

**Low Expression of the IL-23/Th 17 Pathway in Atopic Dermatitis Compared to Psoriasis
Paper Summary Outline**

Brad Baird

Introduction

Atopic dermatitis and psoriasis, inflammatory skin diseases, are both directly connected to barrier alterations in the skin. These barrier alterations in both diseases are directly connected to "underlying immune activity" as well as environmental factors. Both are considered polar diseases on the T-helper 1 and T-helper 2 cells spectrum, along with Interleukin-17. All three are considered helpful in regulating the immune functions of skin cells. In addition T-helper 17 cells are also connected to the regulation of immune responses in skin cells. \

Th 17 cells may also be connected to inflammation in a number of other diseases, including arthritis, multiple sclerosis, bowel disease, as well as airway inflammation. This connection does encourage discussion about the possible contributions that Th17 cells could be making to inflammatory skin disorders.

Psoriasis is known to be directly influenced by both Th17 cells and Il-23 cells, which provide an immune response and help to heal lesions caused by the disease. On the other hand, little is known about how it affects the immune response in Atopic Dermatitis. The experiment described examines how the Th-17/Il-23 pathway finds a low expression in Atopic Dermatitis and could therefore explain why the lesions in Atopic Dermatitis become infected and have difficulty responding at the immune system level.

Materials and Methods

The study began by collecting skin samples from those suffering from atopic dermatitis, psoriasis, and from those without either disease. Cultures were made of the keratinocytes from these samples by treating the cells with human Il-17, Il-22, and IFN- γ for approximately twenty-four hours. In addition, they prepared samples. In addition, they used immunohistochemistry analysis and immunofluorescence analysis to detect antigens in the cells.

Samples were also prepared and underwent a real-time polymerase chain reaction and gene chip analysis, in addition a DNA microarray analysis was also used to help analyze the cells. Finally, an analysis of the real time-polymerase chain reaction process was provided, including a discussion of the DNA primers that catalyzed the amplification of the target genes.

The study also included analysis with bioinformatics software of the hierarchical clustering of the genes expressed through heatmaps, in an attempt to distinguish their interrelationships. The data was also compared using a 2-tailed Student's *t* test to ensure that no errors could have occurred in the analysis. In addition, gene descriptions were collected of the expressed genes using the LocusLink database.

Results

As was predicted, the study found significantly higher expressions of the Th17/IL23 pathway in psoriasis as opposed to those found in atopic dermatitis, particularly in IL 17 and IL 23. In addition, the microarray analysis enabled the study to look at the biological consequences of the expression by studying protein expression.

In this case, the proteins were underexpressed in atopic dermatitis as opposed to psoriasis, indicating that this pathway is directly connected to immune response. The study also found a lack of specific dendritic cells, which may account for the lack of activation of the Th17/IL23 pathway in atopic dermatitis.

Discussion

The traditional Th1/Th2 model of immune response has been challenged with the discovery of Th17 cells. It is unclear how Th17 affects cell response to disease and therefore this study was undertaken to compare atopic dermatitis and psoriasis. The data gathered by the model does indicate a low expression of the Th17/IL 23 pathway in atopic dermatitis as opposed to psoriasis, and clearly demonstrates that the IL 17 cells are clearly responsible for the activation of the immune response by the epithelial cells.

There is also "increasing" evidence that the anti-microbial response is directly connected to the immune response of the epithelial cells. These cells act as an internal anti-biotic and prevent infection in psoriasis, whereas the low expression in atopic dermatitis does not prevent infection.