patient in a hyperbaric chamber, should weigh seriously the possible tragic effects that such treatment may have on the fetus.

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^{14}C-LYSERGIDE IN EARLY PREGNANCY

SIR,—Lysergide (lysergic acid diethylamide, L.S.D.) has been reported to be teratogenic in mice,[1] rats,[2] and hamsters [3] if administered in early pregnancy. Chromosomal aberrations have been found in human fetuses exposed to L.S.D. in utero,[4] and three malformed children have been reported [5] following the use of L.S.D. by mothers during the first weeks of pregnancy. Our earlier results on placental transfer of ^{14}C-L.S.D. in mice [6] encouraged us to expand our study to include pregnant hamsters.

Groups of golden Syrian hamsters (17) were injected intraperitoneally (I.P.) with ^{14}C-L.S.D.[6] 500 or 5 μg. per kg. on the 5th–6th or 15th–16th day of pregnancy. Six more animals were injected (500 μg. per kg.) subcutaneously. Animals were killed by bleeding 15, 30, and 120 minutes, and 24 hours after injection, and both total radioactivity and unchanged ^{14}C-L.S.D. were measured in fetuses, placentas, and maternal blood and brain.[6]

With the higher dose (500 μg. per kg.) the amount of radioactivity in fetuses 15 minutes after I.P. injection of ^{14}C-L.S.D. on the 5th–6th day of pregnancy was 4–5 times higher than in fetuses exposed to the same dose of ^{14}C-L.S.D. on the 15th–16th day of pregnancy. The placenta accumulated the radioactivity 1·5–2·0 times more than did the maternal plasma. From 30 to 120 minutes after L.S.D. injection the placental radioactivity decreased, and the difference in radioactivity in fetuses in early and late pregnancy had disappeared. These findings were confirmed in the animals injected with ^{14}C-L.S.D. subcutaneously. With the lower dose (5 μg. per kg.) the early fetuses accumulated 2 times more radioactivity than fetuses during the last days of pregnancy. The highest amount of unchanged ^{14}C-L.S.D. measured in fetuses was 66% (15 minutes after intravenous injection), and a detectable amount of L.S.D. was present in fetuses up to 24 hours after injection.

These results indicate that L.S.D. easily penetrates into the young hamster fetus, but the placenta seems to slow down the transfer during the last week of pregnancy. These data confirm earlier observations in mice.[6] Our results are also in agreement with findings on the potential teratogenic effect of L.S.D. in early pregnancy,[1–3 5] at the time of tissue differentiation. Further, it seems that the chromosome breaks induced by L.S.D. in fetuses in utero [4] could be due to the direct action of L.S.D. on the fetal chromosomes.

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A TRANSMISSIBLE AGENT FROM SARCOID TISSUE

SIR,—Short of culturing the transmissible agent, Dr. Mitchell and Dr. Rees (July 12, p. 81) have satisfied Koch's postulates. Passage of the sarcoid agent from man to mouse, and successful transfer of susceptibility to Kveim's fluid in three sarcoid-inoculated mice is a great advance. Had the tests been made slightly earlier perhaps more of the reactions might have been positive. Two patients of ours with sarcoidosis showed reversion of the Kveim test to negative within a year. Timing is likely to be as critical in mouse experiments as it is clinically. Inert or living, the agent seems to lose its potency rapidly. In attempting to cause systemic lesions in their mice we hope that Dr. Mitchell and Dr. Rees will be using sarcoid homogenate, an ultrafiltrate, and Kveim's fluid intravenously. We believe that their experimental model would then almost exactly resemble "natural" miliary sarcoidosis.

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CHOICE OF POTASSIUM SUPPLEMENTS

SIR,—Dr. McMahon's letter [1] on 'Slow-K' and possible ulceration of the small intestine suggests that this preparation may not always be the ideal potassium supplement. A further consideration is the difficulty many older patients have in swallowing a solid preparation. Liquid preparations are much more suitable for such patients; and this is an important point since older patients are especially likely to develop potassium depletion,[2] even in the absence of diuretic therapy, probably as a consequence of dietary inadequacy.[3] It is now generally accepted that in the treatment of potassium depletion the chloride ion is essential, since other salts of potassium may potentiate the coexistent hypochloræmia.[4] In the light of these observations, a survey of potassium preparations available in this country is interesting. The current *British National Formulary* does not list any liquid preparation of potassium chloride—the effervescent potassium tablets *N.F.* are chloride-free and hence of no value.[5] One tablet of potassium chloride is included in the formulary, but some patients find this preparation unacceptable.[6] Similarly, the four mixtures containing potassium chloride described in *Martindale* [7] are often badly tolerated. Of the proprietary preparations of potassium in the current issue of *MIMS*,[8] there is only one in liquid form. This is 'Katorin', which contains no chloride. 'Camcopot', the only listed tablet of potassium chloride other than the enteric-coated tablets whose use has been discouraged[6], resembles the *National Formulary* tablet in that it dissolves with difficulty—thus there is a risk that the careless patient may ingest the undissolved salt. The remaining two preparations, 'Kloref' and 'Sando-K', are effervescent and contain both potassium and chloride. For the past year both of these have been in regular use in this department, and they have proved therapeutically effective and acceptable to our patients.

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