques and erythematous lesions are but surface indications of profound immune dysregulation. The Th17 axis at the very center of this dysregulation is what

governs an immune response in many autoimmune disorders and chronic inflammatory diseases. In this pathway, IL-17 is a central effector molecule that causes the hyperproliferation of keratinocytes and subsequent production of chemokines, antimicrobial peptides and other pro-inflammatory mediators. IL-6 has been described as transducing immunity between innate and adaptive immunity and promotes variation of naïve T cells into Th17 cells, which promotes the perpetuation of the inflammatory cycle.

Emerging evidence suggests that the chronic, low-grade inflammation sustained by *H. pylori* may not remain confined to the gastric tissue. There are similarities in the outcome of psoriatic patients and infected patients with respect to elevated systemic IL-6 and IL-17 concentrations leading to a potential of immunological convergence between the two conditions. The mentioned cytokines do not only lead to the local tissue impairment but also to the systemic inflammatory condition which is related to metabolic syndrome, cardiovascular pathology, and autoimmune patterns. Therefore the immunological effects of *H. pylori* infection can have a very wide range that can lead to being a co-factor and /or adjuvant in diseases such as psoriasis.

IL-6 is rather a multifunctional cytokine, which regulates immune response, acute-phase reaction and hematopoiesis. Its dysregulation contributes to chronic inflammation and has been implicated in various autoimmune conditions including psoriasis, rheumatoid arthritis, and systemic lupus erythematosus. Importantly, IL-6 is known to facilitate the differentiation of naïve T-cells into Th17 cells in the presence of TGF- β , further linking it with the overexpression of IL-17 and perpetuation of inflammation.

10. The Gut-Skin Axis: A Bi-Directional Channel of Influence

Emerging research underscores the **gut-skin axis**, a bidirectional communication system where gut health significantly influences skin homeostasis and vice versa. Within this framework, microbial dysbiosis in the gastrointestinal tract is increasingly being examined as a contributing factor in the pathogenesis and progression of dermatological conditions, including psoriasis.

H. pylori, though a gastric pathogen, can disrupt gut microbial homeostasis, leading to systemic immune activation. Through mechanisms involving molecular mimicry, immune cell trafficking, and cytokine overproduction, it potentially primes the immune system towards an inflammatory phenotype characteristic of psoriatic disease. Several epidemiological studies have indicated a higher prevalence of *H. pylori* infection in psoriatic patients compared to non-psoriatic controls, especially in moderate to severe cases. However, this association is complex and modulated by host genetics, environmental triggers, and microbial virulence factors.

11. Mechanistic Insights: *H. pylori*, IL-6, and IL-17

The inflammation triggered by *H. pylori* is sustained by the activation of **nuclear factor-kappa B (NF-κB)** and **signal transducer and activator of transcription 3 (STAT3)** pathways. These molecular cascades are critical drivers of IL-6 and IL-17 production. The persistent immune response to *H. pylori* infection leads to the systemic dissemination of inflammatory signals which can influence distant tissues, including the skin.

11.1. IL-6 Induction by *H. pylori*:

Upon infection, *H. pylori* activates gastric epithelial cells and resident immune cells to release IL-6. This cytokine not only promotes local gastric inflammation but enters systemic circulation, contributing to the inflammatory burden elsewhere in the body. IL-6's ability to stimulate acute-phase proteins, including C-reactive protein (CRP), and facilitate the differentiation of Th17 cells, links it directly with the immunopathogenesis of psoriasis. Elevated IL-6 levels have been consistently reported in psoriatic lesions and sera of affected individuals, and their correlation with disease severity has been noted in multiple studies.

11.2. Th17 Response and IL-17 Overexpression:

H. pylori infection is also associated with an increase in Th17 cells and their signature cytokine, IL-17. The presence of bacterial components such as lipopolysaccharides (LPS) and peptidoglycans activates pattern recognition receptors (PRRs), including toll-like receptors (TLRs), which in turn signal for IL-17 production. This cytokine, when overproduced, amplifies tissue inflammation by recruiting neutrophils and inducing keratinocytes to secrete pro-inflammatory mediators. This mechanism is mirrored in psoriatic pathology, where IL-17 drives keratinocyte hyperproliferation and sustains inflammatory circuits within the skin.

12. METHODOLOGY:

The research methodology involves the systematic techniques used to gather data, analyze it, and derive findings related to the subject under investigation. A research technique is a crucial element in the study's plan. The creation and establishment of a systematic approach is a crucial stage in attaining the objectives of a research effort. Using an effective and dependable research methodology assures that conclusions are grounded in sound scientific principles. Furthermore, the detailed aspects of the strategy enable researchers to maintain focus, leading to a more streamlined and manageable process overall. The researcher's methodology explains the cognitive processes and systematic approaches that resulted in the study's conclusions.

12.1 Aim of the study:

- To isolate *H. pylori* and compare the inflammation rate between Psoriasis infected and non-infected individuals.
- To quantify IL-6 and IL-17A cytokines in blood serum using ELISA.
- To compare cytokine levels between Psoriasis infected and non-infected individuals.
- To evaluate the potential of IL-6 and IL-17 as biomarkers of Psoriasis related inflammation.

12.2 Ethical statement:

Participants and/or legal guardians are given verbal permission for them to take part in the investigation (in this instance of children). The participants were given extensive information regarding the study's methodology and its objectives. The identity of the respondents was also concealed.

12.3 Area of study and participants:

The survey was conducted at two hospitals from two different cities and the samples were taken from Kurmitola General Hospital. The research was conducted from September 15, 2024 to April 20, 2025. Prior to beginning any data and sample collection, the relevant authorities were consulted and provided authorization. We were granted access to patient records and given permission to communicate with attending physicians and other hospital staff. The survey included a total of 602 patients: 300 from Dhaka and 302 from Chittagong. The survey primarily consisted of patients who received medical care at the institution. The age of the patients varied from the stage before reaching sexual maturity to the period long beyond the cessation of menstruation. The inclusion of participants from various socioeconomic backgrounds and educational levels enhances the study's comprehensiveness. In order to be eligible for the study, the patient must be confirmed of

the disease by the doctors of the Dermatology department. Among 300 patients from Dhaka, blood and stool samples of 50 patients were collected.

12.4 Questionnaire:

An organised questionnaire was formed to gather data from the participants. The questionnaire included information on age, gender, first symptoms, time of onset of symptoms, family history of similar conditions, severity of symptoms, and sites affected by sun exposure. It also assessed the effect of stress or anxiety, the presence of joint pain, seasonal variation in symptoms, co-existing conditions such as diabetes, and features like skin thinning or bleeding. Participants were further asked about the use of herbal medicines including their effect, history of other diseases or medications, food triggers, irritation during showering, and medications previously prescribed for psoriasis or related symptoms.

12.5 Statistical Analysis:

All the collected data were coded, input into the computer and analyzed by SPSS software programme 25 version.

DHAKA

Age

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	<20	34	11.3	11.3	11.3
	>60	38	12.7	12.7	24.0
	20-30	56	18.7	18.7	42.7
	30-40	61	20.3	20.3	63.0
	40-50	65	21.7	21.7	84.7
	50-60	46	15.3	15.3	100.0
	Total	300	100.0	100.0	

Table 1.1: Frequency table of current age of individuals in the the sample

The table 1.1 presented indicates that the individual patients who participated in the survey were in the age range of <20, >60 to 70 years, with a percentage of 11.3%, 12.7%, 18.7%, 20.3%, 21.7%, 15.3%, 14.6%, .3 respectively. According to the frequency distribution table, it can be observed that the most affected patients are in the age range of 40-50 years; and the least number are less than 20 years.

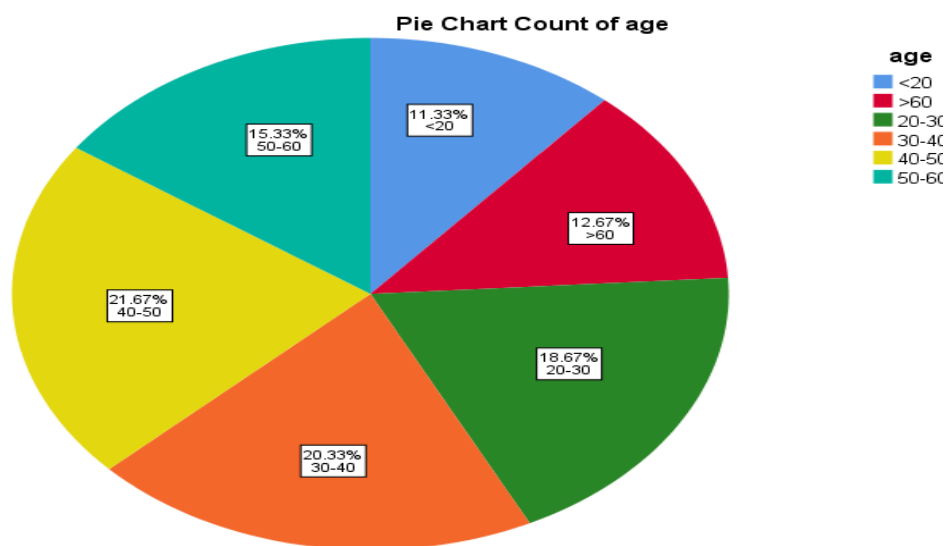


Figure 1.1: Distribution of percentage of age range that were affected by psoriasis

The figure 1.1, pie chart indicates the percentage of age range that were affected by psoriasis. Here, <20 years were 11.33%, >60 years were 12.67%, 20-30 years were 18.67%, 30-40 years were 20.33%, 40-50 years were 21.67%, 50-60 years were 15.33%. Here, the chart indicates that the majority age

range of the patients was 40-50 years; 21.67% and the minority of the patient age range was <20 years which was only 11.33%.

Gender

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	female	45	15.0	15.0	15.0
	male	255	85.0	85.0	100.0
	Total	300	100.0	100.0	

Table 1.2: Frequency table of gender individuals in the sample

The table 1.2 indicates that a significant percentage of individuals 85.5% are male, which indicates that the sample contains larger male patients than the female patients. Whereas there are about 15.0% of female patients. In contrast, the percentage of male patients is higher than the female patients.

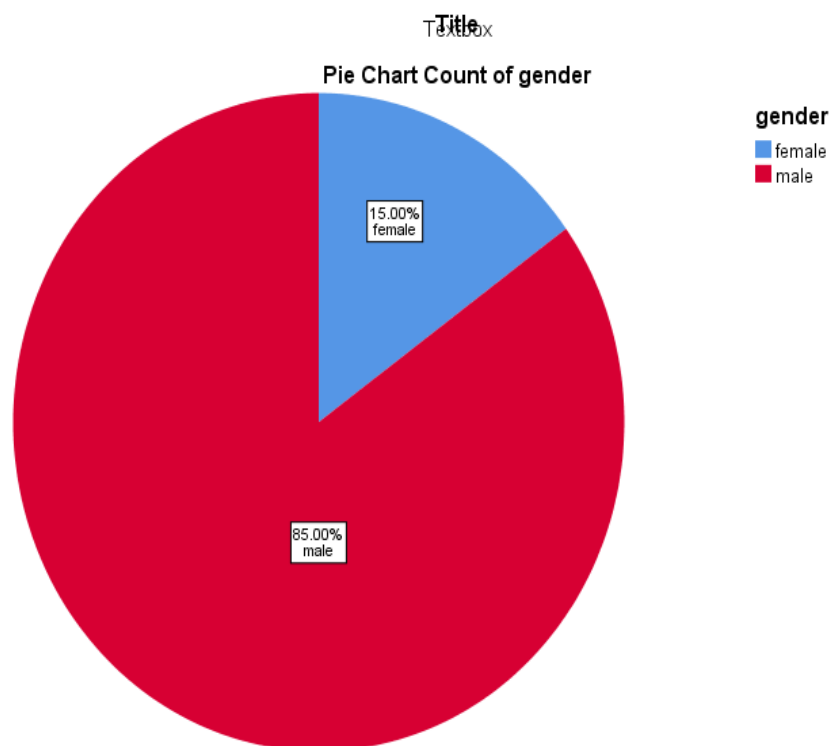


Figure 1.2 : Distribution of percentage of gender that were most affected by psoriasis

The figure 1.2, represents the gender most affected by Psoriasis. Among the patients 85.00% of patients were male and only 15% female.

Starting of symptoms

	Frequency	Percent	Valid Percent	Cumulative Percent
Valid	2	.7	.7	.7
<1 year	64	21.2	21.2	21.9
<6 months	12	4.0	4.0	25.8
>2 years	154	51.0	51.0	76.8
1-2 years	70	23.2	23.2	100.0
Total	302	100.0	100.0	

Table 1.3: Frequency table of start of symptoms in the sample

The table 1.3, represents the frequency of duration of the patients suffering from the diseases. A substantial portion of the individual patients, 51.0% suffering for more than 2 years, and only 4% of the patients were suffering for less than two years. On the other hand, 21.2% of the patients suffered for less than one year and 23.2% of the patients suffered for 1-2 years. It indicates the patients are mostly suffering from Psoriasis for more than 2 years, since Psoriasis is a long-term or chronic disease.

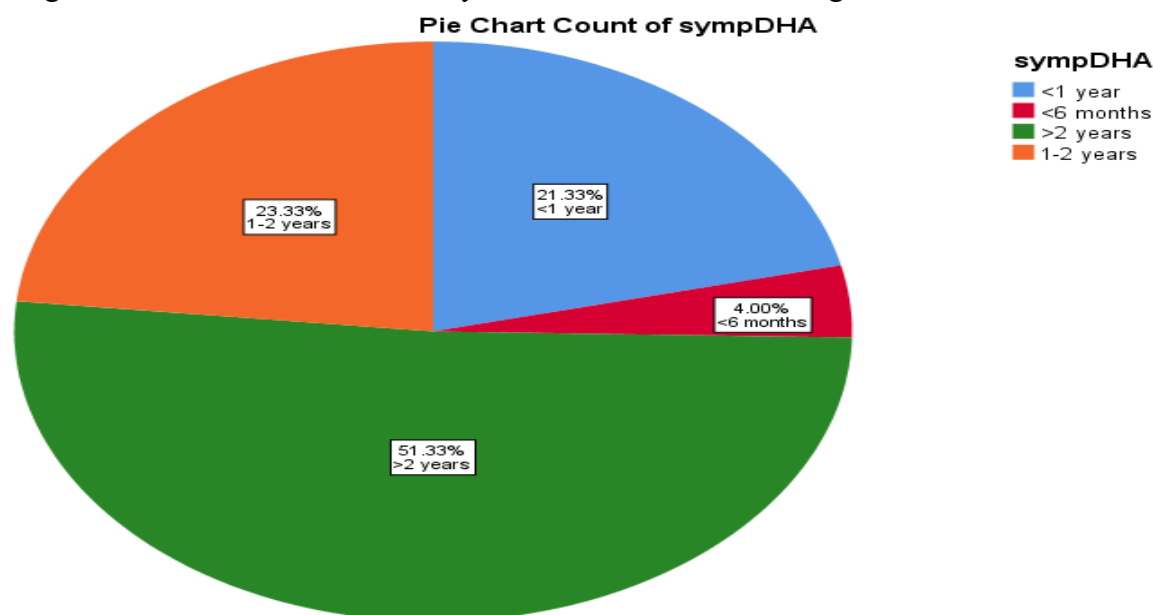


Figure 1.3 : Distribution of the percentage of duration of the patients suffering from psoriasis

The figure 1.3 of the pie chart represents the percentage of duration of the patients suffering from the diseases. A substantial portion of the individual patients, 51.33% suffering for more than 2 years, and only 4% of the patients were suffering for less than 6 months. On the other hand, 21.33% of the patients suffered for less than one year and 23.33% of the patients suffered for 1-2 years.

Severity

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Mild	130	43.3	43.3	43.3
	Moderate	104	34.7	34.7	78.0
	Severe	66	22.0	22.0	100.0
	Total	300	100.0	100.0	

Table 1.4: Frequency table of severity of a condition in the individuals

Table 1.4 represents a substantial portion of the individual patients, 43.3% have a mild condition, 34.7% of patients have moderate condition. However, there is a smaller group of patients, representing 22.0% of the sample, who have severe conditions. Therefore, the individual patients that participated in the survey had mild conditions followed by moderate conditions and severe conditions were low in number.

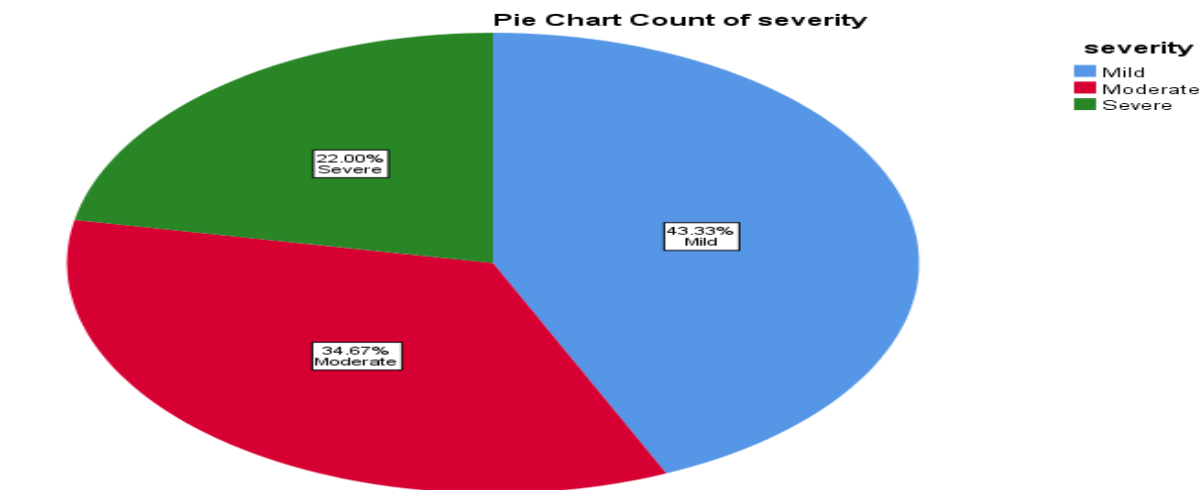


Figure 1.4 : Distribution of the percentage of severity of psoriasis

The diagram 1.4 represents the percentage of severity of the patients. It indicates that the majority of the patients were in mild condition, which was 43.33%. Again, 34.67% of the patients had moderate condition and 22.00% of the patient had severe conditions. It interpret that the majority of the patients

participated in the survey is suffering from the mild condition, followed by moderate condition and severe condition is comparatively low in number

Affected by sun

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	no	154	51.3	51.3	51.3
	yes	146	48.7	48.7	100.0
	Total	300	100.0	100.0	

Table 1.5: Frequency table of affected by sun

The table 1.5 represents the percentage of patients who get affected by exposure to sun. Most patients of 51.3% are not affected by sun, whereas 48.7% of patients are affected by exposure to sun. Therefore it can be interpreted that psoriasis is not triggered by the sun in a high number of people.

Joint pain

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	no	156	52.0	52.0	52.0
	yes	144	48.0	48.0	100.0
	Total	300	100.0	100.0	

Table 1.6: Frequency table of joint pain among patients

Table 1.6 indicates that 52.0% of patients claim they do not have any joint pain, which is the dominant response. However, 48% of patients have joint pain. Here, the majority of the patients claim that they are not suffering from joint pain, and the patients with joint pain are comparatively low.

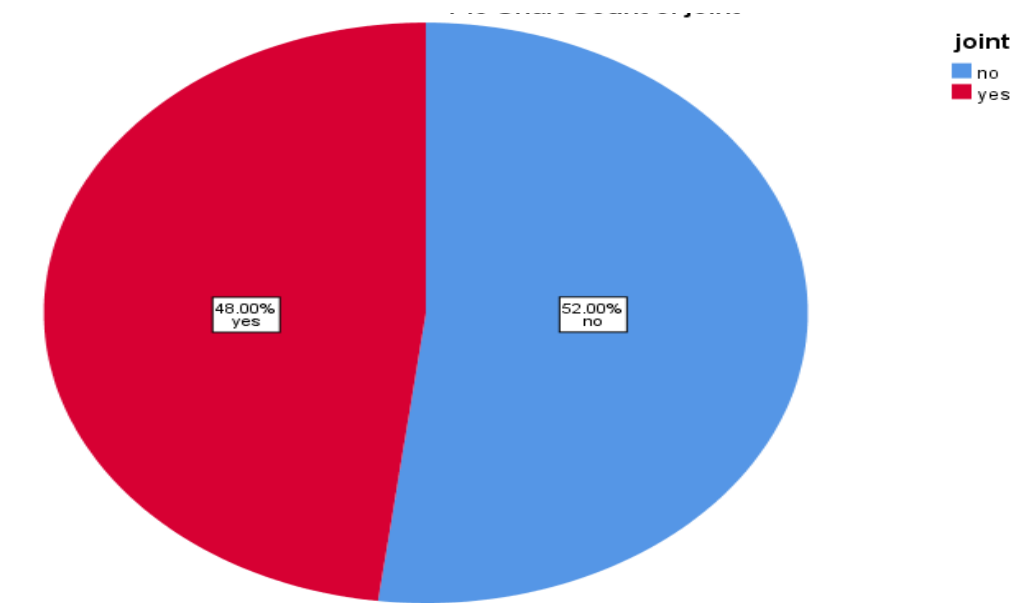


Figure 1.5: Distribution of the percentage of presence of joint pain in psoriasis patients

The figure 1.5 represents whether the psoriasis patient had joint pain or not. Here, only 48.00% of the patients had joint pain and the other 52.00% of the patients had joint pain.

Diabetes

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	no	239	79.7	79.7	79.7
	yes	61	20.3	20.3	100.0
	Total	300	100.0	100.0	

Table 1.7: Frequency table of presence of diabetes among patients

Table 1.7 represents that the majority of the patients of 79.7% do not have diabetes, 20.3% of patients have diabetes. The individuals who participated in the survey, majority do not have diabetes, and a very low number of patients were with diabetes.

Use of herbal

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	no	194	64.7	64.7	64.7
	yes	106	35.3	35.3	100.0
	Total	300	100.0	100.0	

Table 1.8: Frequency table of use of herbal medicine among the patients.

Table 1.8 represents that significant percentage of patients which is 64.7 % do not use herbal products. Whereas, around 35.3% of patients use herbal products. It is a good sign that the majority of the patients do not use any herbal medicine.

Thinning or Bleeding from affected area

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	both	161	53.7	53.7	53.7
	No	78	26.0	26.0	79.7
	only bleeding	20	6.7	6.7	86.3
	thinning only	41	13.7	13.7	100.0
	Total	300	100.0	100.0	

Table 1.9: Frequency table of patients have skin thinning or bleeding problem

The table 1.9 represents that the patients of 53.7% have the both problems (Thinning and Bleeding) which is the most common response. Whereas, 26.0% of patients reported “No” to have either condition. On the other hand 13.7% reported having “Thinning only” and only 6.7% of patients have “Only bleeding”. It indicates that in case of Psoriasis patients thinning and bleeding both are observed in majority.

Irritation in shower

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	no	198	66.0	66.0	66.0
	yes	102	34.0	34.0	100.0
	Total	300	100.0	100.0	

Table 1.10: Frequency table of patients have irritation during shower.

The table 1.10 represents that the majority of patients of 66% do not have irritation during shower. Whereas 34% of patients have irritation during showers. It indicates that water or soap during showers do not trigger Psoriasis patients.

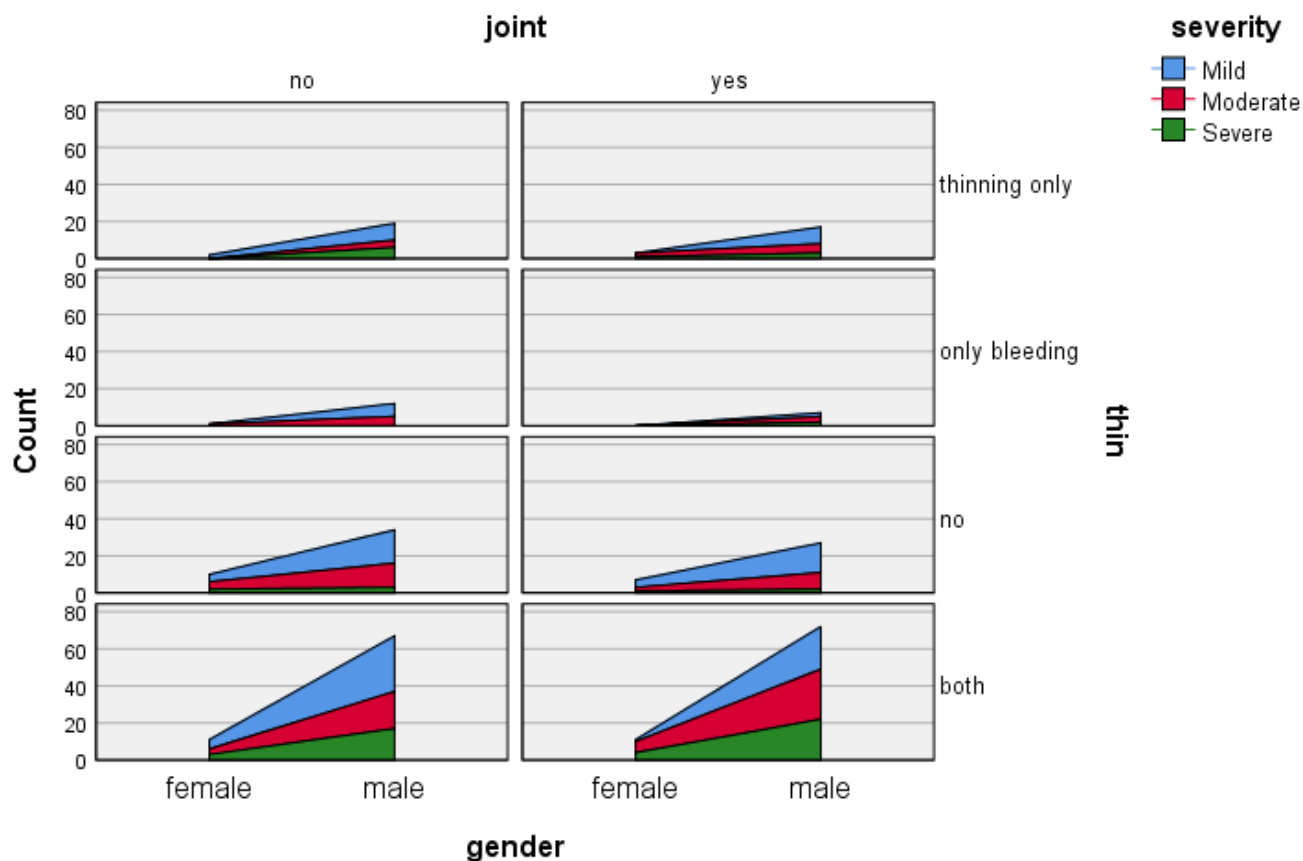


Figure 1.6: Distribution of thinning/ bleeding, joint pain and severity of the disease among male and female patients

Stacked bar chart showing the relationship of gender (Male, Female), thinning or bleeding (No, Both, Thinning only, only bleeding), joint pain (No, Yes), and severity (Severe, Moderate, Mild). The data is split by joint pain status, a large panel for "No" and a small one for "Yes." Within each panel, the data is split by four thinning or bleeding categories. Within each category, there are females' and males' stacked bars of severity levels.

Overall, men have a greater number in almost all of the joint pain and thinning or bleeding combinations than women. Most counts are in the no joint pain category, i.e., in the "Both" and "No" thinning or bleeding categories. In both of these, the severity is more or less evenly distributed among the mild, moderate, and severe cases, with mild having a slightly greater count.

Conversely, the numbers for patients with joint pain are significantly lower overall. Although the overall numbers are lower, there seems to be a slightly larger proportion of the more severe and moderate instances in the joint pain group, i.e., the "Both" thinning and bleeding. "Only bleeding" and "Thinning only" are uncommon with very low numbers for both males and females with or without joint pain.

Chittagong:

Age

		age			
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	<20	21	7.0	7.0	7.0
	>60	22	7.3	7.3	14.2
	20-30	70	23.2	23.2	37.4
	30-40	81	26.8	26.8	64.2
	40-50	64	21.2	21.2	85.4
	50-60	44	14.6	14.6	100.0
	Total	302	100.0	100.0	

Table 2.1: Frequency table of current age of individuals in the the sample

The table 2.1 presented indicates that the individual patients who participated in the survey were in the age range of <20, >60, 20-30, 30-40, 50-60 years, with a percentage of 7%, 7%, 23.2%, 26.8%, .3%, 20.9%, 14.6%, .3 respectively. According to the frequency distribution table, it can be observed that the most affected patients are in the age range of 30-40 years; and the least number of patients that are affected are less than 20 years.

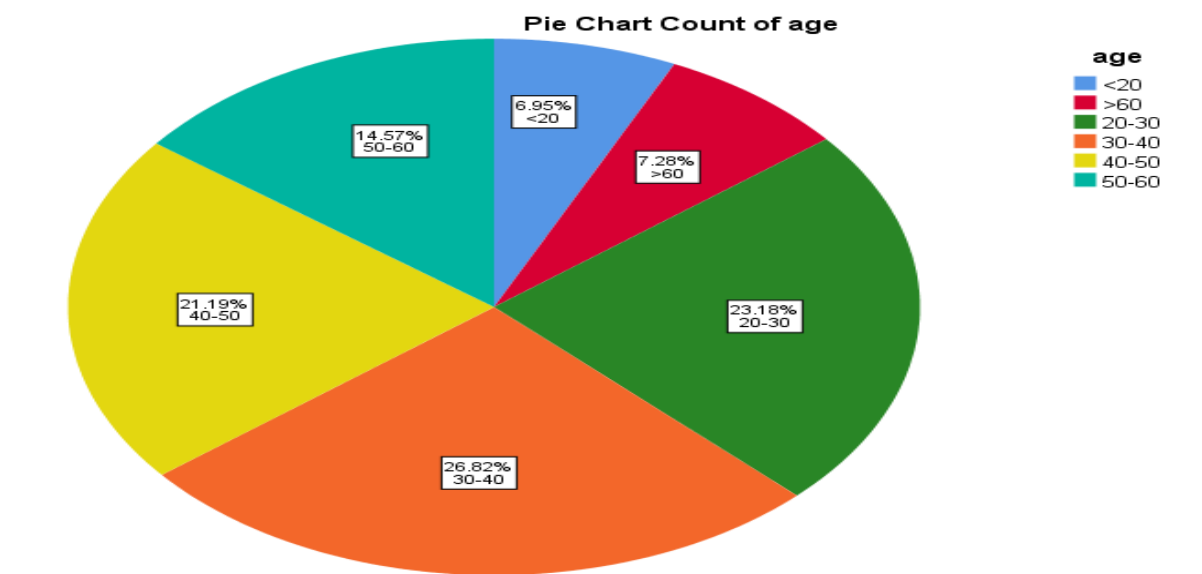


Figure 2.1 : Distribution of the percentage of of age range that were affected by psoriasis

The figure 2.1 of the pie chart indicates the percentage of age range that were affected by psoriasis. Here, <20 years were 6.95%, >60 years were 7.28%, 20-30 years were 23.18%, 30-40 years were 26.82%, 40-50 years were 21.19%, 50-60 years were 14.57%. Here, the chart indicates that the majority age range of the patients was 30-40 years; 26.82% and the minority of the patient age range was <20 years which was only 6.95%.

Gender

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Female	87	28.8	28.8	28.8
	Male	215	71.2	71.2	100.0
	Total	302	100.0	100.0	

Table 2.2: Frequency table of gender of individuals in the sample

The table 2.2 indicates that a significant percentage of individuals 71.2% are male, which indicates that the sample contains larger male patients than the female patients. Whereas there are about 28.8% of female patients. In contrast, the percentage of male patients is higher than the female patients.

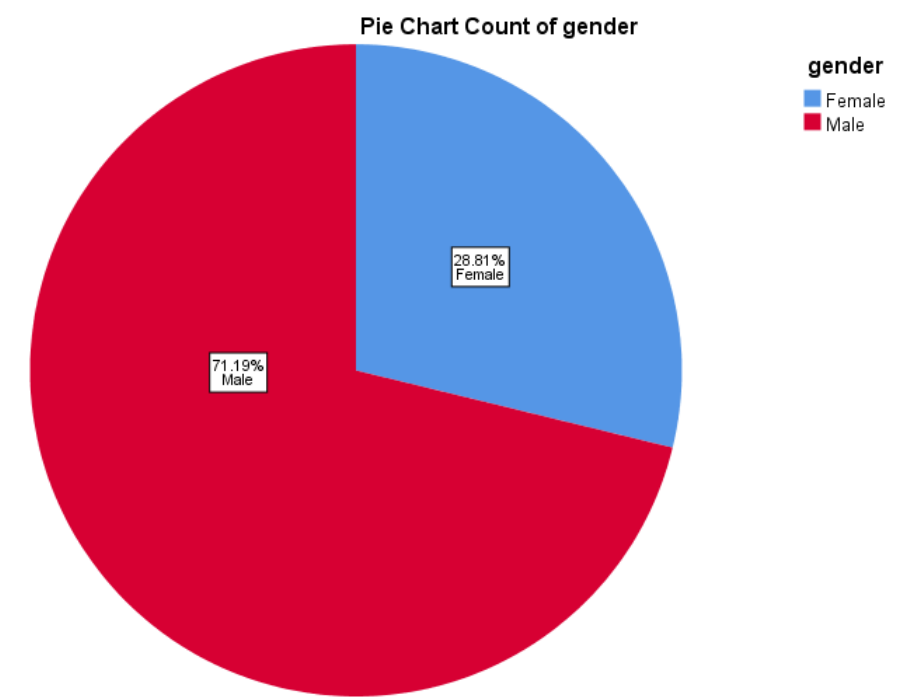


Figure 2.2 : Distribution of percentage of gender that were most affected by psoriasis

The figure 2.2 represents the gender most affected by Psoriasis. Among the patients there was 71.9% of patient was male and only 28.81% female.

Starting of symptoms

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	<1 year	54	17.9	17.9	17.9
	<6 months	1	.3	.3	18.2
	>2 years	170	56.3	56.3	74.5
	1-2 years	77	25.5	25.5	100.0
	Total	302	100.0	100.0	

Table 2.3: Frequency table of start of symptoms in the sample

The table 2.3 represents the frequency of duration of the patients suffering from the diseases. A substantial portion of the individual patients, 56.3% suffering for more than 2 years, and only .3% of the patients were suffering for less than 6 months. On the other hand, 17.9% of the patients suffered for less than one year and 25.5% of the patients suffered for 1-2 years. It indicates the patients are mostly suffering from Psoriasis for more than 2 years, since Psoriasis is a long-term or chronic disease.

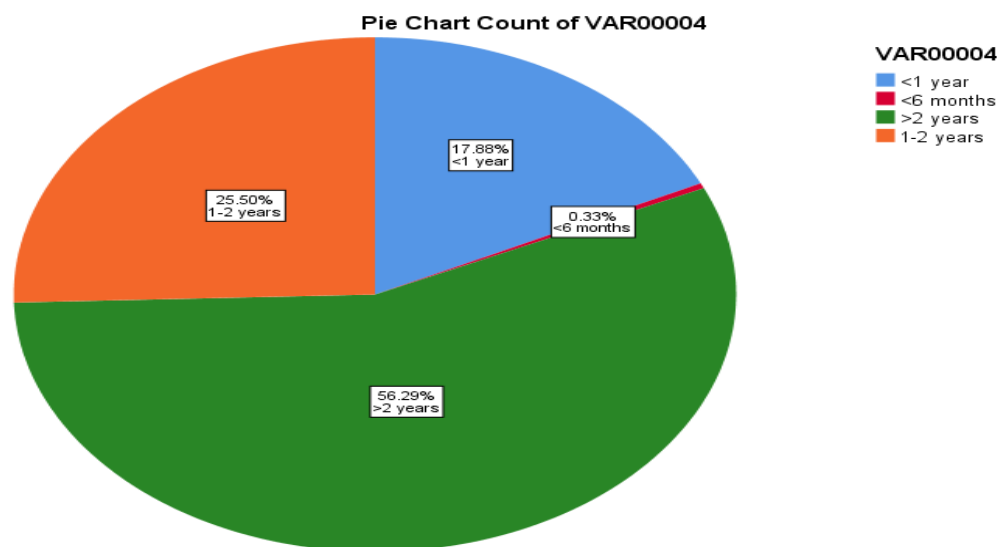


Figure 2.3 : Distribution of the percentage of duration of the patients suffering from psoriasis

The figure 2.3 represents the percentage of duration of the patients suffering from the diseases. A substantial portion of the individual patients, 56.29% suffering for more than 2 years, and only 0.33% of the patients were suffering for less than 6 months. On the other hand, 17.88% of the patients suffered for less than one year and 25.50% of the patients suffered for 1-2 years.

Severity of Condition

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Mild	121	40.1	40.1	40.1
	Moderate	117	38.7	38.7	78.8
	Severe	64	21.2	21.2	100.0
	Total	302	100.0	100.0	

Table 2.4: Frequency table of severity of a condition in the individuals

Table 2.4 represents a substantial portion of the individual patients, 40.1% have a mild condition, 38.7% of patients have moderate condition. However, there is a smaller group of patients, representing 21.2% of the sample, who have severe conditions.

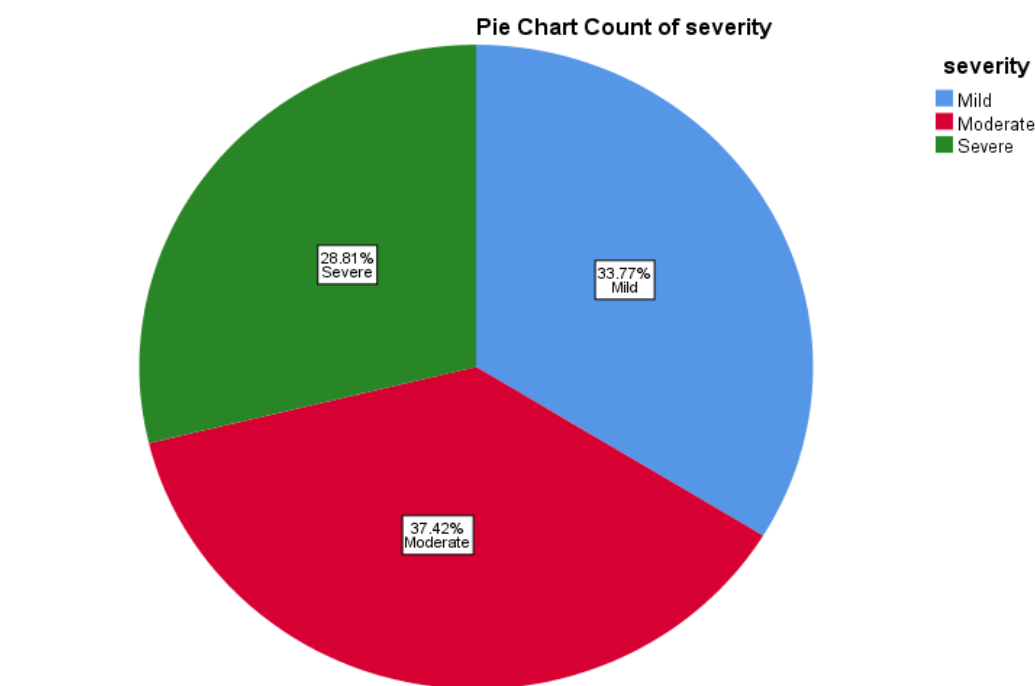


Figure 2.4 : Distribution of the percentage of severity of psoriasis in the patients

The figure 2.4 represents the percentage of severity of the patients. It indicates that the majority of the patients were in moderate condition, which was 37.42%. Again, 33.77% of the patients had mild conditions and 28.81% of the patients had severe conditions.

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate
1	.165 ^a	.027	.021	.753

a. Predictors: (Constant), stress, sun

In this multiple linear regression of the survey, The R value which is 0.165 represents the strength and direction of the linear relationship between the average stress and affected by sun. Since it's a positive value, there's a weak positive correlation between these factors.

An R-squared value of 0.027 indicates that only 2.7% of the observed variation in the change in stress is caused by the effect of the sun. The other 97.3% of the variation might be linked to other factors that are not accounted for in the model.

ANOVA^a

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	4.730	2	2.365	4.172	.016 ^b
	Residual	169.511	299	.567		
	Total	174.242	301			

a. Dependent Variable: severity

b. Predictors: (Constant), stress, sun

In the case of interactions of average stress and affected by sun, it is depicted that taking the factor or independent variable stress and being affected by sun where the p value is less than alpha which is the level of 0.05%. The null hypothesis can be rejected, which suggests that there is a statistically significant difference between the means of the groups being compared. In other words, at least one group differs markedly from zero. A moderate effect size is suggested by the F-value of 4.172. The higher the F-value in an ANOVA, the higher the variation between samples means relative to the variation within the samples. The higher the F-value, the lower the corresponding p-value.

Coefficients

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	1.598	.154		10.385	.000
	sun1	.126	.044	.165	2.889	.004
	stress1	-.011	.044	-.015	-.259	.796

a. Dependent Variable: severity1

Here the β_0 is the intercept, the value of Y when all independent variables (affected by sun and stress) are set to 0. The value is 1.598 in this case.

Moreover, the coefficient for affected by sun indicates that for every one unit increase in affected by sun, the change in severity increases by 0.126 units when stress remains constant. Also, in case of stress the unstandardized coefficient for stress is -0.011 and which means that for 1 unit change in stress we are going to notice -0.011 unit change in severity when affected by sun is constant.

Again, for those affected by sun the significance value or p value is 0.04 and as it is less than 0.05 so it does have statistical significance on the outcome variable. Furthermore, for stress the significance value or p value is 0.796 and as it is greater than 0.05 too so it does not have any statistical significance on the outcome variable (severity) either.

Use of herbal

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Don't know	7	2.3	2.3	2.3
	No	235	77.8	77.8	80.1
	Yes	60	19.9	19.9	100.0
	Total	302	100.0	100.0	

Table 2.5: Frequency table of use of herbal medicine among the patients.

Table 2.5 represents that significant percentage of patients which is 77.8 % do not use herbal products. Whereas, around 19.9% of patients use herbal products, on the other hand only 2.3% of patients are unsure. It is a good sign that the majority of the patients do not use any herbal medicine. And a few number of patients could have used subconsciously

Joint pain

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	No	178	58.9	58.9	58.9
	sometimes	1	.3	.3	59.3
	Yes	123	40.7	40.7	100.0
	Total	302	100.0	100.0	

Table 2.6: Frequency table of joint pain among patients

The table 2.6 indicates that 40.7 % of patients claim they have joint pain, which is the dominant response. However, 58.9% of patients do not have any joint pain and a very small fraction of patients 0.3% have pain sometimes. Here, the majority of the patients claim that they are not suffering from joint pain, and the patients with joint pain are comparatively low.

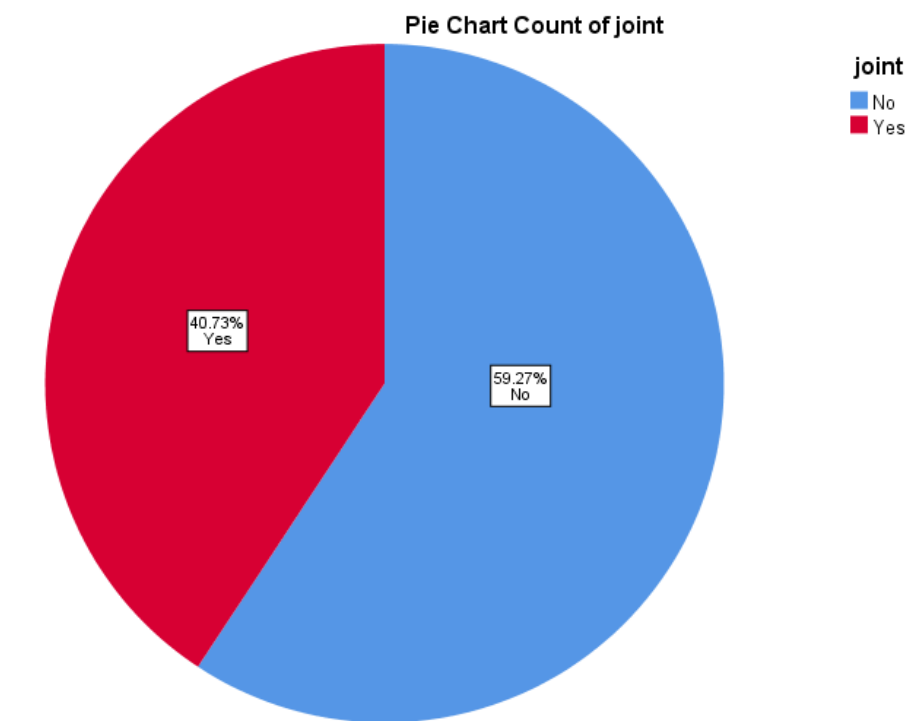


Figure 2.5 : Distribution of the percentage of presence of joint pain in psoriasis patients

The figure 2.5 represents whether the psoriasis patient had joint pain or not. Here, only 40.73% of the patients had joint pain and the other 59.27% of the patients had joint pain.

Diabetes

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Do not know	7	2.3	2.3	2.3
	No	235	77.8	77.8	80.1
	Yes	60	19.9	19.9	100.0
	Total	302	100.0	100.0	

Table 2.7: Frequency table of presence of diabetes among patients

The table 2.7 represents that the majority of the patients of 77.8% do not have diabetes, 19.9% of patients have diabetes whereas only 2.3 % of individuals are not sure. The individuals who participated in the survey, majority do not have diabetes , and a very low number of patients were with diabetes. And a very few patients are not sure.

Affected by Sun

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	No	157	52.0	52.0	52.0
	Not sure	1	.3	.3	52.3
	Yes	144	47.7	47.7	100.0
	Total	302	100.0	100.0	

Table 2.8: Frequency table of those affected by the sun

The table 2.8 represents the percentage of patients who get affected by exposure to sun. Most patients of 52.0% are not affected by sun, and 47.7% of patients are affected by exposure to sun, whereas only 0.3% of patients are not sure about it. Therefore it can be interpreted that psoriasis is not triggered by the sun in a high number of people.

Thinning/ Bleeding

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	No	55	18.2	18.2	18.2
	Only bleeding	12	4.0	4.0	22.2
	Thinning only	32	10.6	10.6	32.8
	Both	203	67.2	67.2	100.0
	Total	302	100.0	100.0	

Table 2.9: Frequency table of patients have skin thinning or bleeding problem

The table 2.9 represents that the patients of 67.2% have the both problems (Thinning and Bleeding) which is the most common response. Whereas, 18.2% of patients reported “No” to have either condition. On the other hand 10.6% reported having “Thinning only” and only 4.0% of patients have “Only bleeding”. It indicates that in case of Psoriasis patients thinning and bleeding both are observed in majority.

Irritation in shower

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	no	194	64.2	64.2	64.2
	yes	108	35.8	35.8	100.0
	Total	302	100.0	100.0	

Table 2.10: Frequency table of patients have irritation during shower.

The table 2.10 represents that the majority of patients of 64.2% do not have irritation during shower. Whereas 35.8% of patients have irritation during showers. It indicates that water or soap during showers do not trigger Psoriasis patients.

Symptoms duration

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	<1 year	64	21.3	21.3	21.3
	<6 months	12	4.0	4.0	25.3
	>2 years	154	51.3	51.3	76.7
	1-2 years	70	23.3	23.3	100.0
	Total	300	100.0	100.0	

Figure 2.11: Frequency table of duration of symptoms in patients

The table 2.11 represents that the patients of 51% are suffering more than 2 years, 23.3% of patients are suffering 1-2 years, the patients that are suffering less than 1 year are 21.3% and only 4% of patients are suffering for less than 6 months. It indicates the patients are mostly suffering from Psoriasis for more than 2 years, since Psoriasis is a long-term or chronic disease.

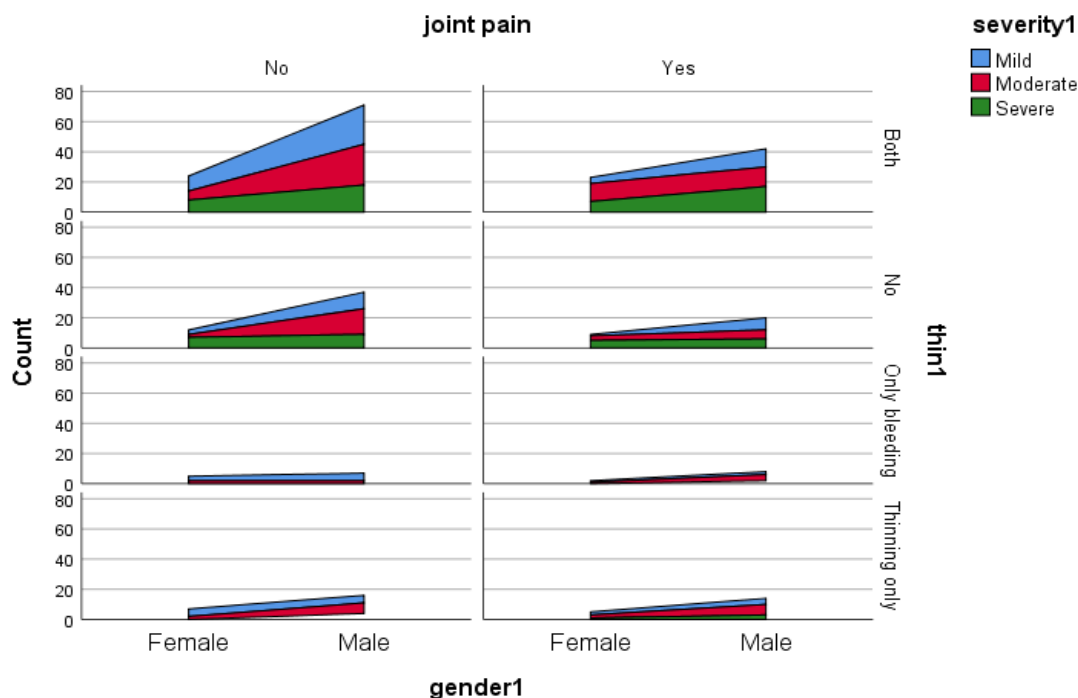


Figure 2.6: Distribution of thinning or bleeding and severity, joint pain of the disease among male and female patients

Stacked bar chart showing the relationship of gender (Male, Female), thinning or bleeding (No, Both, Thinning only, Only bleeding), joint pain (No, Yes), and severity (Severe, Moderate, Mild). The data is split by joint pain status, a large panel for "No" and a small one for "Yes." Within each panel, the data is split by four thinning or bleeding categories. Within each category, there are females' and males' stacked bars of severity levels.

Overall, men have a greater number in almost all of the joint pain and thinning or bleeding combinations than women. Most counts are in the no joint pain category, i.e., in the "Both" and "No" thinning or bleeding categories. In both of these, the severity is more or less evenly distributed among the mild, moderate, and severe cases, with mild having a slightly greater count.

Conversely, the numbers for patients with joint pain are significantly lower overall. Although the overall numbers are lower, there seems to be a slightly larger proportion of the more severe and moderate instances in the joint pain group, i.e., the "Both" thinning and bleeding. "Only bleeding" and "Thinning only" are uncommon with very low numbers for both males and females with or without joint pain.

Cross table analysis

		Severity duration of condition and severity			Total
		Mild	Moderate	Severe	
Symptoms start	<1 year	33	20	11	64
	<6 months	6	4	2	12
	>2 years	55	57	42	154
	1-2 years	36	23	11	70
Total		130	104	66	300

Table 3.1: Cross table analysis between duration of condition and severity

According to the crosstabulation table 3.1, the data shows the relationship between duration of condition and severity. The majority of the patients have long-standing conditions, with 154 out of 300 individuals having conditions that started 2 years ago which is the highest number of individuals of all severity categories.

For conditions that have been in existence for a shorter period of time (<1 year), a greater percentage seems to be mild or moderate severity. Of the 64 individuals who have been diagnosed with a condition that originated less than a year ago, 33 are mild and 20 are moderate, while only 11 are considered severe. In the very recent group (<6 months) most are mild or moderate.

In contrast, for those individuals with the condition for a longer period (>2 years), severity is lesser. In comparison to this group, the counts were more proportionate across mild (55), moderate (57), and severe (42) were more proportionate in this group than in the others. Whereas mild or moderate cases were still seen most frequently, the proportion of severely affected cases was noticeably greater than the more recent categories suggesting that severity may get worse with longer duration. The 1-2 years group seems to follow a similar pattern as there was a relatively low count of severe vs. mild and moderate cases.

diabetes * severity Crosstabulation

Count		Severity			Total
		Mild	Moderate	Severe	
diabetes	no	107	81	51	239
	yes	23	23	15	61
Total		130	104	66	300

Table 3.2: Cross table analysis between duration of diabetes and severity

According to the crosstabulation table 3.2, the data shows the relationship between diabetes and severity. The majority of the patients do not have diabetes of individuals 239 out of 300 of all severity categories, where individuals of 107 have mild severity, 81 have moderate severity, whereas only 51 patients have severe conditions.

For conditions that have been observed that only 61 individuals have diabetes of all severity categories, where 23 of individuals have mild and moderate conditions, and 15 of individuals have severe conditions, are diabetic patients. In contrast, the affected cases were noticeably greater for non-diabetic groups than the diabetic patients.

Joint pain * severity Crosstabulation

Count

		Severity			Total
		Mild	Moderate	Severe	
Joint pain	no	75	50	31	156
	yes	55	54	35	144
Total		130	104	66	300

Table 3.3: Cross table analysis between joint pain and severity

According to the crosstabulation table 3.3, the data shows the relationship between joint pain and severity of conditions, where patients 156 out of 300 do not have joints pain, the most severity condition is mild which is 75, followed by moderate conditions is 50 and only 31 patients do not have joint pain are the condition of severe. In comparison with the, the distribution of severity for the 144 individuals with joint pain is more equal. The numbers of mild (55), moderate (54), and severe (35) are more evenly distributed than for the individuals without joint pain.

This comparison indicates that those without joint pain tend to be mild. The individuals with evidence of joint pain show a relatively higher chance of the condition being moderate or severe than mild.

stress * joint pain Crosstabulation

Count		Joint pain		Total
		no	yes	
stress	No	85	61	146
	Not sure	14	6	20
	Yes	57	77	134
Total		156	144	300

Table 3.4: Cross table analysis between stress and joint pain

The table 3.4 analyzes the relationship between stress and joint pain. The patients of 146 out of 300 reported having no stress, 134 reported having stress and only 20 of individuals were unsure. The patients who reported having stress are the higher number who reported having joint pain that is 77 in number compared to those who did not have joint pain is 57 in number. As a result the correlation is positive. In contrast, among the individuals who reported no stress as well as no joint pain is 85 in number which represents the majority of numbers. Only 61 of individuals reported having joint pain but no stress. Therefore, the data represents that the patients having joint pain are more likely to experience stress compared to those who do not experience stress.

Severity * Effect of sun Crosstabulation

Count		Effect of sun		Total
		no	yes	
severity	Mild	75	55	130
	Moderate	54	50	104
	Severe	25	41	66
Total		154	146	300

Table 3.5: Cross table analysis between severity and effect of sun

The crosstabulation table 3.5 represents the relationship between severity and affected by sun. Out of 300 146 reported being affected by sun exposure while 154 are not affected. When outcomes were categorized by severity, patients with sun exposure reported a notable excess of severe conditions, that is 41 in number. On the other hand, mild cases were higher without sun exposure, The overall pattern accords with the hypothesis that sun exposure is related to greater severity, as the number affected by sun exposure is increased according to the severity of severe condition.

Case Processing Summary

		Cases					
		Valid		Missing		Total	
		N	Percent	N	Percent	N	Percent
start	*	302	100.0%	0	0.0%	302	100.0%
severity							

Symptoms start * severity Crosstabulation

Count

		severity			Total
		Mild	Moderate	Severe	
start	<1 year	22	18	14	54
	<6 months	0	0	1	1
	>2 years	45	67	58	170
	1-2 years	35	28	14	77
Total		102	113	87	302

Table 3.6: Cross table analysis between duration of condition and severity

According to the crosstabulation table 3.6, the data shows the relationship between duration of condition and severity. The majority of the patients have long-standing conditions, with 170 out of 302 individuals having conditions that started 2 years ago which is the highest number of individuals of all severity categories.

For conditions that have been in existence for a shorter period of time (<1 year), a greater percentage seems to be mild or moderate severity. Of the 54 individuals who have been diagnosed with a condition that originated less than a year ago, 22 are mild and 18 are moderate, while only 14 are considered severe. In the very recent group (<6 months) only 1 was diagnosed with a severe condition.

In contrast, for those individuals with the condition for a longer period (>2 years), mild is lesser. In comparison to this group, the counts were more proportionate across mild (45), moderate (67), and severe (58) were more proportionate in this group than in the others.

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
severity * thinning bleeding	302	100.0%	0	0.0%	302	100.0%

severity * thinning/ bleeding Crosstabulation

Count

		Thinning or bleeding				Total
		Both	No	Only bleeding	Thinning only	
severity	Mild	52	23	11	16	102
	Moderate	58	28	9	18	113
	Severe	50	27	2	8	87
Total		160	78	22	42	302

Table 3.7: Cross table analysis between severity and thinning and bleeding

According to table 3.7, the data shows a relationship between severity and thinning and bleeding. The most common severity level in the dataset is Moderate, with 113 cases, followed by Mild (102 cases) and Severe (87 cases). The most common thinning and bleeding "category" for the whole dataset is "Both," as it includes 160 of the 302 cases, suggesting that this would mean that when this occurs there is often both thinning and bleeding.

While analyzing the data by severity level, there is a consistent pattern. For all three categories of severity are Mild, Moderate, and Severe the majority of cases all fall into the category of "Both." This was true for 52 out of 102 mild cases, 58 out of 113 moderate cases, and 50 out of 87 severe cases. This would seem to indicate that there is a strong potential relationship between thinning and bleeding when it occurs, and that it generally does not matter how severe it is.

The "No," or no thinning and no bleeding category, accounted for 78 cases combined. "Thinning only" accounted for 42 cases, and "Only bleeding" altogether accounted for 22 cases total. Specifically within the Severe severity category there were only 2 cases that contained "Only bleeding," which would demonstrate that even the bleeding characteristic is rarely only bleeding in the most severe manifestations of the private identity.

Case Processing Summary

		Cases				
		Valid		Missing		Total
		N	Percent	N	Percent	N
severity	*	302	100.0%	0	0.0%	302
diabetes						

severity * diabetes Crosstabulation

		Diabetes		Total
Count		No	Yes	
severity	Mild	80	22	102
	Moderate	91	22	113
	Severe	47	40	87
Total		218	84	302

Table 3.8: Cross table analysis between diabetes and severity

According to the crosstabulation table 3.8, the data shows the relationship between diabetes and severity. The majority of the patients do not have diabetes of individuals 218 out of 302 of all severity categories, where individuals of 80 have mild severity, 91 have moderate severity, whereas only 47 patients have severe conditions.

For conditions that have been observed that only 84 individuals have diabetes of all severity categories, where 22 of individuals have mild and moderate conditions, and 40 of individuals have severe conditions, are diabetic patients. In contrast, the affected cases were noticeably greater for non-diabetic groups than the diabetic patients.

severity * age Crosstabulation

Count		age						Total
		<20	>60	20-30	30-40	40-50	50-60	
severity	Mild	12	8	28	27	14	13	102
	Moderate	6	7	33	33	24	10	113
	Severe	3	7	9	21	26	21	87
Total		21	22	70	81	64	44	302

Table 3.9: Cross table analysis between severity and age

The crosstabulation table 3.9, represents the relationship between age group and severity condition. From a sample of 302 individuals, most patients had moderate severity (113) and the fewest had severe cases (87), with mild cases (102). In terms of age, the 30-40 age group was the largest (81), and showed a fairly even distribution of mild (21), moderate (33), and a high incidence of the severe cases (21). The 20-30 group was the next largest group, (70) with the mild (28) and the moderate (33) patients again showing that younger adults had less severe presentations.

Severe cases tended to be more common in the 40-50 age group (26) and then in the 50-60 age group (21), and the difference in severity between age groups indicates that severity does seem to progress with age. The youngest (< 20) group showed mostly mild cases 12 out of 21, and the oldest (> 60) group, although the smallest (22), showed a higher proportion of severe scoring (7).

Overall, the case severity by age group pattern suggests young individuals tend to have mild or moderate severity cases, whereas the oldest individuals (> 60) are more likely to have severe conditions.

age * start Crosstabulation

		Total				
		<1 year	<6 months	>2 years	1-2 years	
age	<20	7	0	7	7	21
	>60	0	0	18	4	22
	20-30	23	0	28	19	70
	30-40	12	1	42	26	81
	40-50	9	0	41	14	64
	50-60	3	0	34	7	44
Total		54	1	169	77	302

Table 3.10 : Cross table analysis between age and start

The crosstabulation table 3.10 reflects that there is a pattern between age groups and duration of the diseases affected; out of 302, there are large in number who is suffering for more than 2 years (169), than who is suffering for less than a year (54) or within less than 6 months (1).

When considered within age categories individuals between 30-40 years represent the bulk (81 individuals), within which a significant number (42) is suffering for more than 2 years. In relation to individuals under 20 years they appeared to be more evenly distributed across durations. Whereas older individuals (over 60 years) followed the pattern of majority suffering for more than two years (18 out of 22 individuals). The overall proportion indicates that the duration of the symptoms are longer in the older age groups and especially for individuals aged between 30-60 years. However, the early start (under 1 year) is less frequent across groups.

Severity * effect of sun Crosstabulation

Count		Sun			Total
		No	Not sure	Yes	
severity	Mild	71	1	49	121
	Moderate	63	0	54	117
	Severe	23	0	41	64
Total		157	1	144	302

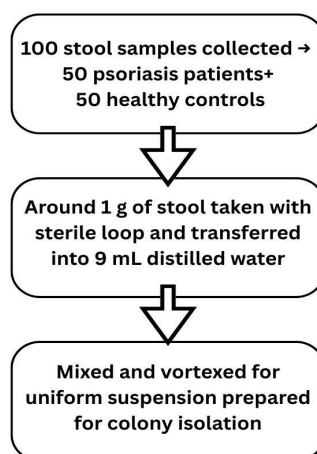
Table 3.11: Cross table analysis between severity and sun effect

The crossstabulation table 3.11 represents the relationship between severity and affected by sun. Out of 302, 144 reported being affected by sun exposure while 157 are not affected. When outcomes were categorized by severity, patients with sun exposure reported a notable excess of moderate conditions, that is 54 in number. On the other hand, mild cases were higher without sun exposure, The overall pattern accords with the hypothesis that sun exposure is related to greater severity, as the number affected by sun exposure is increased according to the severity of severe condition.

12.6 Research Methodology:

12.6.1 Sample Collection

This study involved the collection of 100 stool samples, consisting of 50 from clinically diagnosed psoriasis patients at Kurmitola General Hospital and 50 from healthy individuals serving as controls. For each sample, around 1 gram of stool was collected using a sterile loop and transferred into a test tube containing 9 mL of distilled water. The samples were thoroughly mixed and vortexed to obtain a uniform suspension suitable for further colony isolation.



12.6.2 Blood Collection and Serum Processing:

Approximately 3 to 4 mL of venous blood was drawn from each participant using sterile syringes and collected into plain red-top vacutainer tubes (CURE™, Bangladesh). Samples were immediately placed in an ice box to maintain protein stability. In the laboratory, the blood was allowed to clot at room temperature. For samples where clot separation was not complete, the blood was transferred to sterile microcentrifuge tubes and centrifuged at 3000 rpm for 10 minutes. The clear serum layer was then pipetted into fresh sterile tubes and stored at -80°C for cytokine analysis.

12.6.3 Bacterial Isolation and Culture:

A loopful of the diluted stool suspension was streaked onto Skirrow's selective medium, which contains antibiotics such as vancomycin, polymyxin B, and trimethoprim to suppress the growth of non-target bacteria under sterile conditions. Colonies with morphological features characteristic of *H. pylori* were identified and selected for sub-culturing and preservation. All culture plates were incubated at 37°C for 24 hours.

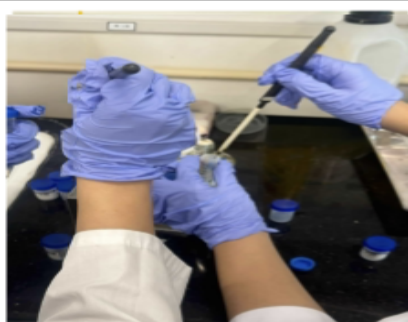


Figure 9: A loopful stool sample were diluted in distilled water

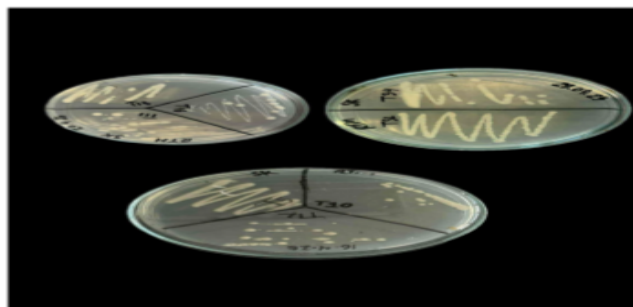


Figure 10: Cultured plates after incubation for 24 hours of loopful of the diluted stool suspension on Skirrow's selective media

Figure 9 shows a loopful of stool was taken as was being diluted inside a test tube filled with distilled water.

Figure 10, shows cultured plates after 24 hours incubation of the diluted stool samples. It illustrates bacterial growth which indicates presence of target microorganisms which was needed for selective isolation and further analysis.

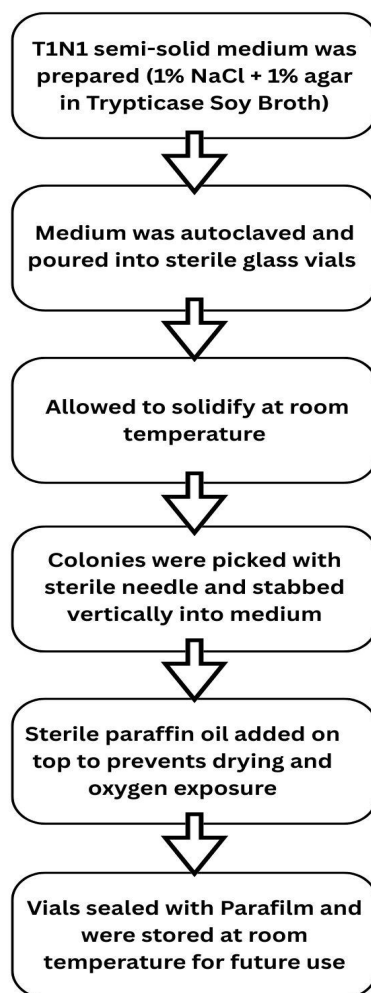
12.6.4 Preparation and Storage of Stab Cultures:

T1N1 medium was used with chemical composition of solid medium, consisting of 1% NaCl and 1% agar in a Trypticase Soy Broth base, autoclaved and poured into sterile glass vials. After solidification at room temperature, selected colonies were picked with a sterile needle and stabbed vertically into the medium. A layer of sterile liquid paraffin oil was added to prevent drying and oxygen exposure. The vials were sealed with Parafilm and stored at room temperature for future use.



Figure 11: Stored stock culture in T1N1 media

Figure 11 shows the stored stock culture in T1N1 media, which indicates the preservation of bacterial strains under stab storage conditions. This method maintains viability and genetic stability of the culture that can be used for long-term in experiments.



12.6.5 Determination of Colony-Forming Units (CFU):

1. To quantify viable bacterial cells, *H. pylori* was re-isolated from the T1N1 stab cultures. A sterile needle was used to inoculate Skirrow's agar, followed by incubation at 37°C for 24 hours.
2. After incubation, single colonies were taken and suspended with 1ml of saline water as diluent in the first mct tube marked 10^0 .
3. The mixture was mixed thoroughly by a vortex mixer.
4. Serial dilution of 10^1 was done by using a sterile micropipette to transfer 10 μ L of mixture from 10^0 and suspending it with 90 μ L of saline water.
5. By maintaining this same procedure, serial dilutions were prepared up to 10^8 .
6. After that, 100 μ L of sample diluted saline water were taken and plating was done in Nutrient Agar by spread plating method.

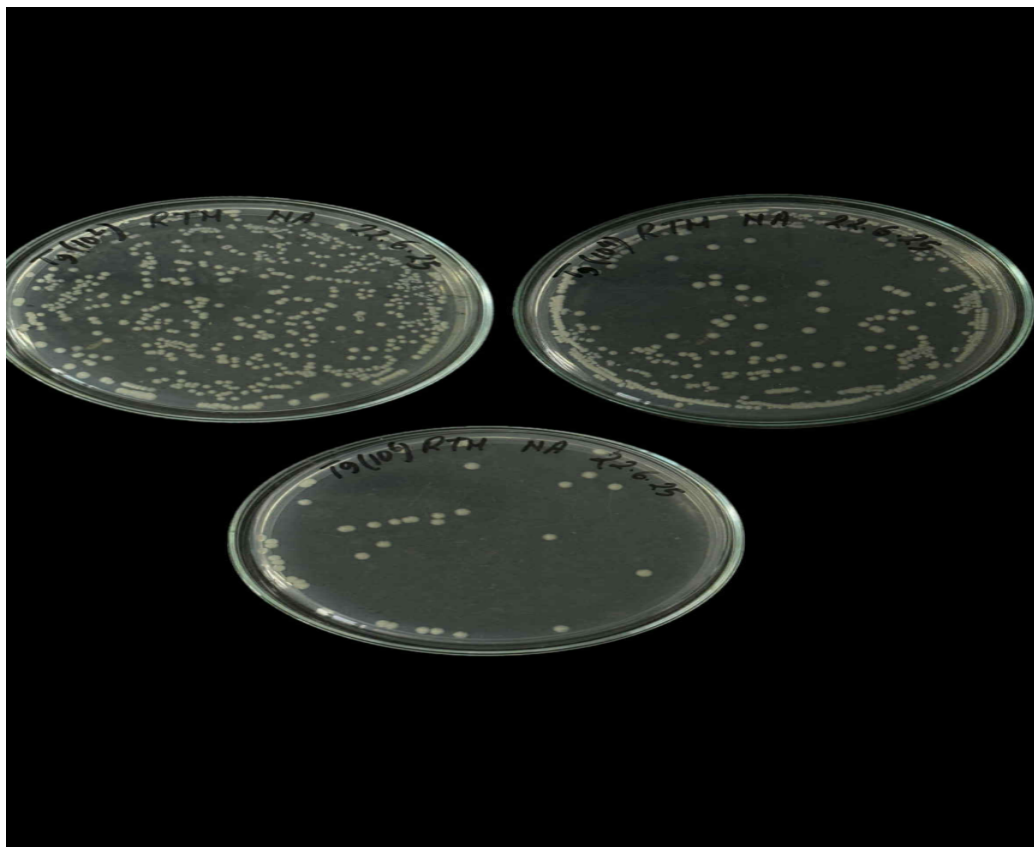


Figure 12: H. pylori CFU plate count in NA media

Figure 12 shows the colony-forming unit (CFU) count of H. pylori on Nutrient Agar (NA) media, which shows the bacterial population.

12.6.6 Quantification of IL-6 and IL-17A Using ELISA

Serum levels of IL-6 and IL-17A were measured using PicoKine® Human ELISA Kits (Boster Biological Technology, Cat. No. EK0410 for IL-6 and EK0430 for IL-17A). All steps were performed in accordance with the manufacturer's protocols.

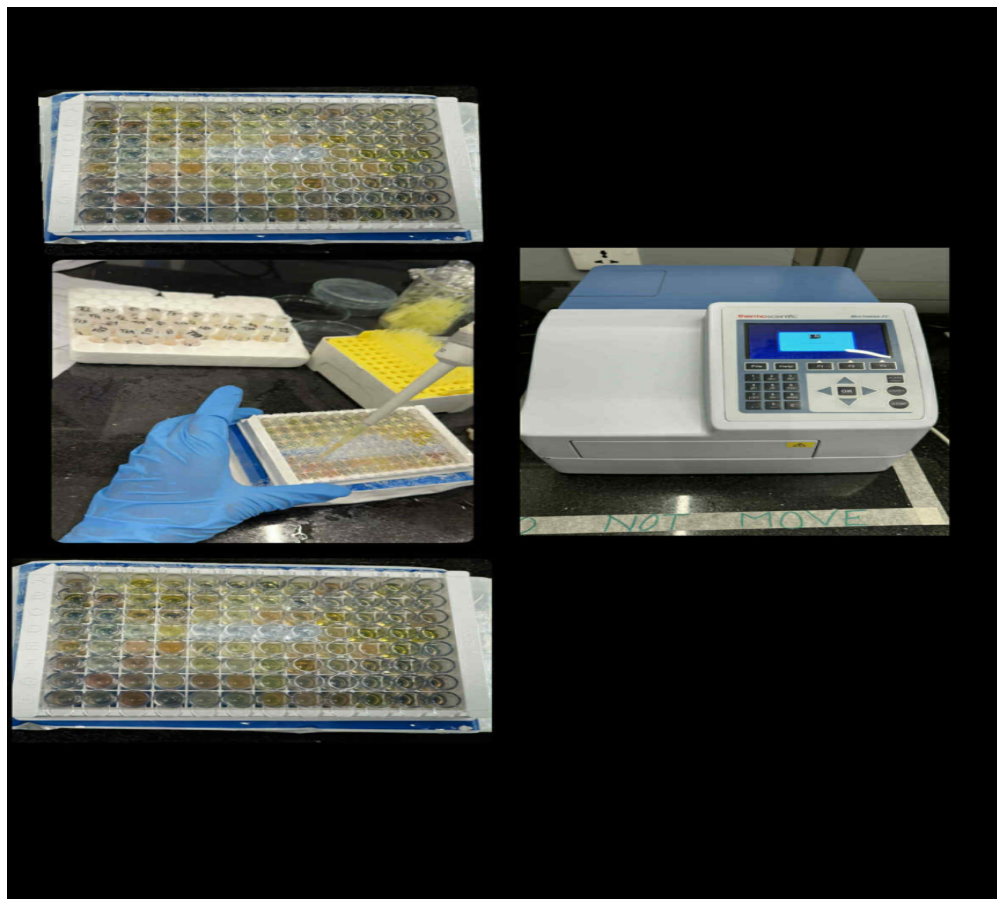


Figure 13: Procedure of ELISA

Figure 13 shows the ELISA procedure, describing the stepwise process of antigen-antibody binding, washing, and detection to quantify specific proteins or antibodies in a sample.

IL-6:

1. All reagents were prepared and working standards were set as per kit instructions.
2. Excess microplate strips were removed from the plate frame and were sealed and stored in the original packaging.
3. 100 µl of the standard, samples, or control were added into each well. 100 µl of the Sample Diluent was added into the zero well. At least two replicates of each standard, sample, or control were used.
4. The plate was covered with the provided plate sealer and was incubated for 90 min. at 37 °C.
5. The cover was removed and the liquid in the wells was discarded into an appropriate waste receptacle. The plate was inverted on the benchtop onto a paper towel and the plate was tapped gently to blot any remaining liquid. The wells were not allowed to completely dry at any time.
6. 100 µl of the prepared 1x Biotinylated Anti-Human IL6 antibody was added to each well.
7. The plate was covered with a sealer and incubated for 60 minutes at 37°C.
8. The plate was washed 3 times with the 1x wash buffer:
 - a. The liquid was discarded in the wells into an appropriate waste receptacle. Then, the plate on the benchtop was inverted onto a paper towel and was tapped to gently blot any remaining liquid. The wells were not allowed to completely dry at any time.
 - b. 300 µl of the 1x wash buffer was added to each assay well and was incubated for 60 seconds
 - c. a-b steps were repeated two additional times.
 - d. The remaining wash buffer was discarded in the wells into an appropriate waste receptacle. Then, the plate was inverted on the benchtop onto a paper towel and the plate was tapped gently to remove any remaining liquid.
9. 100µl of the prepared 1x Avidin-Biotin-Peroxidase Complex was added into each well. The plate was covered with the plate sealer provided and was incubated for 30 minutes at 37°C.
10. The plate was washed five times with the 1x wash buffer:
 - a. The liquid was discarded in the wells into an appropriate waste receptacle. Then, the plate was inverted on the benchtop onto a paper towel and the plate was tapped to gently blot any remaining liquid. The wells were not allowed to completely dry at any time.
 - b. 300 µl of the 1x wash buffer was added to each assay well and was incubated for 60 seconds between each wash.
 - c. steps a-b were repeated four additional times.
 - d. The wash buffer was discarded in the wells into an appropriate waste receptacle and was inverted on the benchtop onto a paper towel and tapped the plate to gently blot any remaining liquid.

11. 90 µl of Color Developing Reagent was added to each well. The plate was covered with the plate sealer provided and incubated in the dark for 15-25 minutes at 37°C). (The optimal incubation time must be empirically determined. A guideline to look for is blue shading the top four standard wells, while the remaining standards remain clear.)
12. 100 µl of Stop Solution was added to each well and the color was immediately changed to yellow.
13. 30 minutes within when the reaction was stopped, the O.D. absorbance was measured at 450 nm using a microplate reader.

IL-17

1. All reagents were prepared and working standards were set as per kit instructions.
2. Excess microplate strips were removed from the plate frame and were sealed and stored in the original packaging.
3. 100 µl of the standard, samples, or control were added into each well. 100 µl of the Sample Diluent was added into the zero well. At least two replicates of each standard, sample, or control were used.
4. The plate was covered with the provided plate sealer and was incubated for 90 min. at 37 °C.
5. The cover was removed, and the liquid in the wells was discarded into an appropriate waste receptacle. The plate was inverted on the benchtop onto a paper towel and the plate was tapped gently to remove any remaining liquid. The wells were not allowed to completely dry at any time.
6. 100 µl of the prepared 1x Biotinylated Anti-Human IL17A antibody was added to each well.
7. The plate was covered with a sealer and was incubated for 60 minutes at 37°C.
8. The plate was washed 3 times with the 1x wash buffer:
 - a. The liquid was discarded in the wells into an appropriate waste receptacle. Then, the plate on the benchtop was inverted onto a paper towel and was tapped gently to remove any remaining liquid. The wells were not allowed to completely dry at any time.
 - b. 300 µl of the 1x wash buffer was added to each assay well and was incubated for 60 seconds
 - c. a-b steps were repeated two additional times.
 - d. The remaining wash buffer was discarded in the wells into an appropriate waste receptacle. Then, the plate was inverted on the benchtop onto a paper towel and the plate was tapped gently to remove any remaining liquid.
9. 100 µl of the prepared 1x Avidin-Biotin-Peroxidase Complex was added into each well. The plate was covered with the plate sealer provided and was incubated for 30 minutes at 37°C.
10. The plate was washed five times with the 1x wash buffer:

- a. The liquid was discarded in the wells into an appropriate waste receptacle. Then, the plate was inverted on the benchtop onto a paper towel and the plate was tapped gently to remove any remaining liquid. The wells were not allowed to completely dry at any time.
 - b. 300 μ l of the 1x wash buffer was added to each assay well and was incubated for 60 seconds between each wash.
 - c. steps a-b were repeated four additional times.
 - d. The wash buffer was discarded in the wells into an appropriate waste receptacle and was inverted on the benchtop onto a paper towel and tapped gently to remove any remaining liquid.
11. 90 μ l of Color Developing Reagent was added to each well. The plate was covered with the plate sealer that was provided and incubated in the dark for 15-25 minutes at RT at 37°C. (The optimal incubation time must be empirically determined. A guideline to look for is blue shading the top four standard wells, while the remaining standards remain clear.)
 12. 100 μ l of Stop Solution was added to each well and the color was immediately changed to yellow.
 13. 30 minutes within when the reaction was stopped, the O.D. absorbance was measured at 450 nm using a microplate reader.

12.6.7 DNA Extraction:

Isolated colonies from each sample are picked and inoculated in the TE buffer and using a modified heat shock method, DNA from each isolate is extracted.

Modified heat shock method:

- Picked a single colony from the Nutrient Agar plate.
- Inoculated and suspended in the TE buffer.
- The tubes were incubated in a Dry heat bath at 95°C for 20 minutes.
- It was then centrifuged at 10,000 rpm for 10 minutes
- The supernatant, containing DNA, was collected and stored at -20°C for further molecular analysis.

12.6.8 Polymerase Chain Reaction (PCR)

Master mix, nuclease-free water, forward and reverse primers were mixed in a microcentrifuge tube depending on the total sample size.

Component	Final concentration (per sample)
Nuclease free water	2.5 μL
Master mix	6.5 μL
Forward primer	1 μL
Reverse primer	1 μL
Template DNA	2 μL
Total volume	13 μL

Table: Amount of products for PCR mix for a sample

1. The mixture was then mixed properly using a gentle vortex and centrifuged for 10 seconds for 5000 rpm.
2. Then, the mixture was transferred into PCR tubes using sterilized micropipette, making a total of 11 μL reaction volume in each PCR tube.
3. 2 μL of template DNA was added to each tube (total concentration 13 μL) and mixed thoroughly by repipetting.
4. The PCR tubes were given a short spin and then were placed into the PCR machine.
5. The program of the thermocycler was set as follows:
 6. Denaturation at 95°C for 3 minutes.
 7. Annealing at 55°C for 30 seconds.
 8. Extension at 72°C for 5 minutes.
 9. Steps had been repeated for 35 cycles.
 10. Holding temperature at 4°C indefinitely.
 11. After taking the pcr tubes out, it was stored in a -20 fridge to avoid DNA denaturation.

12.6.9 Agarose Gel Electrophoresis

1. 1% agarose gel was prepared by dissolving 1 g of agarose powder in 90 ml of distilled water and 10 ml of 10X TBE buffer (total 100 ml).
2. The solution was heated until the agarose was completely dissolved, and was kept until it was cooled slightly, and then ethidium bromide (4 μ l) was added.
3. The molten agarose was poured into a casting tray with a comb that was inserted to form wells and was allowed to solidify.
4. The comb was removed after the gel was solidified and the gel tray had been placed in the mupid, which had been filled with 1X TBE running buffer until the gel was submerged.
5. A PCR sample of 7 μ l of each that was previously stored was loaded into the wells of the gel.
6. A DNA ladder (molecular weight marker) was also loaded to compare band sizes at the first well.
7. The electrophoresis was done at 100 V for 45 minutes.
8. The gel was visualized under UV transillumination after completing the electrophoresis, and the DNA bands were observed accordingly.

13. Results:

13.1 CFU:

Test	10 ⁻²	10 ⁻⁴	10 ⁻⁶	10 ⁻⁸	Control	10 ⁻²	10 ⁻⁴	10 ⁻⁶	10 ⁻⁸
1	TNTC	TNTC	7.60×10 ⁸	6.10×10 ⁸	51	9.37×10 ₈	7.88×10 ⁸	8.22×10 ₈	7.90×10 ⁸
2	TNTC	2.33×10 ⁷	5.20×10 ⁸	4.80×10 ⁸	52	7.29×10 ₈	4.90×10 ⁶	6.77×10 ₈	TFTC
3	TNTC	TNTC	TNTC	5.20×10 ⁸	53	8.58×10 ₈	7.60×10 ⁸	3.87×10 ₈	3.80×10 ⁸
4	TNTC	TNTC	TNTC	5.89×10 ⁸	54	TNTC	TNTC	1.11×10 ₉	1.08×10 ⁹
5	TNTC	TNTC	2.97×10 ⁹	4.35×10 ⁸	55	TNTC	TNTC	9.32×10 ₈	9.00×10 ⁸
6	TNTC	TNTC	7.88×10 ⁸	7.20×10 ⁸	56	TNTC	TNTC	2.99×10 ₉	2.80×10 ⁹
7	TNTC	TNTC	7.60×10 ⁸	6.00×10 ⁸	57	8.79×10 ₈	TNTC	4.12×10 ₈	4.00×10 ⁸
8	TNTC	TNTC	6.20×10 ⁸	5.46×10 ⁸	58	6.30×10 ₈	1.15×10 ⁷	7.55×10 ₈	7.20×10 ⁸
9	TNTC	TNTC	4.40×10 ⁸	4.20×10 ⁸	59	9.41×10 ₈	TNTC	8.77×10 ₈	8.40×10 ⁸
10	TNTC	TNTC	TNTC	7.67×10 ⁸	60	6.17×10 ₈	TNTC	5.66×10 ₈	5.40×10 ⁸
11	TNTC	TNTC	2.42×10 ⁹	2.30×10 ⁹	61	TNTC	TNTC	9.88×10 ₈	7.40×10 ⁸
12	TNTC	TNTC	1.07×10 ⁹	8.60×10 ⁸	62	TNTC	2.90×10 ⁷	8.44×10 ₈	TFTC
13	TNTC	TNTC	2.44×10 ⁹	2.10×10 ⁹	63	TNTC	TNTC	1.23×10 ₉	1.15×10 ⁹

14	TNTC	TFTC	TFTC	TFTC	64	$5.02 \times 10_8$	TNTC	$4.55 \times 10_8$	4.30×10^8
15	2.97×10^3	1.66×10^7	6.30×10^8	5.80×10^8	65	$9.07 \times 10_8$	TNTC	$8.40 \times 10_8$	8.00×10^8
16	TNTC	TNTC	3.10×10^8	2.95×10^8	66	$1.17 \times 10_8$	1.80×10^7	$9.55 \times 10_8$	9.10×10^8
17	TNTC	1.06×10^7	7.88×10^8	6.10×10^8	67	TNTC	TNTC	$7.77 \times 10_8$	5.40×10^8
18	TNTC	TNTC	1.01×10^9	9.90×10^8	68	$9.38 \times 10_8$	TNTC	$6.89 \times 10_8$	6.50×10^8
19	TNTC	TNTC	3.35×10^9	3.10×10^9	69	$4.64 \times 10_8$	7.88×10^8	$2.99 \times 10_9$	2.80×10^9
20	TNTC	TNTC	TNTC	4.42×10^8	70	$5.72 \times 10_8$	TNTC	$5.60 \times 10_8$	5.30×10^8
21	TNTC	TNTC	2.56×10^9	2.40×10^9	71	TNTC	TNTC	$7.88 \times 10_8$	7.40×10^8
22	TNTC	4.55×10^6	8.70×10^8	8.30×10^8	72	TNTC	TNTC	$6.30 \times 10_8$	6.00×10^8
23	TNTC	TNTC	9.77×10^8	9.10×10^8	73	TNTC	3.10×10^7	$9.10 \times 10_8$	TFTC
24	TNTC	TNTC	6.22×10^8	5.99×10^8	74	TNTC	TNTC	$1.14 \times 10_9$	1.10×10^9
25	TNTC	TNTC	8.30×10^8	7.90×10^8	75	TNTC	TNTC	$8.55 \times 10_8$	8.20×10^8
26	TNTC	TNTC	7.14×10^8	6.80×10^8	76	TNTC	TNTC	$4.98 \times 10_8$	4.70×10^8
27	TNTC	TNTC	6.00×10^8	5.40×10^8	77	TNTC	TNTC	$2.44 \times 10_9$	2.30×10^9
28	TNTC	TNTC	9.17×10^8	8.80×10^8	78	TNTC	TNTC	$7.75 \times 10_8$	7.40×10^8
29	TNTC	1.45×10^7	5.80×10^8	5.60×10^8	79	TNTC	2.55×10^7	$9.22 \times 10_8$	4.70×10^8

30	TNTC	TNTC	4.95×10^8	5.10×10^8	80	TNTC	TNTC	$6.88 \times 10_8$	6.50×10^8
31	TNTC	TNTC	3.90×10^8	3.80×10^8	81	TNTC	TNTC	$3.77 \times 10_8$	3.60×10^8
32	TNTC	TNTC	9.77×10^8	7.21×10^8	82	TNTC	TNTC	$1.19 \times 10_9$	1.10×10^9
33	TNTC	TNTC	6.70×10^8	6.50×10^8	83	TNTC	7.88×10^8	$8.44 \times 10_8$	8.20×10^8
34	TNTC	TNTC	8.22×10^8	8.10×10^8	84	$8.88 \times 10_8$	3.22×10^7	$7.66 \times 10_8$	TFTC
35	TNTC	TNTC	2.65×10^9	2.40×10^9	85	TNTC	TNTC	$9.55 \times 10_8$	9.20×10^8
36	TNTC	5.60×10^6	9.10×10^8	8.60×10^8	86	TNTC	TNTC	$7.90 \times 10_8$	7.50×10^8
37	TNTC	TNTC	7.44×10^8	7.00×10^8	87	TNTC	TNTC	$1.28 \times 10_9$	1.20×10^9
38	TNTC	TNTC	8.67×10^8	8.10×10^8	88	$7.21 \times 10_8$	2.44×10^7	$8.77 \times 10_8$	TFTC
39	TNTC	TNTC	9.33×10^8	9.10×10^8	89	TNTC	TNTC	$6.77 \times 10_8$	6.50×10^8
40	TNTC	TNTC	1.11×10^9	1.05×10^9	90	TNTC	TNTC	$9.44 \times 10_8$	9.00×10^8
41	TNTC	TNTC	2.90×10^9	2.70×10^9	91	TNTC	TNTC	$3.66 \times 10_8$	3.50×10^8
42	TNTC	TNTC	5.87×10^8	5.60×10^8	92	TNTC	TNTC	$8.11 \times 10_8$	7.80×10^8
43	TNTC	TNTC	TFTC	TFTC	93	$9.77 \times 10_8$	2.55×10^7	TFTC	TFTC
44	TNTC	TNTC	1.02×10^9	9.90×10^8	94	TNTC	TNTC	$1.20 \times 10_9$	TFTC
45	TNTC	TNTC	4.77×10^8	4.50×10^8	95	TNTC	TNTC	$4.88 \times 10_8$	4.60×10^8

46	TNTC	3.22×10^7	7.99×10^8	7.50×10^8	96	TNTC	TNTC	$9.66 \times 10_8$	9.20×10^8
47	TNTC	TNTC	9.12×10^8	8.70×10^8	97	TNTC	TNTC	$2.55 \times 10_9$	2.40×10^9
48	TNTC	TNTC	3.67×10^9	3.40×10^9	98	$1.01 \times 10_9$	3.40×10^7	$8.88 \times 10_8$	8.50×10^8
49	TNTC	TNTC	TFTC	TFTC	99	TNTC	TNTC	$6.44 \times 10_8$	6.20×10^8
50	TNTC	TNTC	2.45×10^9	2.30×10^9	100	TNTC	TNTC	$1.30 \times 10_9$	1.20×10^9

Out of 50 patients and 50 control stool samples, sample (1-50) showed heavier colony growth than sample (51-100). In case of patient samples (1-50), the majority yield of countable colonies are from 10^{-6} and 10^{-8} dilution, whereas the lower dilutions were mostly TNTC (Too Numerous To Count). The patient samples demonstrated CFU value ranging from 2.97×10^3 to 3.40×10^9 with most clustering around 10^8 - 10^9 CFU/ml range. In comparison, CFU results of control samples show lower bacterial colonial growth at lower dilution and at even higher dilution, the colony numbers are comparatively lower than the numbers from patient samples. Though the ranges are not meaningfully different, however, the gap is still comparable to state that a substantial increase in bacterial colonies among the patients of psoriasis occurred.

13.2 ELISA:

IL-6

<u>IL-6</u>												
<u>2025-06-16T19:42:07+06:00</u>												
<u>Absorbance</u> <u>1</u>												
<u>Wavelength:</u> <u>450 nm</u>												
<u>Plate 1</u>	<u>Colu</u> <u>mn</u> <u>1</u>	<u>Colu</u> <u>mn</u> <u>2</u>	<u>Colu</u> <u>mn</u> <u>3</u>	<u>Colu</u> <u>mn</u> <u>4</u>	<u>Colu</u> <u>mn</u> <u>5</u>	<u>Colu</u> <u>mn</u> <u>6</u>	<u>Colu</u> <u>mn</u> <u>7</u>	<u>Colu</u> <u>mn</u> <u>8</u>	<u>Colu</u> <u>mn</u> <u>9</u>	<u>Colu</u> <u>mn</u> <u>10</u>	<u>Colu</u> <u>mn</u> <u>11</u>	<u>Colu</u> <u>mn</u> <u>12</u>
<u>Abs</u>	<u>1.00</u> <u>00</u>	<u>2.00</u> <u>00</u>	<u>3.00</u> <u>00</u>	<u>4.00</u> <u>00</u>	<u>5.00</u> <u>00</u>	<u>6.00</u> <u>00</u>	<u>7.00</u> <u>00</u>	<u>8.00</u> <u>00</u>	<u>9.00</u> <u>00</u>	<u>10.0</u> <u>000</u>	<u>11.0</u> <u>000</u>	<u>12.0</u> <u>000</u>
<u>A</u>	<u>0.07</u> <u>97</u>	<u>0.04</u> <u>38</u>	<u>0.04</u> <u>36</u>	<u>0.04</u> <u>88</u>	<u>0.04</u> <u>41</u>	<u>0.03</u> <u>93</u>	<u>0.03</u> <u>76</u>	<u>0.04</u> <u>53</u>	<u>0.04</u> <u>21</u>	<u>0.04</u> <u>20</u>	<u>0.03</u> <u>85</u>	<u>0.04</u> <u>38</u>
<u>B</u>	<u>0.02</u> <u>74</u>	<u>0.95</u> <u>26</u>	<u>0.04</u> <u>61</u>	<u>0.05</u> <u>55</u>	<u>0.04</u> <u>74</u>	<u>0.04</u> <u>29</u>	<u>0.03</u> <u>66</u>	<u>0.04</u> <u>87</u>	<u>0.04</u> <u>74</u>	<u>0.04</u> <u>86</u>	<u>0.05</u> <u>29</u>	<u>0.03</u> <u>86</u>
<u>C</u>	<u>0.04</u> <u>93</u>	<u>0.04</u> <u>15</u>	<u>0.04</u> <u>00</u>	<u>0.04</u> <u>75</u>	<u>0.05</u> <u>15</u>	<u>0.04</u> <u>44</u>	<u>0.03</u> <u>65</u>	<u>0.04</u> <u>86</u>	<u>0.04</u> <u>96</u>	<u>0.03</u> <u>86</u>	<u>0.03</u> <u>42</u>	<u>0.05</u> <u>27</u>
<u>D</u>	<u>0.04</u> <u>87</u>	<u>0.03</u> <u>29</u>	<u>0.03</u> <u>92</u>	<u>0.03</u> <u>83</u>	<u>0.00</u> <u>00</u>	<u>0.03</u> <u>88</u>	<u>0.08</u> <u>59</u>	<u>0.57</u> <u>97</u>	<u>0.06</u> <u>01</u>	<u>0.03</u> <u>40</u>	<u>0.04</u> <u>65</u>	<u>0.05</u> <u>78</u>
<u>E</u>	<u>0.28</u> <u>91</u>	<u>0.29</u> <u>39</u>	<u>0.29</u> <u>92</u>	<u>0.08</u> <u>97</u>	<u>0.39</u> <u>19</u>	<u>1.53</u> <u>53</u>	<u>1.41</u> <u>58</u>	<u>0.05</u> <u>04</u>	<u>0.06</u> <u>53</u>	<u>0.08</u> <u>72</u>	<u>0.03</u> <u>46</u>	<u>0.03</u> <u>28</u>
<u>F</u>	<u>0.28</u> <u>79</u>	<u>0.28</u> <u>60</u>	<u>0.28</u> <u>25</u>	<u>0.08</u> <u>98</u>	<u>0.28</u> <u>34</u>	<u>0.28</u> <u>355</u>	<u>0.24</u> <u>95</u>	<u>0.24</u> <u>95</u>	<u>0.23</u> <u>21</u>	<u>0.28</u> <u>21</u>	<u>0.28</u> <u>47</u>	<u>0.20</u> <u>65</u>

<u>G</u>	<u>0.28</u> <u>64</u>	<u>0.29</u> <u>98</u>	<u>0.28</u> <u>34</u>	<u>0.28</u> <u>56</u>	<u>0.28</u> <u>53</u>	<u>0.28</u> <u>44</u>	<u>0.28</u> <u>70</u>	<u>0.28</u> <u>11</u>	<u>0.28</u> <u>93</u>	<u>0.38</u> <u>73</u>	<u>0.10</u> <u>3</u>	<u>0.28</u> <u>05</u>
<u>H</u>	<u>0.28</u> <u>37</u>	<u>0.28</u> <u>98</u>	<u>0.28</u> <u>37</u>	<u>0.28</u> <u>17</u>	<u>0.39</u> <u>18</u>	<u>0.22</u> <u>70</u>	<u>0.26</u> <u>54</u>	<u>0.28</u> <u>24</u>	<u>0.28</u> <u>73</u>	<u>0.28</u> <u>86</u>	<u>0.28</u> <u>11</u>	<u>0.28</u> <u>29</u>
Sample	1	2	3	4	5	6	7	8	9	10	11	12
<u>A</u>	<u>Un0</u> <u>001</u>	<u>Un0</u> <u>002</u>	<u>Un0</u> <u>003</u>	<u>Un0</u> <u>004</u>	<u>Un0</u> <u>005</u>	<u>Un0</u> <u>006</u>	<u>Un0</u> <u>007</u>	<u>Un0</u> <u>008</u>	<u>Un0</u> <u>009</u>	<u>Un0</u> <u>010</u>	<u>Un0</u> <u>011</u>	<u>Un0</u> <u>012</u>
<u>B</u>	<u>Un0</u> <u>013</u>	<u>Un0</u> <u>014</u>	<u>Un0</u> <u>015</u>	<u>Un0</u> <u>016</u>	<u>Un0</u> <u>017</u>	<u>Un0</u> <u>018</u>	<u>Un0</u> <u>019</u>	<u>Un0</u> <u>020</u>	<u>Un0</u> <u>021</u>	<u>Un0</u> <u>022</u>	<u>Un0</u> <u>023</u>	<u>Un0</u> <u>024</u>
<u>C</u>	<u>Un0</u> <u>025</u>	<u>Un0</u> <u>026</u>	<u>Un0</u> <u>027</u>	<u>Un0</u> <u>028</u>	<u>Un0</u> <u>029</u>	<u>Un0</u> <u>030</u>	<u>Un0</u> <u>031</u>	<u>Un0</u> <u>032</u>	<u>Un0</u> <u>033</u>	<u>Un0</u> <u>034</u>	<u>Un0</u> <u>035</u>	<u>Un0</u> <u>036</u>
<u>D</u>	<u>Un0</u> <u>037</u>	<u>Un0</u> <u>038</u>	<u>Un0</u> <u>039</u>	<u>Un0</u> <u>040</u>	<u>Blan</u> <u>k1</u>	<u>Std0</u> <u>001</u>	<u>Std0</u> <u>002</u>	<u>Std0</u> <u>003</u>	<u>Un0</u> <u>041</u>	<u>Un0</u> <u>042</u>	<u>Un0</u> <u>043</u>	<u>Un0</u> <u>044</u>
<u>E</u>	<u>Un0</u> <u>083</u>	<u>Un0</u> <u>084</u>	<u>Un0</u> <u>085</u>	<u>Un0</u> <u>086</u>	<u>Un0</u> <u>087</u>	<u>Un0</u> <u>088</u>	<u>Un0</u> <u>089</u>	<u>Un0</u> <u>090</u>	<u>Un0</u> <u>091</u>	<u>Un0</u> <u>092</u>	<u>Un0</u> <u>045</u>	<u>Un0</u> <u>046</u>
<u>F</u>	<u>Un0</u> <u>071</u>	<u>Un0</u> <u>072</u>	<u>Un0</u> <u>073</u>	<u>Un0</u> <u>074</u>	<u>Un0</u> <u>075</u>	<u>Un0</u> <u>076</u>	<u>Un0</u> <u>077</u>	<u>Un0</u> <u>078</u>	<u>Un0</u> <u>079</u>	<u>Un0</u> <u>080</u>	<u>Un0</u> <u>081</u>	<u>Un0</u> <u>082</u>
<u>G</u>	<u>Un0</u> <u>059</u>	<u>Un0</u> <u>060</u>	<u>Un0</u> <u>061</u>	<u>Un0</u> <u>062</u>	<u>Un0</u> <u>063</u>	<u>Un0</u> <u>064</u>	<u>Un0</u> <u>065</u>	<u>Un0</u> <u>066</u>	<u>Un0</u> <u>067</u>	<u>Un0</u> <u>068</u>	<u>Un0</u> <u>069</u>	<u>Un0</u> <u>070</u>
<u>H</u>	<u>Un0</u> <u>047</u>	<u>Un0</u> <u>048</u>	<u>Un0</u> <u>049</u>	<u>Un0</u> <u>050</u>	<u>Un0</u> <u>051</u>	<u>Un0</u> <u>052</u>	<u>Un0</u> <u>053</u>	<u>Un0</u> <u>054</u>	<u>Un0</u> <u>055</u>	<u>Un0</u> <u>056</u>	<u>Un0</u> <u>057</u>	<u>Un0</u> <u>058</u>

By using a software, the **Cut-off value** was determined to be **0.2049**, we can clearly observe that most of the samples from the patients have values more than the cut off value, which indicates that the target analyte is present in the samples at a detectable range. Some of the patient samples like E4,E8,E9,E10,F4,G11 have values lower than the cut off value, whereas control samples like B2 have a much higher value than the cut off value.

This report is generated by ElisaInfo® (Boster's ELISA Calculator).

Experiment name:		IL-6					
Standard concentration unit		600					
Equation	Linear	ID	Well	Raw Data	Average	Concentration	Std Deviation
		S1	D6	0.0388	0.039	0.0388	0
A	1.00028	S2	D7	0.0859	0.086	0.0859	0
B	0.00014	S3	D8	0.5797	0.58	0.5797	0
		BL	D5	0.0	0	0	0
R2	1	C1	A1	0.0797	0.08	0.08	0
		C10	A10	0.042	0.042	0.042	0
		C11	A11	0.0385	0.038	0.038	0
		C12	A12	0.0438	0.044	0.044	0
		C13	B1	0.0274	0.027	0.027	0
		C14	B2	0.9526	0.953	0.953	0
		C15	B3	0.0461	0.046	0.046	0
		C16	B4	0.0555	0.056	0.056	0
		C17	B5	0.0474	0.047	0.047	0
		C18	B6	0.0429	0.043	0.043	0

			C19	B7	0.0366	0.037	0.037	0
			C2	A2	0.0438	0.044	0.044	0
			C20	B8	0.0487	0.049	0.049	0
			C21	B9	0.0474	0.047	0.047	0
			C22	B10	0.0486	0.049	0.049	0
			C23	B11	0.0529	0.053	0.053	0
			C24	B12	0.0386	0.039	0.039	0
			C25	C1	0.0493	0.049	0.049	0
			C26	C2	0.0415	0.042	0.042	0
			C27	C3	0.04	0.04	0.04	0
			C28	C4	0.0475	0.048	0.048	0
			C29	C5	0.0515	0.052	0.052	0
			C3	A3	0.0436	0.044	0.044	0
			C30	C6	0.0444	0.044	0.044	0
			C31	C7	0.0365	0.036	0.036	0
			C32	C8	0.0486	0.049	0.049	0
			C33	C9	0.0496	0.05	0.05	0
			C34	C10	0.0386	0.039	0.039	0

			C35	C11	0.0342	0.034	0.034	0
			C36	C12	0.0527	0.053	0.053	0
			C37	D1	0.0487	0.049	0.049	0
			C38	D2	0.0329	0.033	0.033	0
			C39	D3	0.0392	0.039	0.039	0
			C4	A4	0.0488	0.049	0.049	0
			C40	D4	0.0383	0.038	0.038	0
			C41	D9	0.0601	0.06	0.06	0
			C42	D10	0.034	0.034	0.034	0
			C43	D11	0.0465	0.046	0.046	0
			C44	D12	0.0578	0.058	0.058	0
			C45	E11	0.0346	0.035	0.035	0
			C46	E12	0.0328	0.033	0.033	0
			C5	A5	0.0441	0.044	0.044	0
			C6	A6	0.0393	0.039	0.039	0
			C7	A7	0.0376	0.038	0.038	0
			C8	A8	0.0453	0.045	0.045	0
			C9	A9	0.0421	0.042	0.042	0

			U1	H1	0.2837	0.284	0.284	0
			U10	H10	0.2886	0.289	0.289	0
			U11	H11	0.2811	0.281	0.281	0
			U12	H12	0.2829	0.283	0.283	0
			U13	G1	0.2864	0.286	0.286	0
			U14	G2	0.2998	0.3	0.3	0
			U15	G3	0.2834	0.283	0.283	0
			U16	G4	0.2856	0.286	0.286	0
			U17	G5	0.2853	0.285	0.285	0
			U18	G6	0.2844	0.284	0.284	0
			U19	G7	0.287	0.287	0.287	0
			U2	H2	0.2898	0.29	0.29	0
			U20	G8	0.2811	0.281	0.281	0
			U21	G9	0.2893	0.289	0.289	0
			U22	G10	0.3873	0.387	0.387	0
			U23	G11	0.103	0.103	0.103	0
			U24	G12	0.2805	0.28	0.28	0
			U25	F1	0.2879	0.288	0.288	0

			U26	F2	0.286	0.286	0.286	0
			U27	F3	0.2825	0.282	0.282	0
			U28	F4	0.0898	0.09	0.09	0
			U29	F5	0.2834	0.283	0.283	0
			U3	H3	0.2837	0.284	0.284	0
			U30	F6	0.28355	0.284	0.284	0
			U31	F7	0.2495	0.25	0.25	0
			U32	F8	0.2495	0.25	0.25	0
			U33	F9	0.2321	0.232	0.232	0
			U34	F10	0.2821	0.282	0.282	0
			U35	F11	0.2847	0.285	0.285	0
			U36	F12	0.2035	0.204	0.204	0
			U37	E1	0.2891	0.289	0.289	0
			U38	E2	0.2939	0.294	0.294	0
			U39	E3	0.2992	0.299	0.299	0
			U4	H4	0.2817	0.282	0.282	0
			U40	E4	0.0897	0.09	0.09	0
			U41	E5	0.3919	0.392	0.392	0

			U42	E6	1.5353	1.535	1.534	0
			U43	E7	1.4158	1.416	1.415	0
			U44	E8	0.0504	0.05	0.05	0
			U45	E9	0.0653	0.065	0.065	0
			U46	E10	0.0872	0.087	0.087	0
			U5	H5	0.3918	0.392	0.392	0
			U6	H6	0.227	0.227	0.227	0
			U7	H7	0.2654	0.265	0.265	0
			U8	H8	0.2824	0.282	0.282	0
			U9	H9	0.2873	0.287	0.287	0

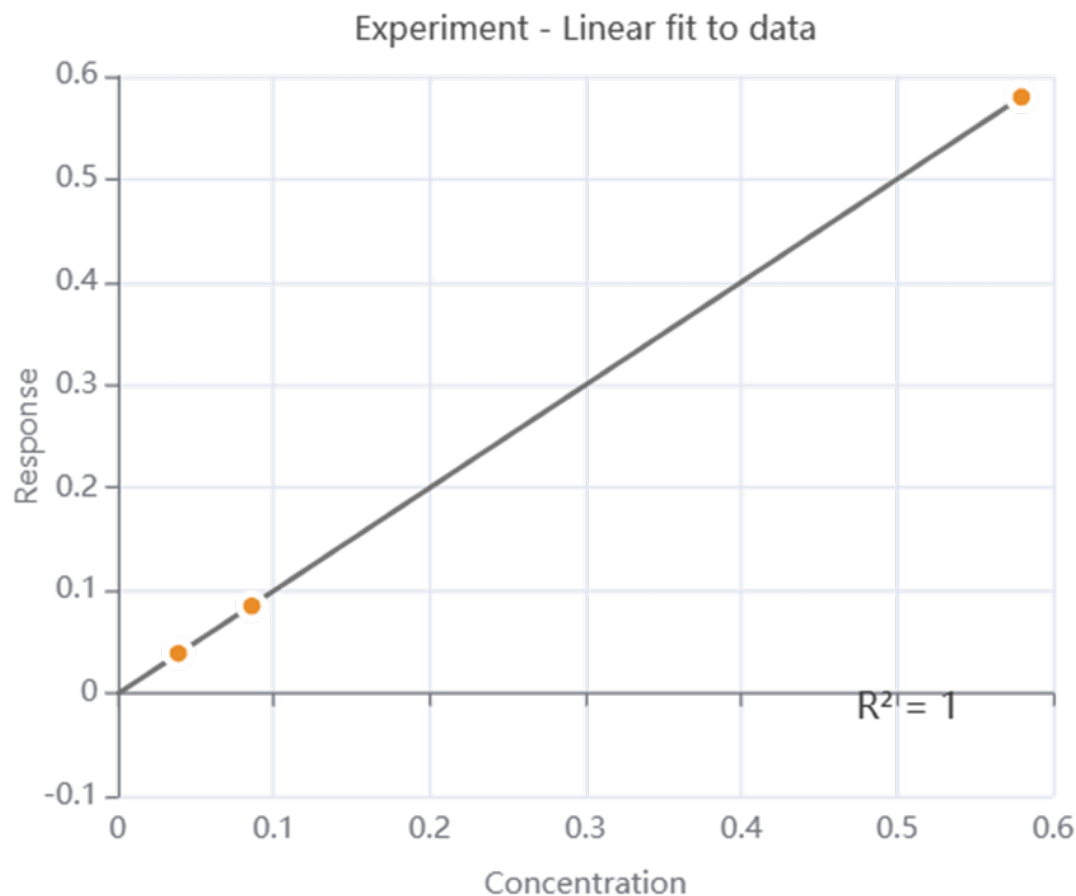


Figure 14: Linear Regression Diagram for IL-6

Figure no. 14 indicates the current ELISA analysis for IL-6, where the regression equation of the standard curve yielded an R^2 value equal to 1.0 showing a perfect correlation between the known absorbance levels of the standards and the known levels of the standards, respectively. This implies that the experiment was conducted with high accuracy and the record was very consistent with the established regression model. It should be noted too though that this ideal fit can be in some measure attributed to the small number of standards that were used. The number of standards employed in this situation was limited to three, which predisposes a greater success in reaching an artificially large R^2 , as the smaller the number of data points, the lower the probability of random fluctuation of the curve fittings.

IL-17

IL17.skax												
2025-06-18T16:32:53+06:00												
Absorbance	1											
Wavelength: 450 nm												
Plate 1	Colu mn 1	Colu mn 2	Colu mn 3	Colu mn 4	Colu mn 5	Colu mn 6	Colu mn 7	Colu mn 8	Colu mn 9	Colu mn 10	Colu mn 11	Colu mn 12
Abs	<u>1.00</u> <u>00</u>	<u>2.00</u> <u>00</u>	<u>3.00</u> <u>00</u>	<u>4.00</u> <u>00</u>	<u>5.00</u> <u>00</u>	<u>6.00</u> <u>00</u>	<u>7.00</u> <u>00</u>	<u>8.00</u> <u>00</u>	<u>9.00</u> <u>00</u>	<u>10.0</u> <u>000</u>	<u>11.0</u> <u>000</u>	<u>12.0</u> <u>000</u>
A	<u>0.04</u> <u>59</u>	<u>0.04</u> <u>53</u>	<u>0.04</u> <u>47</u>	<u>0.04</u> <u>58</u>	<u>0.04</u> <u>64</u>	<u>0.04</u> <u>98</u>	<u>0.04</u> <u>75</u>	<u>0.04</u> <u>47</u>	<u>0.04</u> <u>64</u>	<u>0.04</u> <u>40</u>	<u>0.04</u> <u>39</u>	<u>0.04</u> <u>68</u>
B	<u>0.05</u> <u>00</u>	<u>0.61</u> <u>50</u>	<u>0.04</u> <u>54</u>	<u>0.04</u> <u>66</u>	<u>0.04</u> <u>59</u>	<u>0.04</u> <u>69</u>	<u>0.04</u> <u>99</u>	<u>0.04</u> <u>52</u>	<u>0.04</u> <u>40</u>	<u>0.04</u> <u>65</u>	<u>0.04</u> <u>94</u>	<u>0.04</u> <u>49</u>
C	<u>0.04</u> <u>45</u>	<u>0.04</u> <u>79</u>	<u>0.04</u> <u>45</u>	<u>0.02</u> <u>23</u>	<u>0.04</u> <u>76</u>	<u>0.04</u> <u>51</u>	<u>0.04</u> <u>75</u>	<u>0.04</u> <u>78</u>	<u>0.04</u> <u>42</u>	<u>0.04</u> <u>45</u>	<u>0.06</u> <u>90</u>	<u>0.04</u> <u>94</u>
D	<u>0.04</u> <u>47</u>	<u>0.04</u> <u>33</u>	<u>0.04</u> <u>42</u>	<u>0.04</u> <u>54</u>	<u>0.00</u> <u>00</u>	<u>1.04</u> <u>46</u>	<u>0.55</u> <u>07</u>	<u>0.87</u> <u>7</u>	<u>0.04</u> <u>34</u>	<u>0.04</u> <u>40</u>	<u>0.04</u> <u>57</u>	<u>0.04</u> <u>92</u>
E	<u>0.14</u> <u>54</u>	<u>0.14</u> <u>51</u>	<u>0.15</u> <u>63</u>	<u>0.34</u> <u>46</u>	<u>0.24</u> <u>56</u>	<u>0.04</u> <u>55</u>	<u>0.24</u> <u>56</u>	<u>0.24</u> <u>44</u>	<u>0.24</u> <u>54</u>	<u>0.24</u> <u>57</u>	<u>0.04</u> <u>65</u>	<u>0.04</u> <u>66</u>
F	<u>0.24</u> <u>59</u>	<u>0.15</u> <u>37</u>	<u>0.24</u> <u>66</u>	<u>0.24</u> <u>82</u>	<u>0.14</u> <u>38</u>	<u>0.14</u> <u>77</u>	<u>0.24</u> <u>53</u>	<u>0.24</u> <u>50</u>	<u>0.14</u> <u>54</u>	<u>0.24</u> <u>72</u>	<u>0.24</u> <u>98</u>	<u>0.34</u> <u>80</u>
G	<u>0.24</u> <u>85</u>	<u>0.25</u> <u>01</u>	<u>0.24</u> <u>48</u>	<u>0.24</u> <u>54</u>	<u>0.43</u> <u>50</u>	<u>0.24</u> <u>46</u>	<u>0.24</u> <u>65</u>	<u>0.14</u> <u>55</u>	<u>0.14</u> <u>51</u>	<u>0.24</u> <u>50</u>	<u>0.34</u> <u>91</u>	<u>0.25</u> <u>15</u>
H	<u>0.16</u> <u>77</u>	<u>0.24</u> <u>54</u>	<u>0.24</u> <u>34</u>	<u>0.24</u> <u>49</u>	<u>0.24</u> <u>48</u>	<u>0.25</u> <u>24</u>	<u>0.24</u> <u>25</u>	<u>0.24</u> <u>57</u>	<u>0.25</u> <u>33</u>	<u>0.14</u> <u>63</u>	<u>0.25</u> <u>32</u>	<u>0.24</u> <u>58</u>

Sample	1	2	3	4	5	6	7	8	9	10	11	12
<u>A</u>	<u>Un0</u> <u>001</u>	<u>Un0</u> <u>002</u>	<u>Un0</u> <u>003</u>	<u>Un0</u> <u>004</u>	<u>Un0</u> <u>005</u>	<u>Un0</u> <u>006</u>	<u>Un0</u> <u>007</u>	<u>Un0</u> <u>008</u>	<u>Un0</u> <u>009</u>	<u>Un0</u> <u>010</u>	<u>Un0</u> <u>011</u>	<u>Un0</u> <u>012</u>
<u>B</u>	<u>Un0</u> <u>013</u>	<u>Un0</u> <u>014</u>	<u>Un0</u> <u>015</u>	<u>Un0</u> <u>016</u>	<u>Un0</u> <u>017</u>	<u>Un0</u> <u>018</u>	<u>Un0</u> <u>019</u>	<u>Un0</u> <u>020</u>	<u>Un0</u> <u>021</u>	<u>Un0</u> <u>022</u>	<u>Un0</u> <u>023</u>	<u>Un0</u> <u>024</u>
<u>C</u>	<u>Un0</u> <u>025</u>	<u>Un0</u> <u>026</u>	<u>Un0</u> <u>027</u>	<u>Un0</u> <u>028</u>	<u>Un0</u> <u>029</u>	<u>Un0</u> <u>030</u>	<u>Un0</u> <u>031</u>	<u>Un0</u> <u>032</u>	<u>Un0</u> <u>033</u>	<u>Un0</u> <u>034</u>	<u>Un0</u> <u>035</u>	<u>Un0</u> <u>036</u>
<u>D</u>	<u>Un0</u> <u>037</u>	<u>Un0</u> <u>038</u>	<u>Un0</u> <u>039</u>	<u>Un0</u> <u>040</u>	<u>Blan</u> <u>k1</u>	<u>Std0</u> <u>001</u>	<u>Std0</u> <u>002</u>	<u>Std0</u> <u>003</u>	<u>Un0</u> <u>041</u>	<u>Un0</u> <u>042</u>	<u>Un0</u> <u>043</u>	<u>Un0</u> <u>044</u>
<u>E</u>	<u>Un0</u> <u>083</u>	<u>Un0</u> <u>084</u>	<u>Un0</u> <u>085</u>	<u>Un0</u> <u>086</u>	<u>Un0</u> <u>087</u>	<u>Un0</u> <u>088</u>	<u>Un0</u> <u>089</u>	<u>Un0</u> <u>090</u>	<u>Un0</u> <u>091</u>	<u>Un0</u> <u>092</u>	<u>Un0</u> <u>045</u>	<u>Un0</u> <u>046</u>
<u>F</u>	<u>Un0</u> <u>071</u>	<u>Un0</u> <u>072</u>	<u>Un0</u> <u>073</u>	<u>Un0</u> <u>074</u>	<u>Un0</u> <u>075</u>	<u>Un0</u> <u>076</u>	<u>Un0</u> <u>077</u>	<u>Un0</u> <u>078</u>	<u>Un0</u> <u>079</u>	<u>Un0</u> <u>080</u>	<u>Un0</u> <u>081</u>	<u>Un0</u> <u>082</u>
<u>G</u>	<u>Un0</u> <u>059</u>	<u>Un0</u> <u>060</u>	<u>Un0</u> <u>061</u>	<u>Un0</u> <u>062</u>	<u>Un0</u> <u>063</u>	<u>Un0</u> <u>064</u>	<u>Un0</u> <u>065</u>	<u>Un0</u> <u>066</u>	<u>Un0</u> <u>067</u>	<u>Un0</u> <u>068</u>	<u>Un0</u> <u>069</u>	<u>Un0</u> <u>070</u>
<u>H</u>	<u>Un0</u> <u>047</u>	<u>Un0</u> <u>048</u>	<u>Un0</u> <u>049</u>	<u>Un0</u> <u>050</u>	<u>Un0</u> <u>051</u>	<u>Un0</u> <u>052</u>	<u>Un0</u> <u>053</u>	<u>Un0</u> <u>054</u>	<u>Un0</u> <u>055</u>	<u>Un0</u> <u>056</u>	<u>Un0</u> <u>057</u>	<u>Un0</u> <u>058</u>

By using a software, the **Cut-off value** was calculated to be **0.1424**, we can clearly observe that most of the samples from the patients have values more than the cut off value, which indicates that the target analyte is present in the samples at a detectable range. One of the patient samples like E6 have values lower than the cut off value, whereas control samples like B2 have a much higher value than the cut off value.

This report is generated by ElisaInfo® (Boster's ELISA Calculator).							
Experiment name	IL-17						
Standard concentration unit							
600							

Equation	Linear		ID	Wells	Raw Data	Average	Concentration	Std Deviation
			S1	D6	1.0446	1.045	1.0446	0
A	1.00005		S2	D7	0.5507	0.551	0.5507	0
B	0.00019		S3	D8	0.877	0.877	0.877	0
			BL	D5	0.0	0	0	0
R2	1		C1	A1	0.0459	0.046	0.046	0
			C10	A10	0.044	0.044	0.044	0
			C11	A11	0.0439	0.044	0.044	0
			C12	A12	0.0468	0.047	0.047	0
			C13	B1	0.05	0.05	0.05	0
			C14	B2	0.615	0.615	0.615	0
			C15	B3	0.0454	0.045	0.045	0
			C16	B4	0.0466	0.047	0.047	0
			C17	B5	0.0459	0.046	0.046	0
			C18	B6	0.0469	0.047	0.047	0
			C19	B7	0.0499	0.05	0.05	0
			C2	A2	0.0453	0.045	0.045	0

			C20	B8	0.0452	0.045	0.045	0
			C21	B9	0.044	0.044	0.044	0
			C22	B10	0.0465	0.046	0.046	0
			C23	B11	0.0494	0.049	0.049	0
			C24	B12	0.0449	0.045	0.045	0
			C25	C1	0.0445	0.044	0.044	0
			C26	C2	0.0479	0.048	0.048	0
			C27	C3	0.0445	0.044	0.044	0
			C28	C4	0.0223	0.022	0.022	0
			C29	C5	0.0476	0.048	0.048	0
			C3	A3	0.0447	0.045	0.045	0
			C30	C6	0.0451	0.045	0.045	0
			C31	C7	0.0475	0.048	0.048	0
			C32	C8	0.0478	0.048	0.048	0
			C33	C9	0.0442	0.044	0.044	0
			C34	C10	0.0445	0.044	0.044	0
			C35	C11	0.069	0.069	0.069	0
			C36	C12	0.0494	0.049	0.049	0

			C37	D1	0.0447	0.045	0.045	0
			C38	D2	0.0433	0.043	0.043	0
			C39	D3	0.0442	0.044	0.044	0
			C4	A4	0.0458	0.046	0.046	0
			C40	D4	0.0454	0.045	0.045	0
			C41	D9	0.0434	0.043	0.043	0
			C42	D10	0.044	0.044	0.044	0
			C43	D11	0.0457	0.046	0.046	0
			C44	D12	0.0492	0.049	0.049	0
			C45	E11	0.0465	0.046	0.046	0
			C46	E12	0.0466	0.047	0.047	0
			C5	A5	0.0464	0.046	0.046	0
			C6	A6	0.0498	0.05	0.05	0
			C7	A7	0.0475	0.048	0.048	0
			C8	A8	0.0447	0.045	0.045	0
			C9	A9	0.0464	0.046	0.046	0
			U1	H1	0.1677	0.168	0.168	0
			U10	H10	0.1463	0.146	0.146	0

			U11	H11	0.2532	0.253	0.253	0
			U12	H12	0.2458	0.246	0.246	0
			U13	G1	0.2485	0.248	0.248	0
			U14	G2	0.2501	0.25	0.25	0
			U15	G3	0.2448	0.245	0.245	0
			U16	G4	0.2454	0.245	0.245	0
			U17	G5	0.435	0.435	0.435	0
			U18	G6	0.2446	0.245	0.245	0
			U19	G7	0.2465	0.246	0.246	0
			U2	H2	0.2454	0.245	0.245	0
			U20	G8	0.1455	0.146	0.146	0
			U21	G9	0.1451	0.145	0.145	0
			U22	G10	0.245	0.245	0.245	0
			U23	G11	0.3491	0.349	0.349	0
			U24	G12	0.2515	0.252	0.252	0
			U25	F1	0.2459	0.246	0.246	0
			U26	F2	0.1537	0.154	0.154	0
			U27	F3	0.2466	0.247	0.247	0

			U28	F4	0.2482	0.248	0.248	0
			U29	F5	0.1438	0.144	0.144	0
			U3	H3	0.2434	0.243	0.243	0
			U30	F6	0.1477	0.148	0.148	0
			U31	F7	0.2453	0.245	0.245	0
			U32	F8	0.245	0.245	0.245	0
			U33	F9	0.1454	0.145	0.145	0
			U34	F10	0.2472	0.247	0.247	0
			U35	F11	0.2498	0.25	0.25	0
			U36	F12	0.348	0.348	0.348	0
			U37	E1	0.1454	0.145	0.145	0
			U38	E2	0.1451	0.145	0.145	0
			U39	E3	0.1563	0.156	0.156	0
			U4	H4	0.2449	0.245	0.245	0
			U40	E4	0.3446	0.345	0.345	0
			U41	E5	0.2456	0.246	0.246	0
			U42	E6	0.0455	0.046	0.046	0
			U43	E7	0.2456	0.246	0.246	0

			U44	E8	0.2444	0.244	0.244	0
			U45	E9	0.2454	0.245	0.245	0
			U46	E10	0.2457	0.246	0.246	0
			U5	H5	0.2448	0.245	0.245	0
			U6	H6	0.2524	0.252	0.252	0
			U7	H7	0.2425	0.242	0.242	0
			U8	H8	0.2457	0.246	0.246	0
			U9	H9	0.2533	0.253	0.253	0

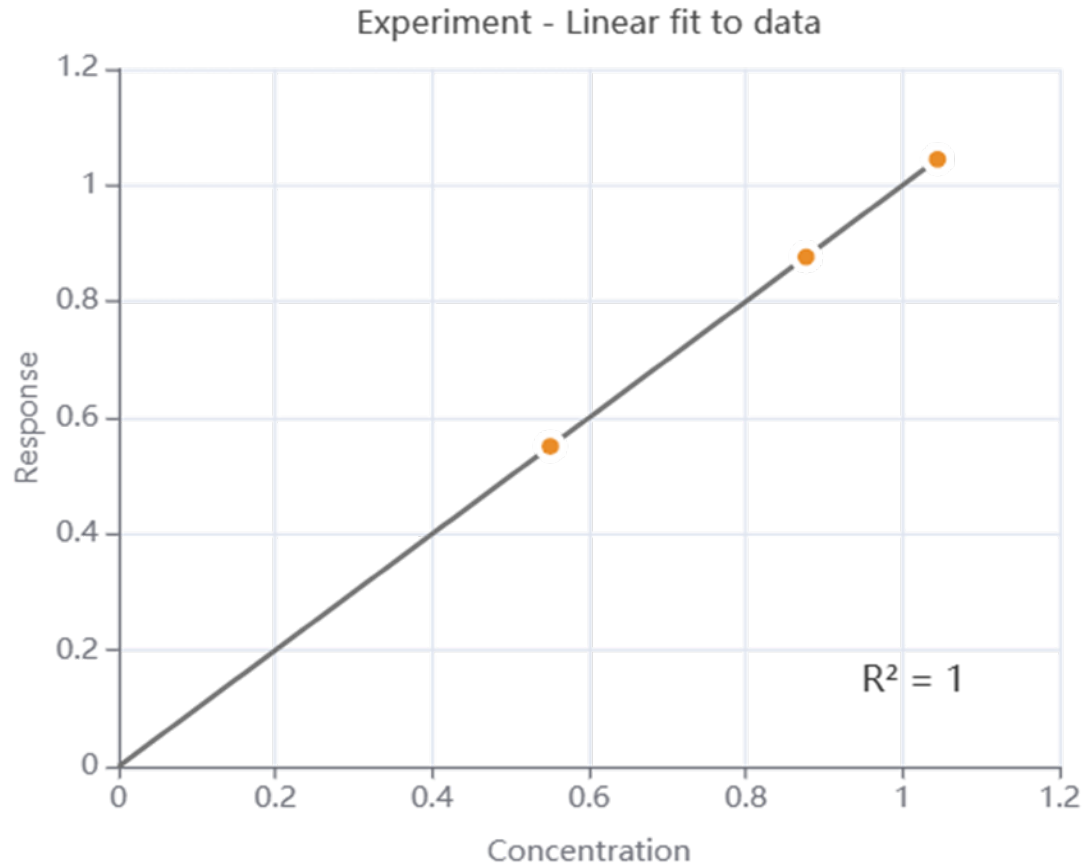


Figure 15: Linear Regression Diagram for IL-17

Figure no. 15 indicates the current ELISA analysis of IL-17 where the regression equation of the standard curve yielded an R^2 value equal to 1.0 showing a perfect correlation between the known absorbance levels of the standards and the known levels of the standards, respectively. This implies that the experiment was conducted with high accuracy and the record was very consistent with the established regression model. It should be noted too though that this ideal fit can be in some measure attributed to the small number of standards that were used. The number of standards employed in this situation was limited to three, which predisposes a greater success in reaching an artificially large R^2 , as the smaller the number of data points, the lower the probability of random fluctuation of the curve fittings.

13.3 PCR:

PCR amplification targeting the *H. pylori* bacteria using 16S rRNA primer produced an expected band of ~500-600 bp. After completing gel electrophoresis,

- The ladder (100 bp) used dispersed properly
- Sample outcomes:

- Positive = Distinct band at ~500-600 bp (sharp bands indicates a strong identification of *H. pylori* and faint bands are still considered positive if not present in NTC)
- Negative = No bands at ~500-600 bp
- Invalid = Samples required repeat testing as a result of issues with amplification, smear or multiple bands

Among the samples, (numbers) produced bands at ~500-600 bp.

The presence of *Helicobacter pylori* in the studied cohort was strongly supported by the results of the amplification of the 16S rRNA gene that yielded distinct bands of ~500-600 bp in the samples of the both control and patient-derived DNA. This finding was in line with what was theoretically expected on the amplicon size; therefore, confirming the primer design and the overall PCR methodology. This can be put in context of a wider picture of molecular diagnostics, epidemiology and clinical implications of detection of *H. pylori*.

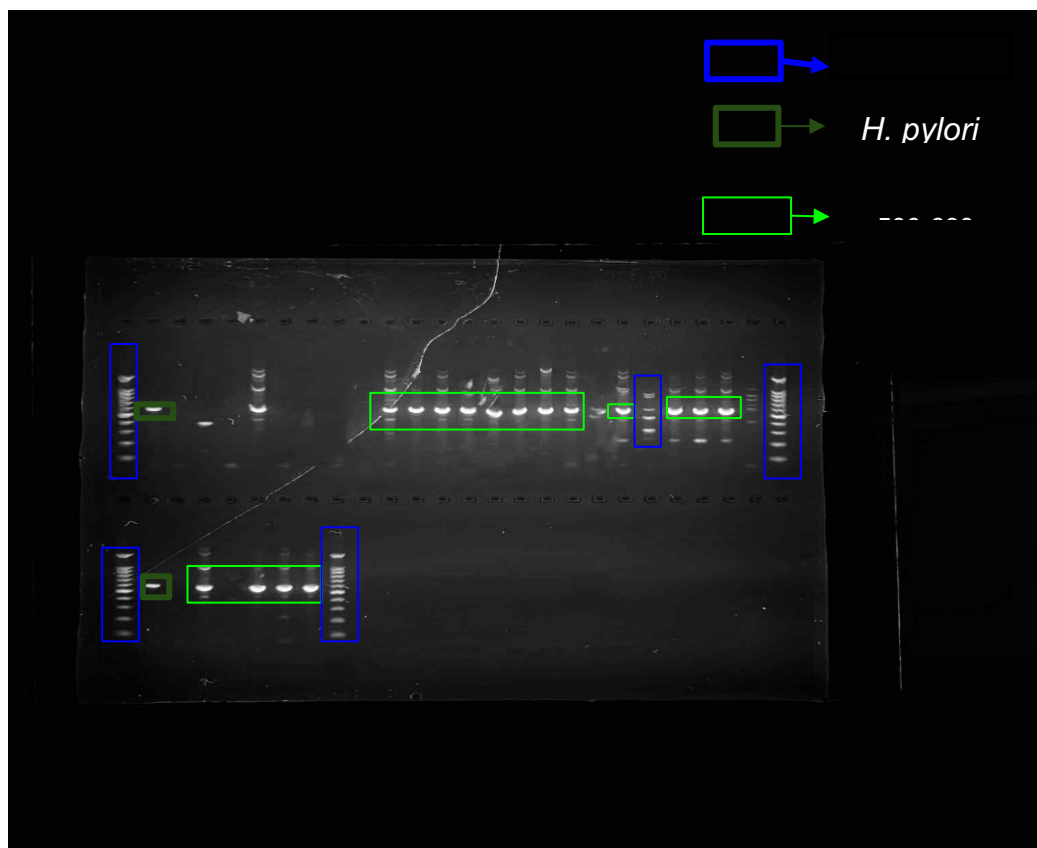


Figure 16 : Results of PCR

*Figure 16 shows the PCR results for *H. pylori* detection. As a reference 100 bp ladder was used shown in blue boxes , and the presence of a 500–600 bp band in the sample confirms positive detection of *H. pylori* DNA that shows in green boxes.*

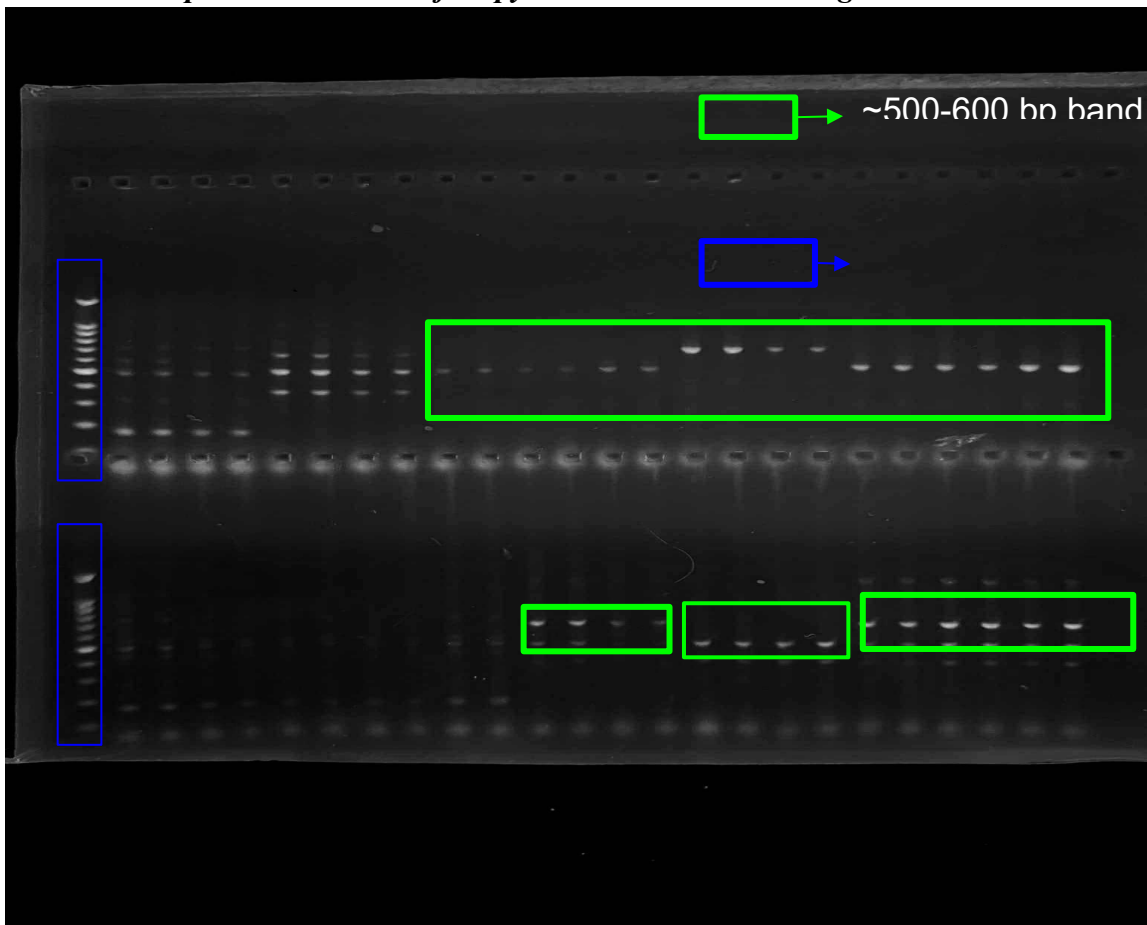


Figure 17: Results of PCR

Figure 17 shows PCR results with a 100 bp DNA ladder used as reference marked in blue boxes. And clear 500-600 bp indicated in green boxes.

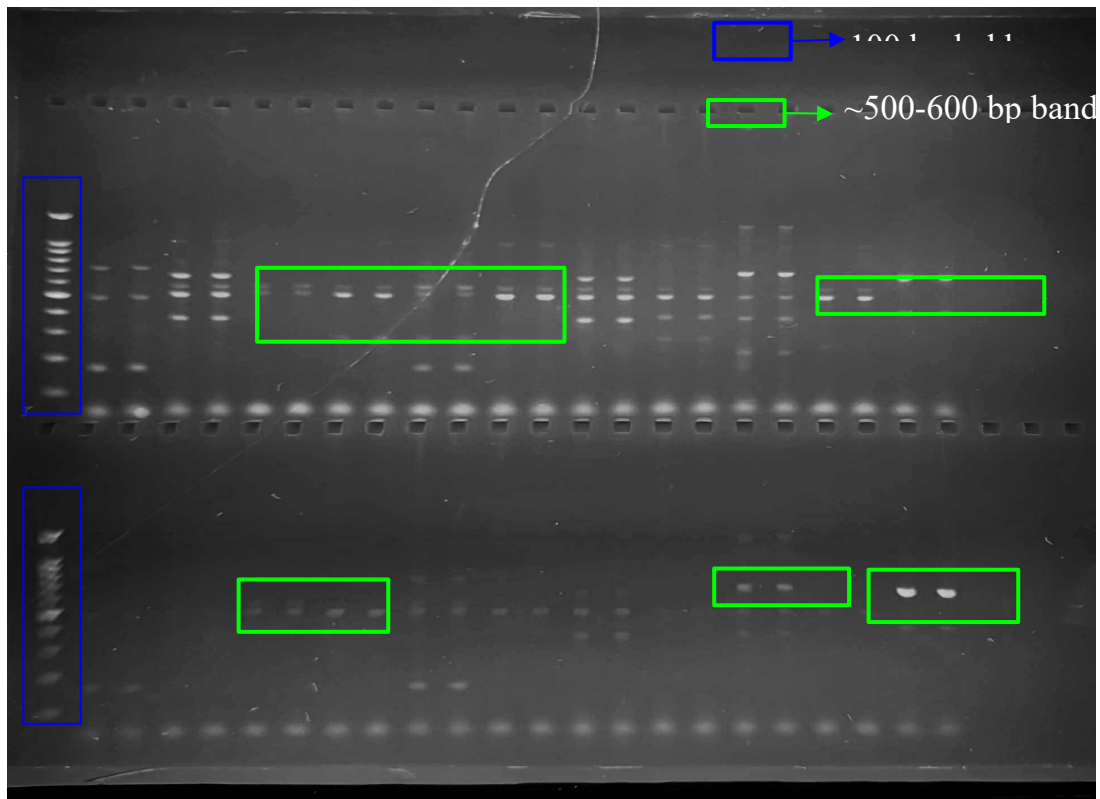


Figure 18: Results of PCR

Figure 18 shows PCR results with a 100 bp DNA ladder used as reference marked in blue boxes. And clear 500-600 bp indicated in green boxes.

14. Discussion:

Psoriasis is a long-lasting, immune-mediated inflammatory disease that can expand beyond the cutaneous manifestations of the disease to systemic involvement and comorbidities. Overwhelming evidence has described the centrality of pro-inflammatory mediators, especially interleukin-6 (IL-6) and interleukin-17 (IL-17) in maintaining the pathogenic loop of immune stimulation and hyperproliferation of the keratinocyte. Such cytokines are central to immunopathology of the disease and their study is clinically relevant as they are potential biomarkers and even therapeutic targets.

Coupled with immune dysregulation, recent studies have focused on the gut-skin axis whereby disproportionate changes in the microbiota of the intestine have systemic immunologic impacts and inflammatory pathways. Dysbiosis has been associated with driving Th17-mediated pathways and has been used to enhance psoriatic inflammation. It is expected that the better we understand this microbial-immune interaction, the better we are able to look at the way a disease progresses and how best to manage it.

In this respect, the given research focused on psoriasis prevalence in Dhaka and Chittagong patients as well as levels of IL-6 and IL-17 in serum and the pattern of the gut microbiota. Ensuing discussion will be grouped into cytokine profiling findings followed by the description of microbial changes captured, then their findings will be combined to discuss their clinical and biological value within the context of the study of psoriasis.

Colony Forming Unit (CFU) counts of *Helicobacter pylori* were done to quantitatively determine its presence and abundance in stool samples and to investigate how it could be associated with psoriasis. *H. pylori* as a gastric pathogen has systemic effects by suppressing host immunity. Chronic *H. pylori* colonization has been associated with changes in the response of cytokines, including the upregulation of pro-inflammatory mediators, which are at the center of the pathogenesis of psoriasis, such as IL-6, IL-17, and TNF- α . We could identify the viable bacterial load of *H. pylori* by enumeration of CFUs on selective media, instead of just by molecular detection of DNA, which can be a result of dead or non-viable bacteria. This quantification of culture-based active colonization is a functional quantification of active colonization that is more pertinent to the interpretation of immune modulation in patients. In addition, by comparing the number of *H. pylori* CFU and the clinical severity scores and cytokine profiles, we can examine how increased levels of colonization relate to increased levels of psoriatic inflammation.

Therefore, CFU counts of *H. pylori* were necessary to create a measurable and biologically significant correlation between colonization of the gut microbes and the systemic immune dysregulation that is seen in psoriasis.

The most noticeable was the trend of CFU/mL displaying a distinction between patients and controls in all countable dilutions. Samples derived from patients had a range of 10⁸-10⁹CFU/mL, whereas control samples tended to group at 10⁵-10⁶ when detectable. This not only shows a

quantitative expansion in regard to the cultivable gut microbiota in patients, but also a qualitative difference in terms of the dilution at which counts could be attained. TNTC outcomes among the patient samples at 10⁻² and 10⁻⁴ dilutions suggests a significant difference in the amount of biomass, whereas the frequency of TFTC outcomes in the controls suggests a decrease in microbial density. Methodological uniformity between the two groups supports the fact that these differences observed are biologically significant and not artifacts of plating or culture conditions. Psoriasis is an immune-mediated chronic inflammatory condition with aberrant IL-17 axis activation and persistence of pro-inflammatory cytokine production including IL-6, IL-17 and TNF- α . There is emerging evidence to support the idea that gut-skin axis has a central role to play when it comes to immune regulation in psoriasis.

Microbiome dysbiosis has also been implicated in systemic immune activation due to increased intestinal permeability, and increased antigen presentation and production of microbial metabolites and pathogen-associated molecular patterns (PAMPs).

The results on CFU/mL increase in psoriasis patients indicate that one symptom of gut dysbiosis is not only the altered composition but also an absolute growth of cultivable microbiome biomass. The higher bacterial load predisposes the possibility of translocation of microbial products including lipopolysaccharides (LPS), peptidoglycans, and bacterial DNA to the circulation. These molecules are extremely strong inducers of the innate immune pathways through the Toll-like receptors and the additional pattern recognition receptors. Such activation may increase the systemic inflammation and maintain the chronic cycle of inflammation peculiar to psoriasis. The culture-derived FLO evidence in this article will therefore resonate with mechanistic postulations regarding the connection between gut dysbiosis and pathologic outcomes using culture-independent. For instance, Yan et al. (2019) found lower alpha-diversity and an abundance of pro-inflammatory taxon *Ruminococcus gnavus* in psoriatic individuals. Fahl more to the point, Fahl et al. (2012) have shown varying relative abundance of Firmicutes and Actinobacteria in patients with psoriasis and in controls. Although these studies did not emphasize the particle-associated microbial biomass, they obliquely indicate that compositional changes in microbial communities can have an effect on microbial biomass.

The current study provides a complementary picture of the findings of quantitative culture-related variations. High-throughput sequencing identifies both culturable and unculturable organisms whereas culture identifies specifically those microbial organisms that grow well under standard laboratory conditions, which may include facultatively pathogens or opportunistic commensals. The increased CFU/mL in patients thus triggers the possibility that psoriasis is correlated with the increase of cultivable, potentially pro-inflammatory skin microbial loads. Similar to IBD, it has been demonstrated that the increased levels of CFU of *E. coli* and other Enterobacteriaceae are associated with mucosal inflammation. The similarity of psoriasis and IBD as both inflammatory diseases, but having systemic immune activation, underscores the validity of the present findings. The fact that higher bacterial burden was demonstrated in psoriasis patients yields several important implications. First, it finds support in the hypothesis that gut dysbiosis is a cause of systemic immune dysregulation in psoriasis. The associated augmented microbial biomass can

contribute to increased antigenic intestinal load, and such predisposition to immune activation. Second, it provides indications that treating psoriatic inflammation with strategies to change the composition of gut microbiota, such as probiotics, prebiotics, dietary interventions, or specific antibiotics, could be useful. The implication of increased microbial load in the gastrointestinal tract in psoriasis is less obvious when the biology of cytokines is considered too. It is known that the components of bacteria like LPS induce IL-6 synthesis by the cells of the innate immune system and this cytokine then activates Th17 cell differentiation and IL-17 release. These cytokines play primary roles in the pathogenesis of psoriasis (psoriatic plaques) and systemic inflammation. High CFU/mL in patient samples, therefore, offers reasonable prior cause of cytokine dysregulation. This is consistent with therapeutic findings: biologic blockade of the IL-17/IL-23 cytokine axis is clinically effective in psoriasis, indirectly confirming the importance of the gut-immune axis in maintaining the disease process. Future research adequate levels of CFU and circulating levels of cytokines in the similar cohort would be indicative of this causative relationship.

In the current study, researchers used ELISA technology to measure interleukin-6 (IL-6) and interleukin-17A (IL-17A) concentration circulating in psoriasis patients and healthy control in the Boster PicoKine Human IL-6 ELISA Kit (EK0410) and Boster PicoKine Human IL-17A ELISA Kit (EK0430). These assays enabled a very sensitive and reproducible systemic inflammation assessment with low detection limits of < 0.3 pg/mL and < 1 pg/mL, respectively, against IL-6 and IL-17A. The immobilized monoclonal capture antibodies and polyclonal biotinylated detection antibody implies the specificity and robustness of the assay to make confident detection of cytokines within the physiological and pathologic limits possible.

The results indicated a high level of IL-6 and IL-17A in psoriasis patients relative to individuals in the control group. This finding correlates with the pathogenic theory of psoriasis as a chronic immune-mediated disease, and the key role of the IL-23/Th17 axis. High IL-6 levels favor the differentiation of naive CD4 + T cells to Th17 subsets that in turn produce IL-17A. IL-17A then onwards influences keratinocytes, and other resident immune cells, triggering additional secretions of IL-6, TNF-alpha, and chemokines completing the cycle of the partially self-sustaining inflammatory loop (Korn et al., 2009; Lowes et al., 2014). The current cytokine patterns identified through ELISA therefore enable a confirmation of the IL-6/IL-17 axis as a central feature in the psoriasis immunopathogenesis.

IL-6 is syndromic and has several roles that include immune stimulator, hematopoiesis, and acute-phase response. The IL-6 has a role to play in psoriasis through the following means:

- Th17 differentiation: IL-6 together with TGF- are key factors that drive Th17 differentiation of naive CD4+ T cells(Hunter & Jones, 2015). This is core to psoriasis since Th17 cells are the majority makers of IL-17A, a cytokine that plays a direct role in hyper-proliferation of keratinocytes, and neutrophil recruitment.
- Systemic Inflammation: A high level of IL-6 is related to the systemic comorbidities that are related to psoriasis such as insulin resistance, obesity, and cardiovascular risk (Mehta

et al., 2018). Persistent, low-level elevation of IL-6 mediates acute-phase changes in hepatocytes, and enhances C-reactive protein (CRP) production and endothelial dysfunction.

- Keratinocyte Activation: IL-6 can directly activate keratinocytes and stimulate the production of antimicrobial peptides and pro-inflammatory mediators of the skin cells further propagating skin inflammation (Scheller et al., 2011).

The observed higher levels of serum IL-6 in patients with psoriasis than the controls in the present study is in agreement with reports in disparately different populations (Tanaka et al., 2014). Notably, the sensitivity of a kit used (picogram-level) ensured a reliable detection of IL-6 even in controls whose baseline levels of IL-6 are typically considerably low (<5 pg/mL). This increased sensitivity was needed in elucidating slight differences between groups, reflecting the usefulness of the PicoKine 7 platform in small sample biomarker studies.

Th17 cells on the other hand are the producers of IL-17A that plays a primary role as a potent effector cytokine in psoriasis. The findings of the ELISA examination highlighted greatly enhanced IL-17A in psoriatic patients validating its role outside the lesional skin microenvironment into its systemic component. IL-17A has been found to play many roles in psoriasis:

- IL-17A also stimulates IL-1, IL-6 and TNF-alpha production and also stimulates chemokines like CXCL1, CXCL8, CCL20 accrual of neutrophils and T cells into psoriatic plaques, which is produced by the stimulated keratinocytes (Lowes et al., 2014).
- Synergistic Action with IL-6: IL-17A and IL-6 act in a feed forward manner with IL-17A inducing IL-6 production by both resident and infiltrating cells and IL-6 supporting Th17 differentiation to sustain IL-17 production (Korn et al., 2009). This synergistic effect manifested itself in our data where patients with a greater IL-6 concentration were also more likely to have a greater IL-17A.
- Pathogenesis of Comorbidities: IL-17A is thought to play a role in the systemic comorbidities as psoriatic arthritis, metabolic syndrome and cardiovascular disease. It is shown that due to playing the role of accelerating atherosclerosis through the promotion of endothelial dysfunction and vascular inflammatory processes, IL-17A is a key causative factor of psoriasis (Miossec & Kolls, 2012).

Detection range of the PicoKine(R) IL-17A ELISA (31.2-2000 pg/mL) was suitable to detect high concentrations in patients, demonstrating the applicability of the assay to translational studies.

Among the main conclusions of the given study is the connection existing between IL-6 and IL-17A. These combine with each other to build a positive loop that lies at the core of psoriasis:

IL-6 activates Th17 differentiation and therefore causes a rise in IL-17A. Upon IL-17A production, the keratinocytes, and immune cells are induced to produce IL-6 thus, consolidating further Th17 polarization. This cytokine feedback loop not only augments inflammation at the local level in the psoriatic plaques but also at the systemic level, making the circulating levels quantified using ELISA high.

In addition, this interaction can be used to explain psoriasis comorbidities. Chronic elevation of IL-6 induces insulin resistance, dyslipidemia, and vascular inflammation (Mehta et al., 2018), whereas IL-17A increased neutrophil resident and endothelial dysfunction (Langley et al., 2014). The overall increase in such cytokines therefore offers mechanistic correlates between skin manifestation and systemic burden.

The ELISA results of serum samples showed that the level of IL-6 and IL-17 in psoriasis patients were significantly increased in comparison with healthy controls. The rising trend shows that these cytokines have a close relationship with disease activity. The IL-6 is a pleiotropic pro-inflammatory mediator that has been reported to induce differentiation in Th17 cells and to stimulate angiogenesis, which in turn contributes to the formation of psoriatic lesions. On the same note, IL-17 which is the primary effector cytokine released by Th17 cells directly incites the hyperproliferation of the keratinocytes and attracts more immune cells to the skin to maintain chronic inflammation.

This association between a high level of cytokines and a high severity score of psoriasis further supports their pathogenicity. Patients presenting with greater Psoriasis Area and Severity Index (PASI) values were consistently characterized by greater concentrations of IL-6 and IL-17, indicating not only that these cytokines are biomarkers of systemic inflammation, but also that they have a role in disease progression.

Consequently, the ELISA results indicate that IL-6 and IL-17 are acutely correlated with the inflammatory condition of psoriasis and are potentially valuable for assessing the severity of the disease and/or using to treat the disorder.

The 16S rRNA gene is highly conserved across prokaryotes, and is interrupted by regions of hypervariability. This distinct genetic framework permits the generation of primers that bind to extremely conserved regions as well as amplify variable regions that may differentiate among taxa of bacteria (Clarridge, 2004). In the *H. pylori*, the primers design homologous regions of its 16S rRNA-genes approximately 522 bp amplicon.

In contrast to genes like *ureA*, *cagA* or *vacA* that in some clinical strains can be absent because of genetic variance, the 16S rRNA locus would always be there and thus can be used as a reliable detection system. In addition, 16S gene amplification can also be used as the internal quality control since it will help to verify the integrity of DNA and the absence of inhibitors. This dual utility species identification/methodological validation explains why the 16S rRNA gene is such a basic tool in molecular diagnostic of bacteria (Lu et al., 1999).

Clear and distinct ~500-600 bp bands observed in the patient samples serves as good molecular confirmation of the presence of *H. pylori* DNA. Critically, the fact that no amplification was seen in the control group confirms the specificity of the assay and decreases the probability of false-positive values because of contamination or cross-reactivity.

The variance between the patient and the control outcomes in amplification indicates the biological importance of *H. pylori* colonization within symptomatic patients. The failure in the control samples to amplify means that it is likely that *H. pylori* prevalence in this population was absent or below the sensitivity of conventional PCR. This observation is in line with epidemiological data that demonstrated the prevalence of *H. pylori* was higher in patients with gastric pathology than in asymptomatic people (Shukla et al., 2017).

Also, the negative control (no template =NTC) should help indicate the robustness of the assay and the lack of reagent contamination. NTCs are important in many molecular experiments so as to separate genuine positive signals with false positives that may occur due to minute quantities of contaminating DNA. The negative NTCs are clean in this study and hence support the integrity of the patient derived data.

The result of this study is in conjunction with most of the previous research that showed the applicability of 16s rRNA PCR on detection of *H. pylori*. In a comparison study using five different PCR methods to detect *H. pylori*, Lie and collaborators (2000) concluded that 16S rRNA-based PCR has high sensitivity and specificity especially when used on a sample of gastric biopsies. In the same regard, Shukla et al. (2017) found reliability of 16S rRNA amplification in *H. pylori*-positive patients with similar band sizes of 500-600 bp, which is closely correlated with the ~500-600 bp discovered in this study.

Interestingly, the amplification of 16S rRNA sequence has actually been reported in asymptomatic persons implying that there may be low level colonization of *H. pylori* without apparent disease. Nevertheless, in the current data pinpointing, no intensification was identified in the control group, which can also be an indication of veritable nonaffectedness or bacterial concentration beneath the limit of PCR assay. This observation concurs with findings of Kusters et al. (2006), who noted that not every infected person by *H. pylori* develops clinical symptoms, yet infected persons who exhibit clinical symptoms have higher bacterial loads compared with those who do not.

Determination of *H. pylori* 16S rRNA sequences in the symptomatic patients was strong, which highlights the clinical usefulness of PCR gene-based diagnostics. Although regarded as a gold standard, culture-based approaches have limitations that include the fastidious growth requirement, limited ability to survive outside of a gastric environment, and sensitivity to transport

conditions. PCR does not have these shortcomings in that it directly identifies the bacterial DNA, even when the organism itself is not viable, and therefore appears to be more sensitive as a diagnosis tool.

Epidemiological-wise, the noted disparity of patients over the controls lends credence to the contribution of *H. pylori* in gastric pathology. The molecular detection of *H. pylori* DNA helps in getting important clues on the prevalence pattern, the strain distribution and also the correlation that exists between the results and their clinical outcome. The results of the present research are in line with the high rates of *H. pylori* recorded in south Asian populations with gastro intestinal issues (Rashid et al., 2011).

Also, the presence of *H. pylori* DNA in the symptomatic patient has implications on the therapeutic responses. Proper diagnosis guides the eradication therapy and PCR-based detection gives the clinicians an insight on whether it is a true infection or symptomatic overlap with some non-*H. pylori* diarrheal conditions.

Identification of *H. pylori* DNA in patient samples and not in controls, adds molecular evidence that confirms the pathogenic role of this organism in gastric diseases. Morbidity and mortality Due to the chronicity of the colonization of *H. pylori*, persistent inflammation, damaged epithelial cells, and cytokine exposure have been implicated in gastritis, peptic ulcers, and cancer of the stomach. Bacterial DNA PCR confirmation is thus not only diagnostic but also pathophysiologically significant considering that it shows a direct association between infection and the state of the disease.

In brief, PCR reveals the presence of *H. pylori* when a ~500-600 bp fragment is amplified that corresponds to the 16S rRNA gene in the symptomatic patients, which qualifies 16S rRNA PCR to be a diagnostic test. Negative PCR in the control and no-template reactions show specificity and strength of the assays. Such results are in agreement with previous work and show the utility of molecular diagnostics compared to culture-based methods in detection of this fastidious organism. Limitations, including the potential of cross reactivity and false negatives, still prevail, but results also indicate the utility of 16S rRNA PCR in both clinical and epidemiological work. The study therefore advances efforts to ensure better diagnosis, making of therapeutic options and increasing the knowledge of *H. pylori* pathogenesis. Although the study has successfully offered significant information about the prevalence of psoriasis in the two biggest cities in Bangladesh, and the related gut microbiota pattern, the associated cytokine activities, there still exist few limitations that should be considered in order to translate the findings. An understanding of these limitations is vital to admittedly situating the results in a larger research context as well as where future research in this field should go.

14.4 Limitations:

Among the main limitations associated with the study sample size and geographical scope of the study should be mentioned. The sample of the research was formed of patients of Dhaka and Chittagong cities, two of the largest cities of Bangladesh, which might not be fully representative of the situation in the country. The country of Bangladesh is typified by extensive socio-economic,

cultural, dietary, and environmental variability in the different regions of the country. It is therefore possible that the study population fails to capture the full spectrum of heterogeneity of psoriasis expression, cytokines profile, and gut microbial make-up that may be present in rural communities, or in communities with varying diet or lifestyle habits. It would be more convenient to implement a larger multiplex study that covers a bigger expanse of area and is more representative of the Bangladesh population and can firmly lay hands on the generalizability of the conclusions.

The other critical limitation is that the research design is cross-sectional. Cross-sectional studies are applicable in determining the relationship between different variables at a given time, but could not be used to make conclusions on causality. Although the study was able to find and compare the elevated levels of cytokines including IL-6 and IL-17, and point out the possible connection between psoriasis and changes in the gut microbiota, it cannot be ascertained whether these immunological and microbial disturbances are both causative or resultant factors of the development of psoriasis or merely coincident and correlated phenomena. The stronger evidence of cause and effect could be given by longitudinal or interventional studies that track the life of patients over time or the effects of specific treatments.

The cytokine profiling used in the current study was also somewhat narrow. The selection of IL-6 and IL-17 was valid since these cytokines have been reported to be key regulators in psoriasis pathogenesis and chronic inflammation. Nevertheless, psoriasis is a multifactorial disease, which is the result of a complex involvement of immune mediators, including TNF-alpha, IL-23, IFN-gamma, or IL-22, among others. The limitation of analysis to the two cytokines does not give a complete picture on the immune dysregulation that results in psoriasis. A fuller set of cytokines, or a more high-throughput method (e.g., multiplex assay) would provide a more extensively detailed picture of the immune landscape of psoriatic patients. Another weakness is that screening and controlling clinical variables could be a problem given that the study participants will likely have several such variables interacting. Patients with psoriasis commonly have comorbidities (obesity, metabolic syndrome, cardiovascular disease, and diabetes) that are also accompanied by systemic inflammation and gut microbiota changes. Moreover, since ages, disease severity, lifestyle, and history of immunosuppressive drugs or antibiotics are all circulating factors, they could have affected the cytokine levels and the microbial composition. Although attempts were made to decrease bias, the confounders could not be fully controlled. Therefore, parts of the observed variations in cytokine expression or intestinal microbiota may be the effects of underlying conditions and less related to psoriasis itself. Lastly, a lack of longitudinal study follow-up data is limiting in ability to detect changes in cytokines and gut microbiota composition over time. Psoriasis is a chronic relapsing condition, and immune and microbial parameters are also likely to vary with activity, treatment regimen, and various lifestyle changes. These biological markers are not assessed over time and repeat sampling or follow-up data could not be used to determine trends or consistency in these biological markers. Such longitudinal data would be useful especially in how cytokines response and microbiota profile evolves as flare-ups and remission or during treatment.

15. Conclusion:

The study examines the incidence of psoriasis in Dhaka, Bangladesh, with focus on cytokine analysis and the content of intestinal microbiota. Psoriasis is an immune-mediated chronic inflammatory condition that is increasingly characterized by disproportionate cytokine signaling networks and the microbiome. A cross-sectional study involvement was used that included patient cohorts with a diagnosis of psoriasis and healthy controls. Serum levels of two important cytokines in the Th17-driven inflammatory pathway were determined using quantitative ELISA assays to measure the serum interleukin-6 (IL-6) and interleukin-17 (IL-17) concentrations. The validation in the results revealed that IL-6 and IL-17 were significantly high in psoriatic subjects indicating their role in the pathogenesis of psoriasis as far as their involvement in keratinocyte growth, immune stimulation, and associated systemic diseases.

Comparative biome-wide analysis of fecal specimens implied that cases have very clear compositional changes in intestinal microbiota compared to controls and may have a functional contribution of dysbiosis in shaping immune responses in the system. These results correlate with the recent data on the gut-skin axis and its role in the existence of chronic inflammation. In spite of these limitations of sample size, cross-sectional design, and a limited number of microbial characterization tools, the study offers new findings about the cytokine-microbiome interaction in Bangladesh patients with psoriasis.

Together, the results support the clinical significance of interplay between immune and microbiota in psoriasis and provide possible diagnostic and therapeutic options of precision medicine interventions. A key point to this thesis is the reciprocity that exists between the microbiota of the gut and psoriasis through cytokine imbalance. The current ELISA results provide an overwhelming argument to bridge these areas.

Dysbiosis of the gut is also described in psoriasis (Yan et al., 2020). Those changes contribute to systemic inflammation in a variety of ways-

- Bacterial Products: Immunologically, dysbiosis distorts higher production of short-chain fatty acids (SCFA), which decreases the anti-inflammatory cues to innate immune systems, promoting Th17 proliferation (Kamada et al., 2013).
- Transport of Microbial Products: Microbial products (lipopolysaccharide and others) are transported across the intestinal lining because of impaired intestinal permeability, and these products stimulate the production of IL-6 and IL-1-beta via TLRs.
- Skewing of Th17: Commensal imbalance induces dendritic cells to secrete IL-6 and IL-23 on Th17 cell polarization and IL-17A production, thus showing a connection between gut-derived factors and psoriatic inflammation (Yan et al., 2020).

In our cohort of Dhaka and Chittagong where gastrointestinal infections and *H. pylori* prevalence are very high, the role of microbial factor might be still increased. Chronic exposure to gut-derived immune stimuli may drive the increase in IL-6 and IL-17A indicated by ELISA, which represent a more general gut-skin axis to psoriatic disease burden in South Asian communities.

An increase in both IL-6 and IL-17A at the same time confirms that their success would measure disease activity in psoriasis. In addition to diagnosis, cytokine measurement has the promise of helping in stratification and monitoring of therapeutic effect on patients.

Therapeutically, the relative purity of its chronic pain patients is relatively pure.

- Identifying another IL-6 antagonist: Tocilizumab, an IL-6 receptor antagonist, has been successful in treatment of rheumatoid arthritis, and its application in psoriasis should be investigated (Hunter & Jones, 2015).
- IL-17 Block: IL-17A biologics such as secukinumab and ixekizumab have shown very high efficacy and safety in the management of psoriasis with rapid clearance of the skin (Langley et al., 2014).
- Combinatorial Possibility: Since the relationship between IL-6 and IL-17A is feed-forward, it is possible duo blockade therapies could have greater benefit to patients with severe and systemic disease manifestation.

The most important strength of this study is that highly sensitive ELISA kits with low detection limits were used, which made it possible to robustly quantify cytokines even in healthy people. The standardized protocols reduced technical variability and intra/inter-assay precision was within manufactures specifications.

The findings of ELISA-based quantification of IL-6 and IL-17A in psoriasis patients in Dhaka and Chittagong indicated significant systemic increase in IL-6 and IL-17A, which supports the IL-6/IL-17 axis in psoriatic pathogenesis. These cytokines work in a synergistic pathway to increase both local and systemic inflammation and associate cutaneous disease with comorbidities. In addition, the results favor the idea that a specific change in the composition of the gut microbiota is related to cytokine abnormality, which provides a mechanistic route between environmental effects, systemic inflammation, and the severity of psoriatic disease in South Asian populations. Multidimensional insights into mechanisms of psoriatic disease may be afforded by future studies combining ELISA-based cytokine profiling and microbiome sequencing with genetic analyses.

Moreover, the survey was conducted on the incidence of psoriasis in two major urban centers in Bangladesh, Dhaka and Chittagong, with focus on various factors such as age, gender, severity of condition, thinning/ bleeding from affected area, joint pain etc. Among them, it was observed that the least affected age group was under 20 years of age in both cities, and most affected patients were male. The vast majority of patients reported having symptoms for more than two years. This

finding suggests that a significant number of individuals are living with a long-standing version of the disease. The data on severity showed a mixed distribution. Mild cases were the most common in both cities. Moderate cases were also widespread, and severe cases, while less frequent, still represented a significant portion of the patient population. This indicates that while many individuals have manageable symptoms, a considerable number are dealing with severe manifestations of the disease. It was observed that both "thinning/bleeding" and "no thinning/bleeding" were the most frequently observed categories, while "only bleeding" and "thinning only" were uncommon across all groups. However, the most important finding here is that the vast majority of patients reported no joint pain. This suggests that while joint pain can be a symptom, it is not universally present in this patient population. The study only included patients from Dhaka and Chittagong, which may not represent the diverse socio-economic, cultural, dietary, and environmental variations across Bangladesh. For more accurate results, large sample sizes from various areas are needed.

15.1 Awareness:

The importance of awareness of psoriasis can not be ignored, thereby making it an important but ignored part of psoriasis management. Although a prevalent chronic inflammatory illness, there are misconceptions and associated stigma concerning psoriasis in low and middle-income countries e.g. Bangladesh. Individuals still maintain the opinion that psoriasis is actually contagious and this promotes isolation and discrimination against the affected patients. Besides influencing the mental health and quality of life of patients, this inaccurate knowledge also slows down the desire to seek health and decreases adherence to treatments.

What is more, among healthcare professionals awareness is not always sufficient. This could lead to the non-recognition by the following general practitioners and primary health care workers of psoriasis with its onset or unequal treatment of its systemic implications like comorbidities to which it causes cardiovascular diseases, diabetes, and even inflammatory bowel diseases. This disparity is caused by a lack of public health crusades and limited incorporation of skin care training into general medicine.

Raising the level of awareness on community as well as professional levels is consequently necessary. The reduction of stigma and the subsequent achievement of improved outcomes of care can be reached through patient education programs, awareness campaigns through mass media, and training workshops among the healthcare providers. This enhanced awareness could lead to the development of research interest and enhanced health policies to deal with psoriasis as a health issue in the community.

16. Reference:

1. Nograles, K. E., et al. (2008). The IL-17 Family of Cytokines in Psoriasis: IL-17A and Beyond. *Journal of Investigative Dermatology*, 128(9), 2167–2174. [URL: <https://pmc.ncbi.nlm.nih.gov/articles/PMC6088173/>]
2. Kawai, T., & Akira, S. (2010). Toll-like Receptor Signaling and Its Role in Cell-Mediated Immunity. *Immunity*, 32(4), 437–450. [URL: <https://pmc.ncbi.nlm.nih.gov/articles/PMC8927970/>]
3. Mu, Q., et al. (2017). Leaky Gut as a Danger Signal for Autoimmune Diseases. *Frontiers in Immunology*, 8, 598. [URL: <https://www.frontiersin.org/journals/immunology/articles/10.3389/fimmu.2017.00598>]
4. Groeger, D., et al. (2013). Effect of Probiotics on Central Nervous System Functions in Animals and Humans: A Systematic Review. *Journal of Neurogastroenterology and Motility*, 19(1), 5–14. [URL: <https://www.jnmjournal.org/view.html?uid=1188&vmd=Full>]
5. Gareau, M. G., et al. (2011). The gut microbiota, HPA axis, and brain in adolescent-onset depression: Probiotics as a novel treatment. *Translational Psychiatry*, 1(1), 1–13. [URL: <https://pmc.ncbi.nlm.nih.gov/articles/PMC9758412/>]
6. A recent study found a high prevalence of *H. pylori* infection among patients with psoriasis, suggesting a possible association. [URL: <https://www.apims.net/apims/article/download/1056/1001/5344>]
7. A meta-analysis also shows an increased risk of *H. pylori* infection in psoriasis patients. [URL: <https://pmc.ncbi.nlm.nih.gov/articles/PMC5996629/>]
8. Tocilizumab is an IL-6 receptor antagonist. Search results indicate it is used for rheumatoid arthritis and other inflammatory conditions, but there is little information about its specific use for psoriasis. [URL: <https://www.pharmacytimes.com/view/study-high-similarity-efficacy-between-tocilizumab-biosimilar-and-reference-product-in-ra>]
9. These are anti-IL-17A drugs that are highly effective in treating moderate-to-severe plaque psoriasis by neutralizing IL-17A. [URL: <https://www.actasdermo.org/en-clinical-efficacy-safety-ixekizumab-for-articulo-S0001731016303544>]
10. Lowes, M. A., Suarez-Farinas, M., & Krueger, J. G. (2014). Immunology of psoriasis. *Annual Review of Immunology*, 32, 227–255. <https://doi.org/10.1146/annurev-immunol-032713-120225>
11. Nestle, F. O., Kaplan, D. H., & Barker, J. (2009). Psoriasis. *New England Journal of Medicine*, 361(5), 496–509. <https://doi.org/10.1056/NEJMra0804595>.
12. Prinz, I. (2019). Interleukin-17 cytokines: Effectors and targets in psoriasis. *Journal of Experimental Medicine*, 216(2), 201–214. <https://doi.org/10.1084/jem.20191397>

13. Di Cesare, A., Di Meglio, P., & Nestle, F. O. (2009). The IL-23/Th17 axis in the immunopathogenesis of psoriasis. *Journal of Investigative Dermatology*, 129(6), 1339–1350. <https://doi.org/10.1038/jid.2009.59>
14. Salem, I., Ramser, A., Isham, N., & Ghannoum, M. A. (2018). The gut microbiome as a major regulator of the gut–skin axis. *Frontiers in Microbiology*, 9, 1459. <https://doi.org/10.3389/fmicb.2018.01459>
15. Campanati, A., Marani, A., Martina, E., Diotallevi, F., Radi, G., & Offidani, A. (2021). Psoriasis as an Immune-Mediated and Inflammatory Systemic Disease: From Pathophysiology to Novel Therapeutic Approaches. *Biomedicines*, 9(11), 1511. <https://doi.org/10.3390/biomedicines9111511>
16. Szántó, M., Dózsa, A., Antal, D., Szabó, K., Kemény, L., & Bai, P. (2019). Targeting the gut–skin axis—Probiotics as new tools for skin disorder management? *Experimental Dermatology*, 28(11), 1210–1218. <https://doi.org/10.1111/exd.14016>
17. Liatsos, C., Papaefthymiou, A., Kyriakos, N., Galanopoulos, M., Doulberis, M., Giakoumis, M., Petridou, E., Mavrogiannis, C., Rokkas, T., & Kountouras, J. (2022). *Helicobacter pylori*, gastric microbiota and gastric cancer relationship: Unrolling the tangle. *World journal of gastrointestinal oncology*, 14(5), 959–972. <https://doi.org/10.4251/wjgo.v14.i5.959>
18. Yan, Y., Deng, W., Shi, C., Xie, J., & Sui, D. (2024). *Helicobacter pylori* infection and its impact on psoriasis: a systematic review and meta-analysis. *Frontiers in medicine*, 11, 1500670. <https://doi.org/10.3389/fmed.2024.1500670>
19. Saggini, A., Chimenti, S., & Chiricozzi, A. (2014). IL-6 as a druggable target in psoriasis: Focus on pustular variants. *Journal of Immunology Research*, 2014, 964069. <https://doi.org/10.1155/2014/964069>
20. Anyfanti, P., Margouta, A., Goulas, K., Gavriilaki, M., Lazaridou, E., Patsatsi, A., & Gkaliagkousi, E. (2022). Endothelial Dysfunction in Psoriasis: An Updated Review. *Frontiers in medicine*, 9, 864185. <https://doi.org/10.3389/fmed.2022.864185>
21. Oliveira, A., Augustin, S., Benlloch, S., Ampuero, J., Suárez-Pérez, J. A., Armesto, S., Vilarrasa, E., Belinchón-Romero, I., Herranz, P., Crespo, J., Guimerá, F., Gómez-Labrador, L., Martín, V., & Carrascosa, J. M. (2023). The Essential Role of IL-17 as the Pathogenetic Link between Psoriasis and Metabolic-Associated Fatty Liver Disease. *Life (Basel, Switzerland)*, 13(2), 419. <https://doi.org/10.3390/life13020419>
22. Planas, A., Simó-Servat, O., Hernández, C., & Simó, R. (2022). Advanced Glycations End Products in the Skin as Biomarkers of Cardiovascular Risk in Type 2 Diabetes. *International Journal of Molecular Sciences*, 23(11), 6234. <https://doi.org/10.3390/ijms23116234>
23. Schwarz, C. W., et al. (2020). Effect of anti-interleukin-17 treatment on hemoglobin A1c levels in patients with psoriasis. *Acta Dermato-Venereologica*, 100(8), adv3620. <https://doi.org/10.2340/00015555-3620>

24. Parisi, S., Ditto, M. C., Ghellere, F., Panaro, S., Piccione, F., Borrelli, R., & Fusaro, E. (2025). Update on tocilizumab in rheumatoid arthritis: a narrative review. *Frontiers in immunology*, 16, 1470488. <https://doi.org/10.3389/fimmu.2025.1470488>
25. Ridker, P. M. (2021). Interleukin-6 signaling and anti-interleukin-6 therapeutics in cardiovascular disease. *Circulation Research*, 129(4), 437–451. <https://doi.org/10.1161/CIRCRESAHA.121.319077>
26. Zhou, X., Chen, Y., Cui, L., Shi, Y., & Guo, C. (2022). Advances in the pathogenesis of psoriasis: from keratinocyte perspective. *Cell death & disease*, 13(1), 81. <https://doi.org/10.1038/s41419-022-04523-3>
27. Pirowska, M., Podolec, K., Lipko-Godlewska, S., Sułowicz, J., Brzewski, P., Obtulowicz, A., Pastuszcak, M., & Wojas-Pelc, A. (2019). Level of inflammatory cytokines tumour necrosis factor α , interleukins 12, 23 and 17 in patients with psoriasis in the context of metabolic syndrome. *Postepy dermatologii i alergologii*, 36(1), 70–75. <https://doi.org/10.5114/ada.2018.73136>
28. Fitch, E., Harper, E., Skorcheva, I., Kurtz, S. E., & Blauvelt, A. (2007). Pathophysiology of psoriasis: recent advances on IL-23 and Th17 cytokines. *Current rheumatology reports*, 9(6), 461–467. <https://doi.org/10.1007/s11926-007-0075-1>
29. Pirowska, M., Podolec, K., Lipko-Godlewska, S., Sułowicz, J., Brzewski, P., Obtulowicz, A., Pastuszcak, M., & Wojas-Pelc, A. (2019). Level of inflammatory cytokines tumour necrosis factor α , interleukins 12, 23 and 17 in patients with psoriasis in the context of metabolic syndrome. *Postepy dermatologii i alergologii*, 36(1), 70–75. <https://doi.org/10.5114/ada.2018.73136>
30. Schön M. P. (2019). Adaptive and Innate Immunity in Psoriasis and Other Inflammatory Disorders. *Frontiers in immunology*, 10, 1764. <https://doi.org/10.3389/fimmu.2019.01764>
31. Rendon, A., & Schäkel, K. (2019). Psoriasis Pathogenesis and Treatment. *International journal of molecular sciences*, 20(6), 1475. <https://doi.org/10.3390/ijms20061475>
32. Qu, N., Xu, M., Mizoguchi, I., Furusawa, J., Kaneko, K., Watanabe, K., Mizuguchi, J., Itoh, M., Kawakami, Y., & Yoshimoto, T. (2013). Pivotal roles of T-helper 17-related cytokines, IL-17, IL-22, and IL-23, in inflammatory diseases. *Clinical & developmental immunology*, 2013, 968549. <https://doi.org/10.1155/2013/968549>
33. Korn, T., Bettelli, E., Oukka, M., & Kuchroo, V. K. (2009). IL-17 and Th17 Cells. *Annual review of immunology*, 27, 485–517. <https://doi.org/10.1146/annurev.immunol.021908.132710>
34. Kamata, M., & Tada, Y. (2023). Crosstalk: keratinocytes and immune cells in psoriasis. *Frontiers in immunology*, 14, 1286344. <https://doi.org/10.3389/fimmu.2023.1286344>
35. Piipponen, M., Li, D., & Landén, N. X. (2020). The Immune Functions of Keratinocytes in Skin Wound Healing. *International journal of molecular sciences*, 21(22), 8790. <https://doi.org/10.3390/ijms21228790>

36. McGeachy, M. J., Cua, D. J., & Gaffen, S. L. (2019). The IL-17 Family of Cytokines in Health and Disease. *Immunity*, 50(4), 892–906.
<https://doi.org/10.1016/j.immuni.2019.03.021>
37. Mosca, M., Hong, J., Haderler, E., Hakimi, M., Liao, W., & Bhutani, T. (2021). The Role of IL-17 Cytokines in Psoriasis. *ImmunoTargets and therapy*, 10, 409–418.
<https://doi.org/10.2147/ITT.S240891>
38. Tanaka, T., Narazaki, M., & Kishimoto, T. (2014). IL-6 in inflammation, immunity, and disease. *Cold Spring Harbor perspectives in biology*, 6(10), a016295. <https://doi.org/10.1101/cshperspect.a016295>
39. Castells-Rodellas, A., Castell, J. V., Ramirez-Bosca, A., Nicolas, J. F., Valcuende-Cavero, F., & Thivolet, J. (1992). Interleukin-6 in normal skin and psoriasis. *Acta Dermato-Venereologica*, 72(3), 165–168. <https://doi.org/10.2340/0001555572165168>
40. Pietrzak, A., Chabros, P., Grywalska, E., Pietrzak, D., Kandzierski, G., Wawrzycki, B., Roliński, J., Gawęda, K., & Krasowska, D. (2020). Serum concentration of interleukin 6 is related to inflammation and dyslipidemia in patients with psoriasis. *Advances in Dermatology and Allergology/Postępy Dermatologii i Alergologii*, 37(1), 41–45.
<https://doi.org/10.5114/ada.2018.78028>
- Andújar, I., Esplugues, J. V., & García-Martínez, P. (2022). Looking beyond the Skin: Pathophysiology of Cardiovascular Comorbidity in Psoriasis and the Protective Role of Biologics. *Pharmaceuticals (Basel, Switzerland)*, 15(9), 1101.
<https://doi.org/10.3390/ph15091101>
41. Hou, W., Jin, Y. H., Kang, H. S., & Kim, B. S. (2014). Interleukin-6 (IL-6) and IL-17 synergistically promote viral persistence by inhibiting cellular apoptosis and cytotoxic T cell function. *Journal of virology*, 88(15), 8479–8489. <https://doi.org/10.1128/JVI.00724-14>
42. Kolbinger, F., Di Padova, F., Deodhar, A., Hawkes, J. E., Huppertz, C., Kuiper, T., McInnes, I. B., Ritchlin, C. T., Rosmarin, D., Schett, G., Carballido, J. M., Häusermann, P., Calonder, C., Vogel, B., Rondeau, J. M., & Bruin, G. (2022). Secukinumab for the treatment of psoriasis, psoriatic arthritis, and axial spondyloarthritis: Physical and pharmacological properties underlie the observed clinical efficacy and safety. *Pharmacology & Therapeutics*, 229, Article 107925.
<https://doi.org/10.1016/j.pharmthera.2021.107925>
43. Paniagua J. A. (2016). Nutrition, insulin resistance and dysfunctional adipose tissue determine the different components of metabolic syndrome. *World journal of diabetes*, 7(19), 483–514. <https://doi.org/10.4239/wjd.v7.i19.483>

44. Hołdrowicz, A., & Żebrowska, A. (2025). Molecular Link Between Psoriasis and Depression—Update on Pathophysiology. *International Journal of Molecular Sciences*, 26(6), 2467. <https://doi.org/10.3390/ijms26062467>
45. Jimenez-Sanchez, M., Celiberto, L. S., Yang, H., Sham, H. P., & Vallance, B. A. (2025). The gut-skin axis: a bi-directional, microbiota-driven relationship with therapeutic potential. *Gut microbes*, 17(1), 2473524. <https://doi.org/10.1080/19490976.2025.2473524>
46. Memariani, M., & Memariani, H. (2025). New horizons in the treatment of psoriasis: Modulation of gut microbiome. *Heliyon*, 11(1), e41672. <https://doi.org/10.1016/j.heliyon.2025.e41672>
47. Orzan, O. A., Tutunaru, C. V., & Ianoși, S. L. (2025). Understanding the Intricate Pathophysiology of Psoriasis and Related Skin Disorders. *International Journal of Molecular Sciences*, 26(2), 749. <https://doi.org/10.3390/ijms26020749>
48. Zhang, D., Jian, YP., Zhang, YN. et al. Short-chain fatty acids in diseases. *Cell Commun Signal* 21, 212 (2023). <https://doi.org/10.1186/s12964-023-01219-9>
49. Kespohl, M., Vachharajani, N., Luu, M., Harb, H., Pautz, S., Wolff, S., Sillner, N., Walker, A., Schmitt-Kopplin, P., Boettger, T., Renz, H., Offermanns, S., Steinhoff, U., & Visekruna, A. (2017). The Microbial Metabolite Butyrate Induces Expression of Th1-Associated Factors in CD4+ T Cells. *Frontiers in immunology*, 8, 1036. <https://doi.org/10.3389/fimmu.2017.01036>
50. Chen, L., Sun, M., Wu, W., Yang, W., Huang, X., Xiao, Y., Ma, C., Xu, L., Yao, S., Liu, Z., & Cong, Y. (2019). Microbiota Metabolite Butyrate Differentially Regulates Th1 and Th17 Cells' Differentiation and Function in Induction of Colitis. *Inflammatory bowel diseases*, 25(9), 1450–1461. <https://doi.org/10.1093/ibd/izz046>
51. Buhaș, M. C., Gavrilăș, L. I., Candrea, R., Căținean, A., Mocan, A., Miere, D., & Tătaru, A. (2022). Gut Microbiota in Psoriasis. *Nutrients*, 14(14), 2970. <https://doi.org/10.3390/nu14142970>
52. Hunter, C. A., & Jones, S. A. (2015). *IL-6 as a keystone cytokine in health and disease*. *Nature Immunology*, 16(5), 448–457. <https://doi.org/10.1038/ni.3153>
53. Kamada, N., Seo, S. U., Chen, G. Y., & Núñez, G. (2013). *Role of the gut microbiota in immunity and inflammatory disease*. *Nature Reviews Immunology*, 13(5), 321–335. <https://doi.org/10.1038/nri3430>
54. Leng, S. X., McElhaney, J. E., Walston, J. D., Xie, D., Fedarko, N. S., & Kuchel, G. A. (2008). *ELISA and multiplex technologies for cytokine measurement in inflammation and aging research*. *The Journals of Gerontology: Series A, Biological Sciences and Medical Sciences*, 63(8), 879–884. <https://doi.org/10.1093/gerona/63.8.879>
55. Lowes, M. A., Suárez-Fariñas, M., & Krueger, J. G. (2014). *Immunology of psoriasis*. *Annual Review of Immunology*, 32, 227–255. <https://doi.org/10.1146/annurev-immunol-032713-120225>

56. Scheller, J., Chalaris, A., Schmidt-Arras, D., & Rose-John, S. (2011). *The pro- and anti-inflammatory properties of the cytokine interleukin-6*. *Biochimica et Biophysica Acta (BBA) – Molecular Cell Research*, 1813(5), 878–888.
<https://doi.org/10.1016/j.bbamcr.2011.01.034>
57. Schett, G. (2018). *Physiological effects of modulating the interleukin-6 axis*. *Rheumatology*, 57(Suppl 2), ii43–ii50. <https://doi.org/jif>
58. Tanaka, T., Narazaki, M., & Kishimoto, T. (2014). *IL-6 in inflammation, immunity, and disease*. *Cold Spring Harbor Perspectives in Biology*, 6(10), a016295.
<https://doi.org/10.1101/cshperspect.a016295>
59. Korn, T., Bettelli, E., Oukka, M., & Kuchroo, V. K. (2009). *IL-17 and Th17 cells*. *Annual Review of Immunology*, 27, 485–517.
<https://doi.org/10.1146/annurev.immunol.021908.132710>
60. Langley, R. G., Elewski, B. E., Lebwohl, M., Reich, K., Griffiths, C. E. M., Papp, K., Puig, L., Nakagawa, H., Spelman, L., Sigurgeirsson, B., Rivas, E., Tsai, T. F., Wasel, N., Tying, S., Salko, T., Hampele, I., Notter, M., Karpov, A., Helou, S., & Papavassilis, C. (2014). *Secukinumab in plaque psoriasis—results of two phase 3 trials*. *New England Journal of Medicine*, 371(4), 326–338. <https://doi.org/10.1056/NEJMoa1314258>
61. Lowes, M. A., Suárez-Fariñas, M., & Krueger, J. G. (2014). *Immunology of psoriasis*. *Annual Review of Immunology*, 32, 227–255. <https://doi.org/10.1056/NEJMoa1314258>
62. Mehta, N. N., Yu, Y., Pinnelas, R., et al. (2011). *Attributable risk estimate of severe psoriasis on major cardiovascular events*. *American Journal of Medicine*, 124(8), 775.e1–e6. <https://doi.org/jif>
63. Miossec, P., & Kolls, J. K. (2012). *Targeting IL-17 and Th17 cells in chronic inflammation*. *Nature Reviews Drug Discovery*, 11(10), 763–776.
<https://doi.org/10.1038/nrd3794>
64. Yan, D., Issa, N., Afifi, L., Jeon, C., Chang, H. W., & Liao, W. (2017). *The role of the skin and gut microbiome in psoriatic disease*. *Current Dermatology Reports*,
<https://doi.org/insert>
65. Clarridge, J. E. (2004). *Impact of 16S rRNA gene sequence analysis for identification of bacteria on clinical microbiology and infectious diseases*. *Clinical Microbiology Reviews*, 17(4), 840–862. <https://doi.org/10.1128/CMR.17.4.840-862.2004>
66. Lu, J. J., Perng, C. L., Shyu, R. Y., Chen, C. H., Lou, Q., & Chong, S. K. F. (1999). *Comparison of five PCR methods for detection of Helicobacter pylori DNA in gastric tissues*. *Journal of Clinical Microbiology*, 37(3), 772–774.
<https://doi.org/10.1128/JCM.37.3.772-774.1999>

