53.7 ± 3.7 (n = 5) by GCMS. This shows that the two methods give comparable results.

In the second example the effect of NADH (2 mM) on the metabolism of prostaglandin E_2 by the 9-oxo-reductase, present in sheep blood has been studied. Previously this has been shown not to stimulate the reduction of PGE_2 to $PGF_{2\alpha}$ by the enzyme (Hensby, 1974). The rates of $PGF_{2\alpha}$ production in the presence and absence of NADH were found to be comparable when quantitatively assayed either by GCMS or tritium isotope analysis.

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A novel method for evaluating anti-inflammatory drugs in the conscious guinea pig

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The dorsal surface of each ear of individually marked guinea pigs of 200-300 g body-weight was depilated by application of Nair cream. After removal of the cream by washing in tap water the animals were placed in their home cages. Twenty hours later they were placed four in a cage, separated from each other by wire barriers. Test substances were applied in 100-150 mg of a water miscible cream (Acid Mantle cream) to one ear of each animal, the other ear being treated with vehicle.

Twenty minutes after each application all four guinea pigs were placed under a 20 watt 57 cm long ultra-violet strip light placed 8 cm above their heads for 30 minutes. In unprotected animals, such irradiation was sufficient to cause marked increase in temperature of the ears, erythema, delayed oedema and blister formation. During experiments, no food or water was given to the animals until observations on ear temperatures were complete, usually 4 h after irradiation.

Substances applied prior to irradiation of the guinea pigs were re-applied after irradiation. Some substances were only applied after irradiation.

Substances in a concentration of up to 10% w/w applied to the ears included the following: para aminobenzoic acid (PABA), propyl gallate, promethazine hydrochloride, uval (2-hydroxy-4-methoxy-benzophenone-5-sulphonic acid) and bufexamac [p-(n-butoxy)phenylacethydroxamic acid)]. In some experiments propyl gallate or bufexamac was applied only after irradiation.

The sodium salts of aspirin or indomethacin were given in aqueous solution by subcutaneous injection 30 min prior to irradiation of the animals. In one experiment two subsequent treatments with sodium aspirin were given 2 h and 4 h after the first.

Erythema was assessed subjectively by two observers.

Pyrexia was estimated from 5-280 min after irradiation by placing a thermometer probe set in plasticine firmly on the dorsal surface of each ear in turn for 2.5 minutes. Temperature (°C) was measured via a Wheatstone bridge to a twin channel Devices polygraph using a DC2-D pre-amplifier.

Ear thickness was measured by a micrometer screw gauge immediately prior to application of test substance and 24 h after irradiation. Oedema was taken as being proportional to the increase in ear thickness.

Blister formation was assessed subjectively by two independent observers from 5-7 days after irradiation.

Erythema and pyresis were inhibited by prior treatment with subcutaneous sodium aspirin (150 mg/kg) or by topically applying adrenaline, PABA, propyl gallate, promethazine, bufexamac or Uval prior to irradiation or by applying propyl gallate after irradiation. PABA, propyl gallate and promethazine also inhibited oedema. Propyl gallate applied after irradiation gave some protection against blister formation.