1970) it may be that inhibition of PG synthesis in the brain affects the actions of amphetamine in the same way as in the periphery.

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Dissociation of bacterial pyrexia from prostaglandin E activity

A. ARTUNKAL, E. MARLEY* & J.D. STEPHENSON

Department of Pharmacology, Institute of Psychiatry, De Crespigny Park, London SE5 8AF

Prostaglandins of the E series (PGE's) elevate body temperature in a number of species and have been implicated as hypothalamic mediators of fever (Milton & Wendlandt, 1971; Feldberg & Saxena, 1971). Whereas PGE_1 infused into the hypothalamus of adult fowls (Nisticò & Marley, 1973) or young chicks (Artunkal & Marley, 1974) elevated body temperature at a thermoneutral ambient temperature, it was found to be markedly hypothermic when infused into the hypothalamus of young chicks at an ambient temperature (Ta) below thermoneutrality, viz 16°C (Artunkal & Marley, 1974); 16°C is not a severe thermal load for chicks of this age (Marley & Stephenson, 1975). Both the hyperthermic and hypothermic effects of PGE_1 in young chicks were potentiated by indomethacin (Artunkal & Marley, 1974), a prostaglandin synthetase and dehydrogenase inhibitor (Ferreira, Moncada & Vane, 1971; Vane, 1971; Flower, 1974).

Present experiments exclude the possibility that this potentiation was solely due to inhibition of prostaglandin dehydrogenase, since similar potentiation was obtained after pretreatment with 5,8,11,14-eicosatetraynoic acid (TYA), 3.4 μmol/100 g, i.v., 30 min previously; TYA is an arachidonic acid analogue which selectively inhibits prostaglandin synthetase (Flower, 1974). The International Pyrogen reference preparation, *Shigella dysenteriae* (1 μg), infused into the hypothalamus of chicks consistently elevated body temperature by 0.5°-2.0°C after a delay of 1-2 h at ambient temperatures of 31° and 16°C, effects that were not potentiated by indomethacin (1.4 μmol/100 g., i.v., 30 min previously).

Comparison of the effects of PGE_1 with those of *Shigella dysenteriae* yielded two important differences: (1) PGE_1 was hypothermic at a TA of 16°C whereas *Shigella dysenteriae* elevated body temperature; (2) the effects of *Shigella dysenteriae* on body temperature, unlike those of PGE_1, were not potentiated by indomethacin. Nor could the hyperthermic effects of *Shigella dysenteriae* be attributed to PGE_2 since at a Ta of 16°C, PGE_2 (14.3 nmol) infused into the hypothalamus, lowered body temperature up to 4.75°C.

The results demonstrate that prostaglandins of the E series are not consistently hyperthermic in all species. Thus, apart from the results in young chicks, hypothermic effects of intraventricular injection of PGE_1 and PGE_2 (each 2 μg) have been demonstrated over a wide range of ambient temperatures in the *Echidna, Tachyglossus aculeatus* (Baird, Hales & Lang, 1974). Additionally, fever evoked in young chicks by bacterial pyrogen can be dissociated from the effects of PGE_1 or PGE_2 in at least two important ways.

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Substrate and strain-dependent differences in the development of monoamine oxidase in the rat brain

D. BLATCHFORD, M. HOLZBAUER* & M.B.H. YOUDIM

A.R.C. Institute of Animal Physiology, Babraham, Cambridge and M.R.C. Unit, Department of Clinical Pharmacology, Radcliffe Infirmary, Oxford

Physicochemical studies and *in vitro* experiments using different enzyme inhibitors have overwhelmingly pointed to the existence of different forms of the enzyme monoamine oxidase (MAO) (for review, see Sandler & Youdim, 1972). Their presence and independent function *in vivo* is more difficult to ascertain. It has been indicated in experiments on rats treated with progesterone in which the increase in adrenal MAO caused by this steroid was significantly different when different

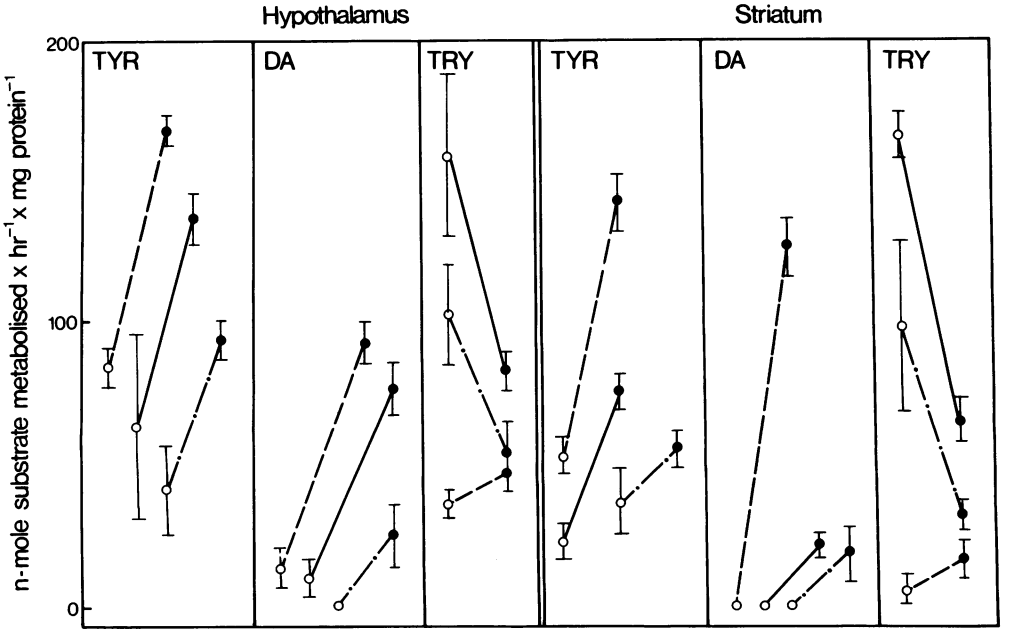

Figure 1 MAO activity in the hypothalamus and striatum of five day old (○) and 20 day old (●) rats. – – –: Porton rats. ——: Wistar rats, bred at Tuck's. · · · · ·: Wistar rats, bred from Tuck stock at Babraham. TYR = tyramine; DA = dopamine; TRY = tryptamine. Mean value ± s.e. of the mean (n = 5).