J. Pharm. Pharmacol., 1964, **16**, 129.

Letters to the Editor

The anaphylactoid reaction in rats

SIR,—The inflammatory anaphylactoid reaction produced in rats by the single intraperitoneal injection of dextran or egg-white is mediated chiefly through a release of 5-hydroxytryptamine and histamine (Parratt & West, 1957). Recently, Harris & West (1963) found that not all rats of the Wistar strain react to this injection although the concentrations of histamine and 5-hydroxytryptamine in the skin of rats not reacting are similar to those in the skin of those reacting. Non-reactivity has since been shown to be a genetically controlled character (Harris, Kalmus & West, 1963) and thus the problem is of wide application. We have re-examined the reactivity of Wistar rats to dextran and egg-white, and studied their responses to various dextrins as Veilleux (1963) has shown that some dextrins of relatively low molecular weight produce the anaphylactoid reaction in rats.

Wistar albino rats obtained from The Wellcome Laboratories, Beckenham and from the Agricultural Research Council's Field Station at Compton were used in all experiments. They were injected intraperitoneally with dextran (Intradex, Glaxo) according to the method of Harris & West (1963) and divided into two types: those which showed the anaphylactoid reaction (hereinafter called Reactors) and those which did not react (called Non-reactors). Both reactor and non-reactor rats were given various agents either intraperitoneally immediately after an intravenous dose of azovan blue dye (18 mg/kg), or intravenously together with the dye. The agents were dextran (Intradex, Glaxo) having a molecular weight of about 145,000, dextran (Rheomacrodex, Pharmacia) with a molecular weight of about 40,000, ovomucoid (L. Light & Co.), fresh hen's egg-white (50% v/v in normal saline), dextrin (Astra), and dextrin (Kerfoot). Fresh hen's egg-white previously boiled for 1 min was also used. The extent of the colloidal dye accumulation in the extremities (e.g. nose, ears, feet and tail) was estimated on a relative scale from 0 to +++. The results shown in Table 1 are the mean scores of the maximal responses from groups of 4 rats, except in the case of both egg-white preparations, where the highest intravenous dose was given to 16 non-reactor rats.

TABLE 1. RESPONSE OF THE AGENTS WHEN INJECTED INTRAPERITONEALLY AND INTRAVENOUSLY INTO REACTOR OR NON-REACTOR RATS, MEASURED ON A RELATIVE SCALE FROM 0 TO +++

Agent	Dose		Intraperitoneal route		Intravenous route	
	mg/kg	ml/kg	Reactors	Non-reactors	Reactors	Non-reactors
Fresh egg-white	—	3	++	0	++	0
	—	6	++	0	++	+
	—	12	+++	0	++	++
Boiled fresh egg-white ..	—	3	++	0	++	0
	—	6	++	0	++	0
	—	12	+++	0	++	+
Ovomucoid	200	—	+++	0	++	0
Dextran (Glaxo)	480	—	+++	0	+++	0
Dextran (Pharmacia) ..	480	—	+++	0	+++	0
Dextrin (Kerfoot)	1,250	—	0	0	0	0
	2,500	—	0	0	0	0
Dextrin (Astra)	1 250	—	+++	+++	++	+
	2 500	—	+++	+++	++	+++

Four interesting points may be deduced from the Table.

(1) Whereas with both samples of dextran given intraperitoneally or intravenously non-reactor rats failed to produce the anaphylactoid reaction, large *intravenous* doses of egg-white were effective.

(2) The component in egg-white responsible for the reaction shown by reactor rats was not destroyed by heat. In contrast, boiled egg-white in non-reactors gave a less intense reaction than did fresh egg-white.

(3) As egg-white was ineffective in non-reactor rats when given intraperitoneally but effective by the intravenous route, the active component when egg-white is given intraperitoneally may not reach the blood stream in concentrations sufficient to produce a response or it may be modified before absorption from the abdominal cavity.

(4) Whereas dextrin (Kerfoot) was ineffective by both routes in reactor and non-reactor rats, dextrin (Astra) when given intraperitoneally was equally effective in the two kinds of rat. However, the larger intravenous dose of dextrin (Astra) was less active in reactor rats than in non-reactors.

Department of Pharmacology,
School of Pharmacy,
University of London,
29/39, Brunswick Square,
London, W.C.1.
December 13, 1963

S. I. ANKIER
G. B. WEST

References

Harris, J. M. Kalmus, H. & West, G. B. (1963). *Genet. Res.*, **4**, 346–355.
Harris, J. M. & West, G. B. (1963). *Brit. J. Pharmacol.*, **20**, 550–562.
Parratt, J. R. & West, G. B. (1957). *J. Physiol.*, **139**, 27–41.
Veilleux, R. (1963). *Brit. J. Pharmacol.*, **21**, 235–237.

Dexamphetamine and lipid mobilization in obesity

SIR,—We have studied the action of dexamphetamine on plasmatic free fatty acids (FFA) in rats. Albino Sprague-Dawley male rats were given amphetamine subcutaneously, 135 min before killing by decapitation. The drug induced a marked rise in levels of such a blood component (Table 1). The rise reached a maximum within 2–3 hr and lasted, at the highest dose levels, for more than 7 hr.

TABLE 1. CHANGES IN PLASMA FREE FATTY ACIDS (FFA) AFTER DEXAMPHETAMINE TREATMENT IN RATS

Dexamphetamine sulphate mg/kg	FFA % rise	P†
0·5	20 ± 9·9*	< 0·05
1·0	48 ± 2·3	< 0·01
2·0	101 ± 8·9	< 0·01
5·0	127 ± 4·7	< 0·01
10·0	69 ± 3·9	< 0·01

* Mean (6 animals) ± s.e.
† Statistical significance of difference from controls.
FFA were determined by the Dole (1956) method.