Conditioned Pyridoxine Deficiency in Rats on Diets Containing Flours of Different Extraction-Rates

THE occurrence of what is considered to be a conditioned pyridoxine deficiency in rats receiving a diet of high aneurin content has previously been reported[1]. The deficiency was recognized by the running screaming fits and convulsive seizures observed in the suckling rats, and the absence of fits in the litters of does which were given a supplement of 40 μgm. pyridoxine daily from parturition. In the basal group with a low vitamin B_1 intake, no fits were observed, and the weight graphs of the litters were normal. The basal diet used in the experiments contained about 61 per cent of white flour, and it was observed, although not reported, that in spite of high vitamin B_1 intake, no fits occurred in the litters of a few rats which had received from parturition the same basal diet with National wheatmeal flour (85 per cent extraction) substituted for white flour.

The effect of extraction-rate on the occurrence of this conditioned deficiency has been investigated in a recent series of experiments. The diet used contained the following percentages of constituents: wheat flour, 60·9; commercial casein, 23·9; dried brewers' yeast, 1·9; salt mixture (McCollum 185, with iron citrate), 2·0; calcium carbonate, 0·5; and vitaminized margarine 10·8, with vitamins A, D and E, and small amounts of potassium iodide and manganese sulphate as previously detailed. The flour was of 72, 77, 80 or 85 per cent extraction, all the samples being prepared from the same batch of wheat. We are indebted to Dr. James Sword, Cereal Laboratory, Scottish Co-operative Wholesale Society, for kindly supplying us with these samples. Each group received the high-level addition of aneurin used in the previous experiment, which on the white flour diet gave a vitamin B_1 intake of approximately 11·27 μgm. per Cal.

Growth. Normal rats of comparable initial weights, fed *ad libitum* from weaning on the experimental diets, which differed only in the extraction-rate of the flour used, showed considerable differences in weight after a test period of eight weeks. On the diets containing 72, 77, 80 and 85 per cent extraction flour respectively, the weight increases for the males averaged 191·0, 200·4, 203·7 and 226·8 gm., and for the females 129·8, 152·3, 150·9 and 157·8 gm. In a group which received the 72 per cent flour diet with a pyridoxine supplement of 40 μgm. daily per rat, the males showed an average weight increase of 198·4 gm. and the females 149·1 gm. in the 8 weeks' period.

Breeding Performance. Eight weeks after weaning, the females in each group were mated with males from the same group, and the marked differences found in the breeding performance are summarized in the following table, which indicates the incidence of the characteristic fits due to the conditioned pyridoxine deficiency in the various groups.

Group	Flour (% Extraction)	No. of litters born	Observed fits*		Av. weaning wt. of litters (gm.)
			No. of litters showing fits	Approx. no. of individual fits	
A	72%	7	7	110	34·3
C	77%	7	6	43	39·1
D	80%	7	2	13	43·5
E	85%	8	0	0	44·8
B	72% +pyridoxine	6	0	0	42·1

* The rats in all groups were under observation for the same period daily, which was approximately eight hours.

It appears that there is a progressive diminution in the degree of the conditioned pyridoxine deficiency as the extraction-rate of the flour is raised, so that with the 85 per cent flour the imbalance of B factors caused by excessive intake of vitamin B_1 has been corrected sufficiently to prevent the occurrence of the fits typical of this deficiency. It will be noted that there is also a progressive increase in the weaning weights of the young rats with the rising rate of extraction, the difference between the 72 and 85 per cent groups amounting to 10·5 gm. per rat.

Weight of Thymus. The average thymus weights of the rats at weaning in this series of experiments were as follows:

Mean Thymus Weights (gm./100 gm. body weight)

Group	*A*	*B*	*C*	*D*	*E*
Female weanlings	0·254	0·345	0·320	0·328	0·330
Male weanlings	0·205	0·317	0·261	0·280	0·317

For our stock weanlings the values usually found for thymus weight lie between 0·30 and 0·40 gm. per 100 gm. body weight, with an average of about 0·35 gm. The figures illustrate the tendency we generally find in weanling rats for the thymus of the females to be heavier than that of the males. Further, the low values for Group *A* are in accordance with our usual findings in pyridoxine deficiency. When these lower values are considered in conjunction with the lower weaning weight of the group, it will be seen that the absolute reduction in weight of the thymus of these young rats is very considerable. It is known that vitamin B factors other than pyridoxine affect the development of the thymus after weaning, and the higher weights found in Groups *C*, *D* and *E* may doubtless be ascribed in part to the influence of these other factors; but the values found in Group *B* would indicate that pyridoxine alone plays a large part in promoting the development of the thymus before weaning.

MARION B. RICHARDS

Rowett Research Institute,
Bucksburn.
Aug. 2.

[1] Richards, M. B., *Brit. Med. J.*, i, 433 (1945).

A Rare *Rh* Gene Triad in Mexican Indians

IN a series of ninety-eight Mexican Indians from Tuxpan, Wiener and others[1] tested bloods for the presence of the antigens *A*, *B*, *M*, *N*, *P*, and also four *Rh* antigens. They reported two findings in their first table as follows:

Blood number 9. *OM* Rh_1Rh_2 *Hr* — *P* —
Blood number 63. *OM* Rh_1Rh_2 *Hr* — *P* —

The four *Rh* antisera which they used are shown in Table 1, in which the names used by Wiener are compared with those of Fisher, Cappell and myself.

TABLE 1. NOMENCLATURE OF ANTISERA

Wiener	Murray	Fisher	Cappell
Anti-*Rh′*	1	Γ	Anti-*C*
Anti-*Rh″*	2	H	Anti-*E*
Anti-Rh_0	3	Δ	Anti-*D*
Anti-*Hr*	4	γ	Anti-*c*

According to Fisher's conception of the *Rh* antigens[3], it is impossible for bloods of type Rh_1Rh_2 (Murray 153/423 or Fisher *CDe/cDE*) to fail to react with serum 4 (Fisher's γ). If indeed their anti-*Hr* is a potent serum 4 as Wiener and his colleagues claim, and it does not miss occasional single doses of antigen in cells, then the reactions of these two bloods correspond to the phenotype Rh_{123}.

The possible genotypes covered by the phenotype Rh_{123} are shown in Table 2, in which 153/123 (Rh_1Rh_2) is much the least rare. The existence of this rare type has been proved in Britain, and the inheritance of the gene triad 123 (Rh_2) has been traced in a fortuitous family pedigree[3].

TABLE 2

Possible genotypes	Phenotype	Reaction with antisera 1 2 3 4
Rh_1Rh_2 153/123 $Rh'Rh_2$ 156/123 Rh_1Rh_y 153/126 Rh_2Rh_2 123/123 Rh_2Rh_y 123/126	Rh_{123}	+ + + −

Since both Race[4] and I[5] have found that the frequency of the type Rh_{123} in the British population is only 1 in 1,000, it is remarkable that two such phenotypes should have been found in less than a hundred cases in Mexican Indians. It appears, therefore, that the gene triad 123 (Rh_2) is not so rare in Mexican Indians as it is in the British. It will be interesting to see the relative frequency of this uncommon gene triad in all races, especially in view of the recent suggestion of Fisher and Race[4] that rare types can arise from common ones by cross-over of genes.

JOHN MURRAY

Bland-Sutton Institute of Pathology,
Middlesex Hospital,
London, W.1.

[1] Wiener, A. S., Zepeda, J. P., Sonn, E. B., and Polivka, H. R., *J. Exp. Med.*, **81**, 559 (1945).
[2] Race, R. R., *Nature*, **153**, 771 (1944).
[3] Murray, J., Race, R. R., and Taylor, G. L., *Nature*, **115**, 112 (1945).
[4] Fisher, R. A., and Race, R. R., *Nature*, **157**, 48 (1946).
[5] Murray, J., *Brit. J. Exp. Path.*, **27**, 102 (1946).

Transfer of Energy Between Centres in Zinc Sulphide Phosphors

IT is well known that the colour of luminescence from zinc sulphide phosphors with at least two types of activator is dependent on the character and intensity of the exciting radiation and also on the temperature. Measurements of the temperature-dependence of the separate emission bands characteristic for each of the activators show that it is very probable that above a certain temperature a transfer of energy takes place from one centre to another. The decrease of the intensity of the blue band above a certain temperature is nearly always accompanied by an increase of the green band. Similar results are obtained for the blue and yellow bands in a phosphor activated with silver and manganese. These and other phenomena can be explained by the following considerations.

Introduction of impurities into a zinc sulphide crystal generally produces new levels between the top of the full S^{--} band and the bottom of the empty Zn^{+} band of the pure lattice. These levels, which are normally occupied, can be emptied either by direct ionization through absorption of a light quantum in the impurity centre or by the transfer of an electron to a hole in the full band, the latter arising from the excitation of an electron from the full S^{--} band to the conduction band. Recombination of free electrons and holes in the impurity levels is responsible for the luminescence. An electron of the full band may also be transferred to an empty impurity-level of an ionized luminescence centre by thermal excitation or by absorption of an infra-red quantum. The hole thus transferred from this level to the S^{--} band may travel through the lattice until it is recaptured by a similar level or captured by another impurity level.

In this way energy transfer between the centres of different kinds will take place mainly from centres the levels of which are close above the S^{--} band to a centre in which this distance is larger. Let us assume that in a zinc sulphide phosphor with only blue and green centres the levels belonging to the blue centres are nearer to the full band, and that the transfer of a hole from a green centre back into the full band by thermal excitation is unlikely. We then have during excitation under equilibrium conditions: