COMPARATIVE METABOLIC STUDIES ON THE SKIN TISSUES OF NORMAL AND PSORIATIC PATIENTS: CHANGES IN GLYCOGEN CONTENT

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Summary

The changes in glycogen content of normal, uninvolved and psoriatic skin tissues were determined. These studies showed considerable increase in glycogen content in psoriatic skin as compared to normal. The uninvolved skin tissues obtained from psoriatic patients also showed significantly higher glycogen levels. The significance of these results are discussed.

Weddel et al[1] pointed out that the uninvolved skin of psoriatic patients differed histologically from the skin of normal persons with regard to the number of nerves and Schwann cells. Subsequently, the studies carried out by Reid and Jarret[2] showed that the uninvolved psoriatic skin differed from the normal skin in its response to the local application of Vitamin A. Histochemical examination of the unaffected psoriatic skin showed a more rapid lysosomal reaction compared to the normal control skin[3].

Glycogen accumulation in psoriatic lesion is a characteristic feature of the disease[4]. On a wet weight basis, the glycogen content of psoriatic skin was 5 times that of the normal skin[4]. Though these studies indicate that substantial metabolic changes may take place in the skin during transition from normal to psoriatic, there is no clear information concerning the metabolic status of the uninvolved psoriatic skin with particular regard to glycogen content. The present investigations were therefore carried out to ascertain whether differences in glycogen content exist between the uninvolved and involved psoriatic skin.

Material and Methods

Fifty psoriatic patients and thirtyfive normal (control) individuals were the subjects of this study. Skin biopsies were taken from the normal subjects and psoriatic patients. In psoriatic patients, the samples were taken from both the involved (lesion) and uninvolved sites. The tissues were immediately placed in formalin until analysed. The control subjects who were comparable to those of psoriatic patients with regard to age and sex had no personal or family history of psoriasis.

The biopsies obtained from a group of five subjects were pooled, digested in KOH and glycogen precipitated by the addition of ethanol as described by Hassid and Abraham[5]. The precipitated glycogen was estimated according to the method of Montgomery[6]. To ascertain

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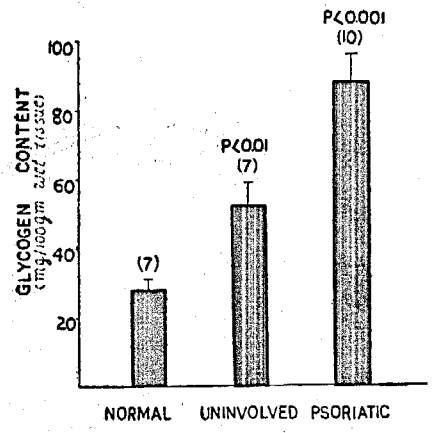
that no alteration in the glycogen content occurred during preservation in formalin, further experiments were carried out on biopsies obtained from all the groups. In such experiments, the skin tissues were not preserved in formalin but immediately analysed. The results were comparable to formalin preserved samples.

Results and Discussion

It is evident from the data presented below that there was about three-fold increase in the glycogen content of



the psoriatic skin as compared to the normal skin. Interestingly, the uninvolved skin tissues of the psoriatic patients also had significantly higher glycogen levels compared to the normal skin. On wet weight basis, there was about 63% increase in glycogen in the uninvolved psoriatic skin and a considerably higher increase of 222% in the psoriatic lesion as compared to the normal skin obtained from persons with no history of the disease. These observations are in agreement with those of Halprin[7]. The exact mechanism of glycogen accumulation in psoriatic lesion and in the uninvolved epidermis of psoriatic patients is not known. The studies involving glycogen synthetase (I Form) and phosphorylase have not revealed any significant difference in the activities of these two enzymes between the normal and psoriatic skin tissues[7]. These studies however suggest that some sort of feed back control may be operating in the psoriatic skin[7].

Although a higher incidence of diabetes in psoriatics has been reported[8] there is also an evidence of higher incidence of glycosuria among non-diabetic psoriatic patients who are administered prednisolone[9].

Burns and Whitehouse[10] found that 8.7% of their uncomplicated psoriatic patients had an abnormal glucose tolerance whereas 38% had abnormal cortisone glucose tolerance. Our earlier studies have also shown abnormal glucose tolerance in 14 3% of patients and abnormal prednisolone glucose tolerance in 12.5% of the cases studied[11].

The finding of an increased glycogen content in the uninvolved epidermis of psoriatic patients, which was much higher than in the normal control skin but less than that in the lesion itself, provides a pointer to the underlying differences in carbohydrate metabolism of these tissues. However, the exact mechanism involved in the pathogenic process remains to be elucidated. Further experimentation with particular regard to the enzymes of carbohydrate metabolism is warranted. Such studies may provide a better comprehension of the metabolic changes taking place during development of psoriasis.

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REFERENCES

1. Weddel G, Crown MA, Palmer E et al: Psoriatic skin, Arch Derm, 91 : 252, 1965.
2. Reid JC and Jarrett A : Enzymic and histological effect of a standard stimulus Arch Derm, 95 : 632, 1967.

3. Jarrett A: The uninvolved psoriatic epidermis Brit J Derm, 89 : 607, 1973.

4. Halprin KM and Ohkawara A: Synthesis of glycogen in the psoriatic skin, J Invest Derm, 45 : 391, 1965.

5. Hassid WZ and Abraham S: Chemical Procedures for analysis of polysaccharides, Methods in Enzymology, Edited by Colowick SP and Kaplan NO, Academic Press, Inc, New York, 1957, Vol 3, p 34.

6. Montgomery R: Determination of Glygogen, Arch Biochem Biophys, 67 : 378, 1957.

7. Halprin KM, Ohkawara A and Levine V: Synthesis of glycogen in the psoriatic lesion, Arch Derm, 107 : 706, 1973.

8. Reed RE, Fusaso RM and Fishes L: Psoriasis Vulgaris, Arch Derm, 89 : 205, 1964.

9. Rechasdt L and Lassus A: Prednisolone glycosuria test in psoriasis, Dermatologica, 138 : 427, 1969.

10. Burns RE and Whitehouse FW: Evidence of impaired glucose tolerance in uncomplicated psoriasis, Arch Derm, 107 : 371, 1973.

11. Hajini GH, Hussain ST and Shah SNA: Impaired Glucose tolerance in Psoriasis, Ind J Derm Vener, 41 : 129, 1975.

TRUE or FALSE?

Flavins protect cellular DNA against UV irradiation injury and is therefore a valuable agent in the propylaxis on UV induced carcinogenesis in mammals.

(Answer page No. 188)