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Commentary

# Site-Specific Analysis in Lupus Erythematosus

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## DESCRIPTION

Prior research had found T cells, B cells, and macrophages in the inflammatory infiltrate and up-regulated their protein products in Discoid Lupus Erythematosus (DLE) skin, but it lacked comprehensive investigations to pinpoint where exactly they were in the skin. As a result, The evaluated expression of a few T cell, B cell, and macrophage markers in five DLE, psoriasis, and healthy skin areas. In biopsies of 23 DLE lesional skin, 11 psoriasis lesional skin, and 5 normal skin, immunostainings for CD3, CD4, CD8, CD20, CD68, CXCR3, CXCL10, and TIA-1 were carried out. The presence of each marker in the Epidermis, Dermato Epidermal Junction (DEJ), Perivascular Area, Periadnexal Area, and Deep Dermis was evaluated by three independent observers using a graded scale.

In comparison to psoriasis and healthy skin, DLE lesional skin had a greater abundance of $CD3^+$, $CD8^+$, and $CD68^+$ cells near the DEJ and $CD20^+$ and $CD68^+$ cells in the periadnexal region. Compared to psoriasis lesional skin, the periadnexal regions of DLE lesional skin had higher levels of CXCR3, CXCL10, and TIA-1. T cells, B cells, macrophages, and their protein byproducts (CXCR3, CXCL10, and TIA-1) may gather in the DEJ and periadnexal region of DLE lesional skin in a coordinated, sophisticated process that may contribute to the pathophysiology of DLE.

The face, scalp, and neck are the most often affected areas by Discoid Lupus Erythematosus (DLE), which is the most prevalent form of cutaneous lupus erythematosus. DLE is characterized by distinct, scaly, erythematous papules and macules with peripheral hyperpigmentation. When there is ongoing inflammation present, DLE lesions usually enlarge before healing with scarring, atrophy, and hyper or hypopigmentation.

Histopathologically, perivascular and periadnexal regions, as well as the Dermo Epidermal Junction (DEJ), become inflammatory infiltrates with a predominance of T cells, B cells, and macrophages in DLE, culminating in vacuolar interface dermatitis. Numerous studies have shown that T cells, which include $CD4^+$ helper T cells and $CD8^+$ cytotoxic T cells, are the most common cells in DLE. A considerable proportion of $CD8^+$ T cell protein products, such as granzyme B and the T-Cell Restricted Intracellular Antigen 1 (TIA-1), are also present in the skin of DLE lesions. It has also been discovered that the chemokine receptor CXCR3 and its ligands CXCL9 and CXCL10 are up-regulated in DLE lesional skin. Recent transcriptome investigations, in which DLE appears to be a mediated process, support these conclusions. Phagocytes, which can be activated by cells, and it has been observed that in DLE lesional skin, they are higher at the DEJ and in the perivascular area. Regarding the expression of B cells in the skin of DLE lesional lesions, the research has not been as consistent. B cells have been found to account for up to more than 25% of the infiltrate in DLE lesional skin, according to researchers, who have noticed a noticeable inflow at the DEJ and in the perivascular area. However, in the dermis of DLE lesional skin, B cells were either scarce or nonexistent.

These earlier investigations described the distribution of these cells in DLE lesional skin but lacked site-specific comparisons with control skin that would identify which cells predominate at specific regions in DLE lesional skin. Understanding the location preferences of these cells can help us better understand the pathogenesis of DLE, and therefore evaluated the expression of T cell, B cell, and macrophage markers as well as the proteins TIA-1, CXCR3, and CXCL10 that these indicators are related with in DLE lesional skin, psoriasis lesional skin, and normal skin. Because T cells and macrophages play a significant role in the pathogenesis of psoriasis, it was chosen as a disease control. T cells in psoriasis also contain a sizeable fraction of cells in addition to showing and polarising. Due to the prominence of the inflammatory infiltration in the DLE lesional skin anticipated that T cells, B cells, and macrophages concentrate in the periadnexal area and Dermo Epidermal Junction (DEJ) of DLE lesional skin when compared with psoriasis lesional skin and normal skin.

In the outpatient clinics of Parkland Memorial Hospital and University of Texas Southwestern Medical Center in Dallas, Texas, skin biopsies of DLE lesional skin lesions were conducted. The diagnosis of DLE was made using clinicopathological correlation. By counting the number of SLE criteria set forth by

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the American College of Rheumatology that were met, DLE patients were also assessed for Systemic Lupus Erythematosus (SLE). Each DLE case's medical records were examined for demographic data, SLE criteria, and treatment at the time of the biopsy. Biopsies of normal skin and psoriasis lesions were obtained from the Cockerell Dermatopathology archives.

## CONCLUSION

Each DLE, psoriasis, and normal skin biopsy specimen was cut into 4-micron thick tissue sections that were then formalin-fixed, paraffin-embedded, and placed on glass slides. Hematoxylin and eosin was used to stain the preliminary sections. Cell Marque Corporation, Hot Springs, Arkansas; Biocare Medical, Concord, California, Hot Springs, Arkansas, Tucson, Arizona, Minneapolis, Minnesota, BD Pharmingen and TIA-1, Concord, primary anti-human antibodies; Hot Springs, and Arkansas; were used. Utilizing previously described techniques and correctly staining negative and positive control samples, immunostaining was carried out on an Automated Ventana Benchmark Equipment (Ventana) using avidin-biotin-peroxidase complex and 3,3'-diaminobenzidine as substrate. Hematoxylin and bluing reagent was used to counterstain the slides.