

SUMMARY AND CONCLUSION

The present study was conducted on 60 human beings comprising 30 psoriatic patients, who stopped treatment for 3 weeks, and 30 normal volunteers. For each case, skin biopsy was obtained and to clarify the hereditary role played in the pathogenesis of psoriasis, pedigrees construction, consanguinity and segregation analysis and chromosomal study were done. The skin biopsies were stained by Hx, & E. stain; Van Gieson's stain, Methyl Green Pyronin (M.G.P.) stain and ATPase histochemical method.

Hx & E stain of psoriatic skin revealed regular elongation of the epidermal ridges with thickening in their deep portion, elongation and edema of the dermal papillae, thinning of the suprapapillary portions of the stratum Malpighii, parakeratosis and absence of granular layer, intermingled with orthokeratosis with presence of granular layer. Also, Munro microabscesses were diagnostic for psoriasis.

By Van Gieson's stain, the nuclei appeared black brown to black, and the collagen fibers stained red. There was thickening in the epidermis of psoriatic skin and collagen fibers in the dermis appeared more condensed and there was

increase in the number of fibroblasts nuclei. This suggested an increase in the activity of psoriatic fibroblasts.

By M. G. P. stain, the RNA of cytoplasm appeared red and nuclear DNA appeared blue green. The amount of DNA and RNA increased in the epidermis of psoriatic skin when compared to that of normal skin.

By ATPase histochemical staining method, the L. Cs. could be demonstrated and appeared as dendritic cells, polygonal to oval cells, or as dendritic fragments. Normal skin showed the cells as continuous network in the suprabasal Malpighian layer and they were demonstrated in hair follicles. The cells were never seen in the stratum corneum. No cells could be demonstrated in the dermis but they were confined to the ducts of sweat glands.

In psoriatic specimens, however, the distribution of L. Cs. altered completely from that of normal skin. In 20 cases, large segments of the epidermis were totally devoid of L. Cs, though their distribution in hair follicles was conserved. In the remainder 10 cases, there was abnormal clustering of L. Cs. in the epidermis and groups of ATPase +ve cells were found at any level of the epidermis and frequently at the tip of dermal papillae and in dermal papillae themselves. Also, L. Cs. reached the stratum corneum. In the dermis of psoriatic skin, clusters of L. Cs. could be demonstrated and clusters also

appeared in the dermal papillae. The mean number of L.Cs / mm^2 was 455 ± 11.6 in normal male skin and 449 ± 10.4 in normal female skin. There was no sex difference of L. Cs. density. The mean number of L.Cs/ mm^2 in psoriatic male skin was 112 ± 17.1 and 114 ± 18.2 in psoriatic female skin. The density of L. Cs. was significantly decreased in psoriatic skin at $P < 0.001$.

Genetic study revealed that : Consanguinity was not found to have any role in the development of psoriasis. Positive family history was recorded in 20% of cases. Segregation analysis revealed that psoriasis is genetic dependent and the mode of inheritance was found to be multifactorial as it did not follow any simple monogenic type of inheritance neither autosomal dominant nor recessive nor sex linked inheritance.

The chromosomal study of both control and psoriatic groups revealed no significant difference.

From this study, it could be concluded that psoriasis is a heredity-dependent and both fibroblasts and L. Cs. may be contributing factors in the pathogenesis of psoriasis. As the mode of inheritance is multifactorial, the environmental factors have a role in precipitating the disease.