which are not accessible to neurally released noradrenaline. One site for this action may be the β-adrenoceptors situated on the longitudinal muscle (Kosterlitz & Watt, 1965). Most of the sympathetic fibres terminate in Auerbach's plexus (Norberg, 1964) and the response to perivascular stimulation is hardly affected by β-adrenoceptor antagonists (Watt, 1971).

Possible explanations for the greater part of the postsynaptic action of prostaglandin are potentiation of the response to acetylcholine (Harry, 1968, Kadlec, Masek & Seferna, 1974) or an increase in the acetylcholine released from the Auerbach's plexus. However, in our experiments the response of guinea-pig ileum to acetylcholine was not affected by indomethacin (7 μM) confirming the findings of Bennett, Eley & Stockley (1975) and indicating that the effects observed in the presence of indomethacin were not due to inhibition of the response to acetylcholine.

G.J.S. is a Science Research Council scholar.

References

BENNETT, A., ELEY, K.G. & STOCKLEY, H.L. (1975). Modulation by prostaglandins of contractions in the guinea-pig ileum. *Prostaglandins*, **9**, 377–384.

CLEGG, P.C. (1966). Antagonism by prostaglandins of the responses to various smooth muscle preparations to sympathomimetics. *Nature (Lond.)*, **209**, 1137–1139.

HARRY, J.D. (1968). The action of prostaglandin-E_1 on the guinea-pig isolated intestine. *Br. J. Pharmac.*, **33**, 213P.

HEDQVIST, P. (1969). Modulating effect of prostaglandin-E_2 on noradrenaline release from the isolated cat spleen. *Acta. physiol. scand.*, **75**, 511–512.

KADLEC, O., MASEK, K. & SEFERNA, I. (1974). A modulating role of prostaglandins in contractions of the guinea-pig ileum. *Br. J. Pharmac.*, **51**, 565–570.

KOSTERLITZ, H.W. & WATT, A.J. (1965). Adrenergic receptors in the guinea-pig ileum. *J. Physiol. (Lond.)*, **177**, 11P.

NORBERG, K.A. (1964). Adrenergic innervation of the intestinal wall studied by fluorescence microscopy. *Int. J. Neuropharmacol.*, **3**, 379–382.

WATT, A.J. (1971). The inhibitory effect of perivascular stimulation on the responses of guinea-pig isolated ileum. *J. Physiol.*, **219**, 38–39P.

The effect of subcutaneous injections of adrenaline on platelet MAO activity

R.C. BOURNE, C.D. FRITH, F. OWEN & G.J. RILEY (introduced by T.J. CROW)

Division of Psychiatry, The Clinical Research Centre, Harrow.

Gentil, Greenwood & Lader (1975) have recently reported large increases in platelet monoamine oxidase (MAO) activity following the subcutaneous injection of adrenaline and suggested that "stress" might produce rapid changes in the activity of the enzyme. In view of the relevance of this finding to the conflicting reports of a reduction of platelet MAO activity in schizophrenia (Murphy & Wyatt, 1972; Meltzer & Stahl, 1974; Shaskan & Becker, 1975; Bailey, Crow, Johnstone & Owen, 1975) we have attempted to verify the observations of Gentil and his colleagues and to improve the experimental design by (a) more frequent blood samples using an in-dwelling

Table 1 The effect of adrenaline and placebo on platelet MAO activity

Benzylamine concentration	*Sample*	*Injection received*		
		Placebo (n=6)	*Adrenaline (n=6)*	* t
1 mM	Pre-injection	27.8 ± 2.7	33.2 ± 6.3	—
1 mM	+20 min	27.7 ± 3.9	37.3 ± 5.9	4.61†
1 mM	+40 min	31.0 ± 4.8	33.5 ± 5.6	0.32
1 mM	+60 min	29.4 ± 4.3	33.7 ± 6.4	0.21
1 mM	+80 min	29.8 ± 4.3	35.1 ± 6.6	0.11
2.1×10^{-5} M	Pre-injection	1.70 ± 0.30	1.82 ± 0.44	—
2.1×10^{-5} M	+20 min	1.82 ± 0.26	2.08 ± 0.37	1.93
	Age (yrs)	28.2 ± 5.6	27.5 ± 3.0	0.25

* *'t'* values refer to a comparison between pre- and post-injection samples of subjects receiving adrenaline only. † $P < 0.05$.
Results, mean ± s.d. expressed as nmoles product formed per mg protein 30 min^{-1}.

catheter, (b) determination of platelet counts on all samples and (c) the inclusion of a group receiving placebo.

Twelve male volunteers received subcutaneous injections of either water or adrenaline tartrate (1 : 1000 solution, 1 ml/70 kg body weight). Blood samples (20 ml) were collected into a citric acid-sodium citrate-dextrose buffer (ACD; 5 ml) for platelet MAO assay and 5 ml into disodium ethylenediamine tetraacetate (EDTA) for platelet count determination. Samples were taken, from an indwelling catheter in the ante-cubital vein, immediately prior to injection and at 20, 40, 60 and 80 min post-injection. Platelet MAO activity was assayed by the method of Robinson, Lovenberg, Keiser & Sjoerdsma (1968) with benzylamine as substrate at a concentration of 1 M for all samples, and at the much lower concentration of 2.1×10^{-5}M used by Gentil *et al.* (1975) for the first two samples only. Platelet counts were determined with a Technicon Autocounter. The results of the platelet MAO assays are presented in Table 1.

There was a small but significant increase ($P < 0.05$, paired '*t*' test) in platelet MAO activity of the group receiving adrenaline in the 20 min post-injection samples only, with benzylamine as substrate at 1 mM concentration. There was a small but not significant increase in the activity of platelet MAO, in both groups of subjects, with benzylamine at a concentration of 2.1×10^{-5} M. Platelet counts were significantly increased in the 20 min ($P < 0.001$) 40 min ($P < 0.05$) and 60 min ($P < 0.05$) only in the group receiving adrenaline. As suggested by Gentil *et al.* (1975) the efflux of a different population of platelets from the spleen may account for the increase in platelet MAO activity observed in the 20 min post-injection samples.

We were unable, therefore, to fully confirm the findings of Gentil and his co-workers. The small changes in platelet MAO activity attributable to 'stress', at least in as far as it is mimicked by injections of adrenaline seem unlikely to have an important bearing on the current controversy over the activity of the enzyme in schizophrenia.

References

BAILEY, A.R., CROW, T.J., JOHNSTONE, E.C. & OWEN, F. (1975). Platelet monoamine oxidase activity in patients with chronic schizophrenic illnesses untreated by neuroleptic drugs. *Br. J. clin. Pharmac.*, **2**, 380P.

GENTIL, V., GREENWOOD, M.H. & LADER, M.H. (1975). The effect of adrenaline on human platelet MAO activity. *Psychopharmacologia*, **44**, 187–190.

MELTZER, H.Y. & STAHL, S.M. (1974). Platelet monoamine oxdiase activity and substrate preferences in schizophrenic patients. *Res. Commun. Pathol. Pharm.*, **7**, 419–431.

MURPHY, D.L. & WYATT, R.J. (1972). Reduced monoamine oxidase activity in blood platelets from schizophrenic patients. *Nature (Lond.)*, **238**, 225–226.

ROBINSON, D.S., LOVENBERG, W., KEISER, H & SJOERDSMA, A. (1968). Effects of drugs on human blood platelet and plasma amine oxidase activity *in vitro* and *in vivo*. *Biochem. Pharmac.*, **17**, 109–119.

SHASKAN, E.G. & BECKER, R.E. (1975). Platelet monoamine oxidase in schizophrenics. *Nature (Lond.)*, **253**, 659.

Role of lymphocytes in accumulation of fibrin in rabbit skin homografts

CATHERINE S. BACH & M.K. JASANI

Horsham Research Centre, CIBA Laboratories, Horsham, West Sussex, RH12 4AB.

Indomethacin-like drugs relieve joint pain and stiffness in rheumatoid arthritis but do not reduce the swelling. Further insight into this problem has been obtained by investigating the mechanisms influencing the fibrin content of homografts, as suggested by Jasani, Lewis & Tweed (1974).

Fibrin accumulation was studied by administering iodinated ^{125}I-labelled human fibrinogen (100 μCi in 1 ml water, i.v.) to New Zealand White rabbits bearing six homografts (from Norfolk donors) of the right hind limb and an equal number of autografts on the left leg. One homograft and its corresponding autograft were removed daily from day four. ^{125}I-labelled fibrinogen was given on day five and a blood sample taken daily thereafter. Grafts were kept frozen (−20°C), homogenized (Jasani, 1973) and the water-insoluble radioactivity determined and expressed as a fraction of blood radioactivity according to Colvin & Dvorak (1975), thus minimizing inter-animal variations.

Figure 1a shows that the insoluble ^{125}I-labelled fibrinogen content, although similar at first in both types of graft, began to increase significantly in the homografts following the appearance of cyanosis.

determined and expressed as a fraction of blood radioactivity according to Colvin & Dvorak (1975), thus minimizing inter-animal variations.

Figure 1a shows that the insoluble ^{125}I-fibrinogen content, although similar at first in both types of graft,