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The effect of short wavelength ultraviolet C (254 nm), irradiation on arachidonic acid and prostaglandins E_2 and $F_{2\alpha}$ concentrations in human skin

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The inability of indomethacin, which inhibits prostaglandin synthetase, to suppress consistently the erythema produced by ultraviolet C (UVC; 254 nm) irradiation suggests that prostaglandins may not be the principal cause of the erythema (Eaglstein & Marsico, 1975).

We have irradiated human abdominal skin with 6 times the minimum erythema dose of UVC and obtained exudate samples by a technique previously described (Black, Greaves, Hensby & Plummer, 1976). Individual samples have been quantitatively analysed at different times after irradiation by combined GC–MS for arachidonic acid, PGE_2 and $PGF_{2\alpha}$ and by radioimmunoassay for $PGF_{2\alpha}$ (Table 1).

Erythema was maximal at approximately 12 h after irradiation and the greatest concentrations of the three compounds were found in the 18 h samples. Oral indomethacin whilst decreasing the PGE_2 and $PGF_{2\alpha}$ concentrations to control values at 24 h only partially reduced the erythema. The relative contributions of blood pooling and increased blood flow to the observed erythema is now being investigated.

Although UVC activates the production of arachidonic acid, PGE_2 and $PGF_{2\alpha}$ the relationship of the latter two compounds to the erythema is uncertain. The possibility that the erythema is mediated by metabolites of arachidonic acid, produced by an indomethacin insensitive pathway, or different pharmacological mediators requires investigation.

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Table 1 The effect of UVC (254 nm) irradiation on exudate concentrations of PGE_2, $PGF_{2\alpha}$ and arachidonic acid as measured by radioimmunoassay and GC-MS

		Time after irradiation (h)				
	Control	*6*	*18*	*24*	*24 (+ oral indomethacin)*	*48*
Radioimmunoassay ng/ml						
$PGF_{2\alpha}$ equivalents	29.5 ± 7.1 (n=32)	53.7 ± 9.5 (n=7) P=0.1	50.4 ± 18.6 (n=5) P=0.2	55.1 ± 8.3 (n=13) P=0.05	6.6 ± 3.9 (n=4) P=0.01	32.3 ± 9.0 (n=3) P=0.9
GC-MS ng/ml						
(a) Arachidonic acid	284.6 ± 24.6 (n=46)	472.7 ± 35.5 (n=7) P=0.01	557.7 ± 64.5 (n=7) P=0.001	535.1 ± 73.02 (n=13) P=0.01	635.8 ± 124.0 (n=5) P=0.05	357.0 ± 26.9 (n=3) P=0.4
(b) PGE_2	21.9 ± 1.2 (n=46)	38.5 ± 2.6 (n=7) P=0.001	64.4 ± 12.0 (n=7) P=0.02	43.3 ± 6.4 (n=12) P=0.01	18.4 ± 1.7 (n=5) P=0.3	35.0 ± 4.9 (n=3) P=0.001
(c) $PGF_{2\alpha}$	18.2 ± 1.2 (n=46)	33.7 ± 4.4 (n=7) P=0.02	49.4 ± 10.2 (n=7) P=0.025	33.8 ± 4.2 (n=13) P=0.005	19.7 ± 3.2 (n=4) P=0.6	20.2 ± 0.7 (n=3) P=0.1

References

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The effect of anti-inflammatory drugs on the prostaglandin system in human subcutaneous adipose tissue

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Prostaglandins are released from rabbit fat tissue *in vivo* and *in vitro* during lipolysis stimulated by ACTH and from rat adipose tissue *in vitro* during adrenaline-induced lipolysis or electrical stimulation (Shaw & Ramwell, 1968). The experiments described show that human subcutaneous fat also releases prostaglandins during lipolysis.

Human subcutaneous fat was obtained (with informed consent) from patients (22–69 years) undergoing major abdominal surgery. The fat was washed several times in Krebs solution, divided into portions weighing 1 g, chopped into pieces approximately 2 mm^3 and rinsed again. Control and test samples of fat were incubated in Krebs solution for 4 hours. Lipolysis was investigated in fat incubated with either ACTH or adrenaline. Lipolysis was measured by glycerol release into the incubation medium; this and the levels of prostaglandins formed in the fat and incubation medium were measured as previously described (Chang, Lewis & Piper, 1977). Dexamethasone (2–4 μg/ml) and indomethacin (10 μg/ml) were used to inhibit prostaglandin release.

ACTH did not cause significant increase in glycerol levels but adrenaline induced lipolysis in a dose-