tions (D.P.H.) and of reduced levels of serum and C.S.F. folate. In weanling rats placed on a folate-deficient diet profound falls of serum and tissue folate were observed, in contrast to cerebral folate concentrations which were maintained within the normal range. A similar reduction in serum and tissue folate concentrations was demonstrated in rats administered toxic doses of D.P.H. daily for up to 120 days together with a folate deficient diet; in the presence of substantial serum and tissue concentrations, there was no significant change in cerebral folate levels was observed. Tissue folate and D.P.H. concentrations were determined in cats rendered ataxic by the daily oral administration of D.P.H. for one to four weeks. In the presence of moderate depression of the serum, tissue, and C.S.F. folate concentrations, only minor changes were demonstrated in regional cerebral folates. Whether receiving D.P.H. together with folic acid supplements also exhibited ataxia in the presence of substantial increases in serum tissue and C.S.F. folate and a minor increase in cerebral folate concentration.

These findings indicate that D.P.H.-induced ataxia in cats and the prolonged administration of D.P.H. to rats are not associated with significant alterations of cerebral folate. The ability of D.P.H. to raise, with low toxicity, the convulsive threshold for electrical stimuli in cats led to its successful introduction in the management of epilepsy 30 years ago. Although the anticonvulsant action of D.P.H. was not investigated in the studies described, the maintenance of normal cerebral folate activity in the presence of D.P.H. suggests that this therapeutic action is not mediated through change in cerebral folate concentration.

D.P.H.-induced folate deficiency appears to be a complication unrelated to any thera
topic folate deficiency. Whether receiving the anticonvulsant, unresponsive to folic acid supple
tments, and mental deterioration observed in some epileptic subjects are related to an ultimate reduction in cerebral folate activity or to a direct neurotoxic action of D.P.H. on neural tissue has still to be defined. The unique case of folic acid deficiency from malabsorption with mental retardation, epilepsy, and basal ganglia cal
cification reported by Lanzkowsky et al. confirmed the importance of cerebral transport mechanisms in the maintenance of cerebral folate activity in man.—I am, etc.,

C. D. ALLEN.

Royal United Hospital, Bath.

REFERENCES
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Skin Disease and the Gut

SIR,—I read with interest your leading article in the above subject (1 August, p. 240). On the whole, the relation of skin disease to sprue occurs with some frequency in the

European population. I carried out a survey of skin diseases occurring for the first time since arrival in the tropics to see whether intestinal malabsorption, which is a cardinal feature of sprue, is associated with any one skin disease. Various abnormalities of the skin have been associated with tropical sprue and idiopathic steatorrhoea which include pigmentation and psoriasisiform eruptions.

In my survey I found one group of 35 patients, mostly women, presenting with a patchy eczematous dermatitis of hands and feet, in 17 of whom there was evidence from absorption studies and jejunal biopsy of an enteropathy consistent with early tropical sprue. The enteropathy and the skin disease responded to therapy with folic acid and broad spectrum antibiotics normally given for tropical sprue.

I was also able to substantiate the findings of England and O'Brien that apparently normal Europeans in Singapore had normal mucosa, also that there was no enteropathy associated with tropical impetigo, prickly heat, and acne in the tropics.—I am, etc.,

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