perinatal thymus [3], and in normal as well as abnormal glands. These cells were present in various stages of development, but it can be seen (figs. 1–3) that they possess the sarcomeric and myofilamentous structure of striated muscle.

The occurrence of these muscle cells at all ages, in normal and abnormal thymic glands, argues against their being present as a mere accident of evolution. Perhaps they should be referred to as striated-muscle cells rather than myoid cells.

Middlesex Hospital Medical School,
London W.1. KRISTIN HENRY.

IS LYSERGIDE A TERATOGEN?

SIR,—Lysergide (lysergic-acid diethylamide, L.S.D.) causes chromosomal aberrations in cultured human leucocytes,[9] and maybe teratogenic in rats,[10] mice,[11] and hamsters.[12] A mother who took lysergide during the first trimester of pregnancy had a congenitally malformed baby.[13] We have not been able to detect any significant teratogenic effects in the offspring of does treated with lysergide.

Lysergide tartrate (20 or 100 μg. per kg. body-weight) was administered daily by stomach tube as an aqueous solution to pregnant New Zealand white does for different intervals during the early stages of gestation (see table). This treatment produced a hyperthermic effect, similar to that reported by Horita and Dille.[15] Two groups of pregnant does served as controls: the first group received no treatment during pregnancy; the second group received daily doses of thalidomide (150 mg. per kg.) as an aqueous suspension by stomach tube on days 8–12 of gestation. On the 28th day of pregnancy, the does were killed by the rapid intravenous injection of a solution of sodium pentobarbitone (60 mg. per kg.). The uterus was rapidly exposed, opened, and the numbers of implantation-sites, resorptions, and viable fœtuses were recorded. The viable fœtuses were then individually weighed and examined for external and internal gross malformations. In some cases the skeletal development of the fœtuses was determined by X-radiography or by staining with alizarin. The results obtained are shown in the table.

Thalidomide exerted its well-known embryotoxic effect: increased incidence of resorptions, decrease in the mean fœtal weight at 28 days, and malformations of the fœtuses similar to those already described.[16] By contrast, pregnant rabbits given lysergide tartrate produced litters which were not significantly different from those of the untreated does in average number of implantations, incidence of resorptions, and average size. In addition, no gross malformations could be detected in the 28-day-old fœtuses of does treated with this compound. Thus a decrease in the mean fœtal weight at 28 days was the only effect which could be detected in the litters of does treated with daily doses as high as 100 μg. per kg. of lysergide tartrate on days 7–9 of pregnancy. These results indicate that lysergide does not possess significant teratogenic activity in the New Zealand white rabbit, but do not exclude the possibility that it may be a teratogen in other animal species, including man, for species difference in the teratogenic activity of compounds is well known.

Department of Pharmacology,
George Washington University
School of Medicine,
Washington, D.C. 20005. S. FABRO
S. M. SIEBER.

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SIR,—You have warned twice [1] of the dangers of lysergide (lysergic acid diethylamide, L.S.D.). Professor Zellweger and his associates [2] reported a newborn with unilateral fibular aplastic syndrome born to a mother who had taken L.S.D. four times during pregnancy, notably on the 25th day after her last menstrual period (L.M.P.) and three times between the 45th and 98th days. Cohen et al.[3] found greatly increased frequencies of structural chromosome aberrations, but no congenital malformations, in 2 offspring whose mothers ingested L.S.D. during the 3rd and 4th months of pregnancy. We describe here our findings in a newborn whose mother had taken L.S.D. before and during early pregnancy.

A 20-year-old primagravida Caucasian woman, with no history of viral infection or irradiation, ingested illegally obtained L.S.D. twice in November, and twice in December, 1966. L.S.D. was again taken on March 6 and 20, 1967. The patient also reported the use of 60 contraceptive pills (' C-Quens ', Eli Lilly Co.) from the beginning of January until the end of March, 1967. However, she became pregnant during this time, her L.M.P. being Jan. 23, 1967, so that L.S.D. was taken on the 43rd and 57th days thereafter. The pregnancy was uneventful and on Oct. 23, 1967, she gave birth to a full-term healthy girl, Apgar score 9, weight 3290 g., height 51 cm., and head circumference 34 cm.

NUMBER, TYPE, AND DISTRIBUTION OF STRUCTURAL CHROMOSOME ABERRATIONS IN NEWBORN, MOTHER, AND CONTROL

Subject	No. of cells counted with chromosomal complement of:					No. of cells with:			No. of polyploidies
	<45	45	46	47	>47	Chromatid gaps	Isochromatid gaps	Chromatid breaks	
Mother	34	23	139	1	3	5 (C, C, C, D, E)*	1 (B)*	2 (A2, C)*	2
Newborn	21	25	152	0	2	3 (A2, C, C)	0	2 (A1, A2)	0
Control	5	7	187	0	1	3 (A2, A3, D)	0	1 (C)	1

* Indicates individually identifiable chromosomes or chromosome groups

Chromosome studies of the mother and baby were begun on the 2nd day after delivery. A healthy laboratory technician with no history of drug intake, irradiation, or recent viral infection served as control. Karyotypic analysis showed normal

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EMBRYOTOXIC ACTIVITY OF THALIDOMIDE AND LYSERGIDE IN THE NEW ZEALAND WHITE RABBIT

Group	Treatment	Daily oral dose per kg. body-weight	On days of pregnancy	No. of does	Total no. of implantations	No. of resorption-sites	Normal fœtuses	Malformed fœtuses	Mean fœtal body-weight (g. ±S.E.M.)
I	None	..	..	6	45	3 (7%)	42	0	33·7±0·7
II ..	Thalidomide	150 mg.	8–12	4	33	8 (*24*%)	12	13 (*39*%)	24·3±0·9*
III	Lysergide tartrate†	20 μg.	4–7	2	24	1 (*4*%)	23	0	32·6±0·8
IV	,, ,,	20 μg.	8–12	9	72	3 (*4*%)	69	0	31·4±0·7
V	,, ,,	100 μg.	7–9	3	27	1 (*4*%)	26	0	28·8±0·9*

* $P<0.001$. † Lysergide tartrate (m.p. 192–194°C) was a gift of Sandoz Pharmaceuticals (batch no. 160019); the infra-red spectrum of the lysergide obtained from this sample was identical to that of a reference compound.[14]

female constitutions, with no significant increase of gaps, breaks, or other structural aberrations; the distribution of chromosome breaks and gaps among various identifiable chromosomes or chromosome groups seemed random in the 17 cells with structural chromosome aberrations (see accompanying table). 1 cell from the mother, not included in the table, showed a possible quadriradial [4] formation. Aneuploidy (loss of chromosomes from the complement) was significantly increased in the mother and the baby ($P < 0.01$). Loss of chromosomes from the complement seemed random in all three subjects.

There is no way of determining the exact dose of L.S.D. ingested by the mother before and during pregnancy, although the dose was sufficient each time to produce a psychedelic effect. She took L.S.D. during the critical stage for production of leg deformities, as in the case reported by Professor Zellweger and his associates,[2] but no fœtal limb deformities developed.

We believe that there is a pressing need for additional data on babies born to parents taking L.S.D., since there is insufficient information on the genetic and developmental effects of ingesting this drug before and during pregnancy.

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Medical Genetics Section,
Department of Pediatrics,
Michael Reese Hospital
and Medical Center,
Chicago, Illinois 60616, U.S.A.

HIDEO SATO
EUGENE PERGAMENT.

LEFT-ADRENAL PHLEBOGRAPHY

SIR,—In the article by Dr. Sutton (March 2, p. 453) the technique of selective left-adrenal phlebography used is supposed to have been introduced by Starer in 1965. My own technique for adrenal-vein phlebography seems to be the same as Starer's. It was published in 1962.[5] This technique is referred to in Mikaelson's article,[6] which is mentioned in Dr. Sutton's interesting publication. The first tumour diagnosed in this way was reported by my co-workers and myself in 1964.[7]

St. Erik's Hospital,
Stockholm K, Sweden.

HÄRJE BUCHT.

WHO CAN SPOT A DRUG-ADDICT?

SIR,—Dr. Freeman has loosed off quite a broadside on behalf of psychiatry in his letter last week (p. 589) on the subject of the composition of the advisory panel set out in the memorandum on the Dangerous Drugs (Notification of Addicts) Regulations 1968.[8] He might more prudently have waited until the enemy was at closer range and more positively identified as hostile.

As I understand it, the function of the members of the advisory panel is to give a decision in those cases in which the physical condition of the patient warrants the continued prescription of heroin and the like. Addiction will occur but it will be iatrogenic and therapeutically justified. Under the circumstances one might expect to find a panel largely composed of physicians and surgeons. The fact that there are two psychiatrists is somewhat of a compliment. It is probably the Ministry's intention that the eminent members of our specialty, named by Dr. Freeman, should be free to get on with the serious business of treating addicts who become so by their own misfortune.

Barnsley Hall Hospital
for Mental and Nervous Diseases,
Bromsgrove, Worcs.

A. G. FULLERTON.

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HOW THE SCOTS FEED THEIR BABIES

Sir—Your leader (Feb. 24, p. 403) says:

" Clearly, in the latter part of the 1st year and in the following 3 years the main source of supplementary vitamin D must be the medicinal preparations, since a sufficient intake of vitamin D from the diet is very unlikely in practice."

This is far from clear to some of us. It is common knowledge that a high proportion of children between the ages of one and four years (including, I can assure you, the children of doctors) receive no medicinal vitamin-D supplements. They eat the same food as the rest of their families and usually thrive on it. If your view is correct I wonder why most practitioners, certainly this side of the Border, have not seen a case of rickets for years. Surely, vitamin-D deficiency occurs nowadays only in very exceptional socioeconomic circumstances or in the case of dietary patterns which do not apply to the native population of Britain. Undue emphasis on the importance of supplements may do harm by resulting in more cases of hypercalcæmia due to overdosage.

Council House,
Halesowen, Worcestershire.

J. TWOMEY.

*** Obviously, our leader was *not* clear. We were referring to the situation as recorded in Glasgow by Arneil: we were not making a sweeping national deduction. Perhaps we should have said " an ideal intake", rather than " sufficient". Of course, no-one would expect to find rickets in doctors' children and seldom in mainly rural communities. But Arneil has shown that in large conurbations like Glasgow, with a higher proportion of social classes 4 and 5, rickets (mainly subclinical) is common enough to justify national routine prophylaxis. The risk of hypercalcæmia has been virtually eliminated by statutory limitation of vitamin-D fortification of infant foods.—ED. L.

MEASUREMENT OF BLOOD-GLUCOSE LEVELS

SIR,—I read your leading article (Feb. 24, p. 405) with great interest. I agree completely with your " hope that the non-specific and inaccurate reductiometric methods . . . will now be abandoned ".

I have determined blood-glucose levels in 15 persons in extreme uræmia. The values found by a reductiometric [1] and a glucose-oxidase [2] method differed by about 90 mg. per 100 ml. blood. In uræmia reducing substances accumulate in the blood, and give falsely high values by reductiometric techniques. On the other hand, falsely low figures may be obtained with the glucose-oxidase techniques in patients with uræmia as well as in the neonate.[3]

In my experience the most reliable results are found by the *o*-toluidine method.[4] This is practically specific for glucose, is reproduceable, and is also accurate at low concentrations. The disadvantage with the original procedure is that acetic-acid vapours may be released in the laboratory and be hazardous to technicians and unshielded apparatus. This is, however, overcome in the fully automated method using the Technicon ' Autoanalyser '.[5] I have now used the autoanalyser technique for a year and have found it very useful, practical, and relatively inexpensive. The rate of analyses is 50 samples an hour. Furthermore, the same automated technique can also be successfully used for the determination of urine-glucose. This is important, because the quantitative methods used in clinical practice for measuring urine-glucose are, if possible, still more inaccurate and non-specific than those used for blood-glucose measurement.

Department of Clinical Chemistry,
Central Hospital,
Kristianstad, Sweden.

NILS TRYDING.

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