Table 1 Control and febrile PGE and PGF levels (in pg/ml) in cat CSF

	Sample No.	*Control*	*Long latency*	*Short latency*
PGE	1	137 ± 17	144 ± 24	180 ± 48
	2	137 ± 17	124 ± 4	185 ± 58
	3	138 ± 18	126 ± 6	1756 ± 1355**
	4	142 ± 11	259 ± 101	455 ± 193**
	5	129 ± 10	331 ± 154	1773 ± 682**
PGF	1	84 ± 4	80	109 ± 39
	2	92 ± 8	93 ± 13	130 ± 21
	3	115 ± 24	210 ± 56	759 ± 174***
	4	116 ± 35	181 ± 56	1303 ± 400***
	5	80	142 ± 42	1428 ± 578***
	6	115 ± 35	80	769 ± 170***

Comparison with control values.
Students 't' test *** $P < 0.025$
** $P < 0.05$
Values are the mean ± s.e. mean of 4 observations. Controls $n = 8$.

in pyrogen-induced fever. However, it should be realized that the precursors of these prostaglandins, namely prostaglandins G and H and the thromboxanes are themselves pharmacologically-active and may also be involved in fever.

During the long latency fevers, no significant increases in either the PGE or the PGF levels were found. The reasons for these differences between the short latency and long latency responses have not yet been determined.

This work was supported by a grant from the Medical Research Council. The O-somatic antigen of *Shigella dysenteriae* was kindly supplied by W.H.O. Laboratories, Hampstead, and prostaglandins by Dr John Pike, Upjohn Company.

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Sex differences in guinea-pig brain prostaglandins and the effect of indomethacin

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Adult albino guinea-pigs were stunned and their brains removed quickly. Groups of 2–6 brains were pooled, cut into approximately 2 mm cubes and washed thoroughly with Krebs solution. Samples (1–5 g) were homogenized (30 s; immediately or after incubation at 37°C) in Krebs solution alone or with indomethacin or acid ethanol (Bennett, Stamford & Unger, 1973). The prostaglandin (PG)-like material was extracted (Unger, Stamford & Bennett, 1971) and assayed on rat gastric fundus (Bennett *et al.*, 1973). Results were compared using the Mann-Whitney U test unless otherwise stated.

Using Krebs solution alone the biological activities (medians and semiquartile ranges, ng PGE_2 equivalents/g fresh tissue) were: male 5.8 (4.5 to 17)

$n=8$; female 14 (11.5–22) $n=8$ ($2P=0.19$) after immediate homogenization, and male 7.9 (6.6 to 11) $n=8$; female 10.6 (9.3 to 22) $n=7$ after 30 min incubation. There was less activity in acid ethanol than in Krebs solution homogenates (−71% (−55 to −86), 5 male, 4 female, $2P<0.01$, Wilcoxon test).

With female brain the PG-like activity extracted after immediate homogenization with indomethacin (1 or 10 μg/ml) was always less than controls, (−67% (−63 to −90) $n=5$, and −33 to −65% $n=4$ respectively). In male brains, however, the effect of indomethacin was concentration-dependent: 1 μg/ml increased the activity in 5/6 experiments by 83% (38 to 120) (cf. female, $2P=0.03$), but 10 μg/ml caused inhibition (−28 to −91%, $n=4$). The difference did not seem primarily due to drug penetration or substrate availability. After 30 min incubation with 1 μg/ml indomethacin, biological activity was less in all female brains (−36% (−29 to −52) $n=7$) but the effect in male brains varied (−7% (20 to −13) $n=7$) (cf. female $2P=0.02$). With 40 or 400 μg/ml arachidonic acid in the incubate, 1 μg/ml indomethacin decreased biological activity in 3 female brains (−14 to −66%) but increased activity in 3/4 male brains (0 to 250%).

Extracts of brain homogenized in Krebs solution were chromatographed for group separation of PG (Stamford & Unger, 1972). In female brains only material running as PGE was detected ($n=4$) whereas in male brains PGF-like or PGE + PGF-like material was found ($n=5$) (cf. female $2P=0.016$, Fisher's exact probability test).

Brocklehurst & Dawson (1974) obtained stimulation of PG synthesis with low concentrations of indomethacin, and biological activity sometimes increased in gastrointestinal tissues homogenized with low concentrations of non-steroidal anti-inflammatory drugs (Bennett, Fox & Stamford, 1973). Apart from sex hormones, male and female guinea-pig brains might differ in co-factors for PG synthesis; arachidonic acid or noradrenaline tended to increase thromboxane B_2 but prevented PGE_2 and $F_{2\alpha}$ formation (Wolfe, Rostworowski & Marion, 1976). Alternatively, in male brain, indomethacin might increase the estimated activity by enhancing generation of PGE (to which the bioassay is more sensitive) at the expense of PGF or thromboxanes. Wolfe, Pappius & Marion (1976) found that $PGF_{2\alpha}$ production was more inhibited by indomethacin than PGE_2 production in rat brain.

We thank the Migraine Trust and Wellcome Trust for support.

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Effect of colchicine and lumicolchicine on learning in goldfish

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Cronly-Dillon, Carden and Birks (1974) have reported that intracranial (i.c.) injections of colchicine interfere with long-term memory in goldfish (*Carassius auratus*). Fish were trained in a shuttlebox using an active avoidance task (Agranoff, 1967) in which they were taught to associate a light (CS) with an electric shock (UCS). Using a comparable technique, we have studied the effects of colchicine on goldfish over a longer time period and have, in addition, compared the effects of colchicine with those of its isomer, lumicolchicine.

Fish were trained in a shuttlebox (Aim-Biosciences Ltd.) and the performance of fish receiving 10 μg colchicine or lumicolchicine compared with controls receiving vehicle (Youngs Teleost Ringer solution).