daily. We have registered 250 cases of vitiligo having different presentations, 17 of whom also had involvement of the eyelids. During follow up we observed that 15 of the 17 patients (88.23%) had complete repigmentation of their lesions over eyelids in a period ranging from 6 to 8 weeks. In subsequent visits for treatment of other lesions ranging from 1 to 3 years they did not show any recurrence of the disease over the eyelids. Hence, we conclude that oral levamisole combined with topical hydrocortisone butyrate cream may be the treatment of choice for eyelid vitiligo irrespective of age of the patient and duration of the disease.

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GENESIS OF CUTANEOUS DEPIGMENTATION

To the Editor,

Theories[1] on the aetiology of leucoderma abound but data about its initiation, progression or recession is extremely sparse. This report seeks to understand the causes and progress of cutaneous depigmentation.

Each chemical (2% w/v or v/v in Et. OH): hydroquinone (OH. C_6H_4. OH), p (benzyloxy)phenol (C_6H_5. CH_2O. C_6H_4. OH) p-hydroxypropiophenone (OH. C_6H_4.CO.CH_2.CH_3) butylphenol (C_4H_9.C_6H_4.OH) and amylphenol (C_5H_{11}.C_6H_4.OH) having a common hydrogen donor group (-OH), when appllied topically for 20 days on the vitiligo patients (6 in active stage and 4 in steady stage) and 10 controls without any skin disease, caused pigment dilution on the spots involved, progessing slowly with the repeat application of the chemical. In both groups depigmentation may be the consequence of a reaction between the colour determinant and the chemical in epidermis. The response after the cessation of the chemical application was different; original status of the test spots was restored in control groups after 20 to 30 days, but the acquired transition persisted in the patients with vitiligo. These observations clearly indicate the loss of a mechanism in the vitiligo patients. Further except cosmetic defect, the vitiligo patients show soundness in clinical status like those of healthy individuals. Also both these groups respond alike to the chemical structures with antigenic determinants and are capable to rid off these non-self structures through complement fixation, suggesting that the biomechanisms to neutralise the toxins with and without antigenic determinant(s) are active in the control groups whereas the mechanism to deal effectively with the candidate chemical structures, which fail to elicit antibodies, is lost in the vitiligo patients. Further in controls, skin colour is conditional upon the molecular viability of the colour determining melanolipoprotein as envisaged by Sawhney,[2] and sustainment of the threshold limits of toxin concentration in skin matrix. The bioactive inherent disposition mechanism armed with a neutralising protein does this job through conjugation of the intruders. However the skin faculty is not endowed with the ability to perform protein synthesis,its demand is met through the likely events which include the hepatic synthesis of such neutralising protein and its subsequent transfer via plasma to the

skin faculty. This specific reaction may get stalled in vitiligo patients as their ability to sweat off excess toxin concentration is lost. In acquired hostile environment the colour determinant may undergo structural modification through conjugation with the available toxins with complementary surfaces at the vacant spaces as predicted by Sawhney,[2] losing its property to impart natural shade to epidermis. The resultant structural crisis sets in the trigger mechanism of the pigment dilution in the stratum corneum with slow progression with the percutaneous diffusion of these toxins in skin matrix.

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CLINICAL PROFILE OF PSORIASIS IN WESTERN RAJASTHAN : STUDY OF 300 CASES

To the Editor,

Psoriasis is a common genetically determined, chronic recurrent papulo-squamous dermatosis, characterized by circumscribed, erythematous patches of various sizes covered with silvery white scales. The lesions tend to become confluent and may persist indefinitely. The disease is unpredictable and capricious in its course but is usually chronic.[1]

A total of 300 cases of psoriasis from western Rajasthan were evaluated clinically in a period of one year ie, January to December 1994. Total outdoor registration was 62261 and male to female ration being 1.6:1. Incidence of psoriasis was 0.48% while male to female ration was 3:1. Most of the patients (205;68.34%) had onset of their disease in second to fourth decade of life. Youngest patient was 6 months while oldest was 79 years old. Maximum number of cases were of psoriasis vulgaris (225;75%), second commonest being palmoplantar psoriasis (17;5.67%), followed by sebopsoriasis (14;4.67%) etc.

In our study itching was experienced by 259 (86.33%) of patients while only 41 (13.67%) were asymptomatic, similar observation has been made by others.[2] The incidence of diabetes mellitus in psoriasis reported in literature is 2.4% to 5.7% .[3,4] Similar observation was made in 8 (2.67%) patients in our study. We also noted coincidental diseases in family members of the patients and observed that there was diabetes mellitus in 11 (3.67%) and vitiligo in 6 (2%). Therefore we hypothesise that these three conditions may be interrelated and probably having similar genetic predisposition. The paucity of such a study in literature from this region prompted us to undertake this work

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HYPOHIDROTIC ECTODERMAL DYSPLASIA IN TWO SIBLINGS

To the Editor,