Additional work is being done to determine whether the decrease in the ERG amplitude is related to the pigment concentration, or changes in the structure of the retina and the receptor cells.

I thank Mr. David Buffin for performing many of these experiments.

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PHARMACOLOGY

Adrenergic β-Receptors and Non-shivering Thermogenesis

Sellers, Scott and Thomas[1] demonstrated that after prolonged cold exposure (cold acclimatization) heat production through shivering is gradually replaced by an increase in non-shivering thermogenesis. It is often assumed that muscle tissue is an important site of non-shivering thermogenesis. Recently, Himms-Hagen[2] has suggested that acceleration of the triglyceride cycle of brown adipose tissue is a major mechanism of non-shivering thermogenesis in cold-acclimatized rats. As lipid metabolism of adipose tissue is under β-adrenergic control, a study of the effect of β-adrenergic blockade on electrical muscle activity in rats exposed to about 4° C after acclimatization to 23° C or 4° C would be expected to yield valuable information. This communication presents preliminary data of such an investigation. Cold-acclimatized rats were removed from the cold room, anaesthetized with urethane (1 to 1·25 g/kg)—a drug known to alter only the degree, not the type, of the electrical response of muscle activity[1]—and exposed to a temperature of 4° C. Needle electrodes were inserted into a leg muscle and electrical activity was measured with a Grass model 5 polygraph[3]. Rectal temperature was monitored throughout the experiment. Each rat was tested at least twice, once when given drug treatment, and once when given saline or nothing. The β-adrenergic blocking agent, propranolol (*AY* 64043) (kindly donated by Ayerst, McKenna and Harrison, Ltd., Montreal), was used at doses of 0·3 or 0·9 mg/kg intraperitoneally every 30 min. Fig. 1 shows rectal temperature, duration of exposure to 4° C and the effect of the drug on electrical muscle activity in six cold-acclimatized rats. None of the animals shivered during an experimental period of 2·5 h when treated with saline. A seventh rat showed signs of shivering and was therefore excluded from this series. After treatment with propranolol, all six rats showed a marked increase of electrical muscle activity. Representative tracings of the muscle activity before and after propranolol are shown in Fig. 1: no effort was made to quantitate accurately the observed increase.

It is concluded that β-adrenergic blockade interferes with non-shivering thermogenesis so that heat production, mediated by electrical muscle activity, increased.

In order to determine whether the response was dose-dependent, two dose levels of propranolol were used. As can be seen from Fig. 1, the differences between the effects of the two doses are not obvious, and so this question remains to be settled.

It is not possible to draw any conclusions from these experiments regarding the site of non-shivering thermogenesis (muscle, adipose or other tissues); however, they confirm the importance of β-adrenergic receptors in cold-acclimatization. In other experiments it was found that rats acclimatized to 23° C and exposed to 4° C all shivered and propranolol did not interfere with the shivering.

Further investigations of the mechanisms underlying these observations are in progress. This work was supported by grants from the Defence Research Board and the Medical Research Council of Canada.

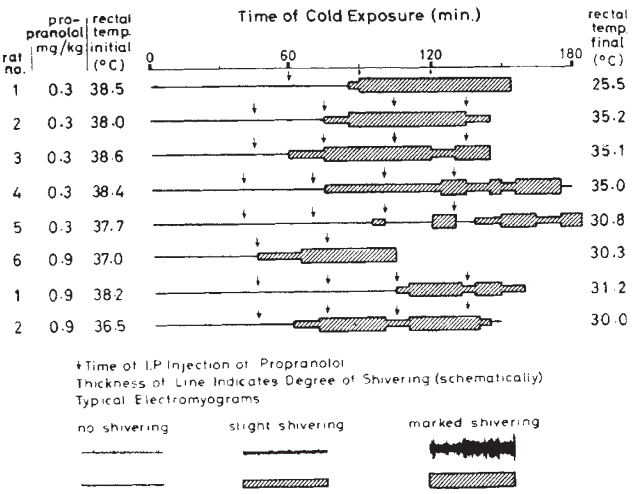

Fig. 1. Effect of propranolol on shivering of cold-acclimatized rats anaesthetized with urethane and re-exposed to 4° C. Note absence of shivering during control period

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Destruction of Cephalosporin C by Ultra-violet Light

WITH the recent development of potent penicillinase-resistant semi-synthetic cephalosporins[1], there is considerable interest in the parent antibiotic, cephalosporin *C* (ref. 2). This antibiotic, which displays a peak of ultra-violet absorption at 260 mμ, is often detected on paper chromatograms by exposure to a source of ultra-violet light. Using this technique during some investigations on the biosynthesis of the antibiotic[3], I noted an extreme lability of cephalosporin *C* to ultra-violet light.

It had been common practice in this laboratory to locate the antibiotic on paper chromatographic strips by placing the strips on the glass filter surface above the ultra-violet light source. After the dark area was circled in pencil, the strip was placed on the surface of an agar medium seeded with either *Escherichia coli* or *Bacillus subtilis*; the plate was incubated at 37° C for 16 h. It was noted that the intensity of the antibiotic activity of the cephalosporin *C* spot varied from experiment to experiment and depended on the type of ultra-violet filter used. (These original observations were made by Robert B. Walton and Richard Burg.) When a red-purple 'Corex *A*' polished filter (No. 9863, Corning Glass Works, Corning, New York) was used, the resulting antibiotic activity was good. On the other hand, use of a clear 'Vycor' glass filter (No. 7910, Corning) resulted in poor antibiotic activity. It was suggested that, because the 'Vycor' filter allows more 260 mμ light to pass, ultra-violet light was being absorbed by the compound on the paper and inactivating it. To investigate this, paper disks (Schleicher and Schuell Co., Keene, New Hampshire) were dipped into a solution containing approximately 100 μg of the sodium salt of cephalosporin *C* per ml. of 0·02 M phosphate buffer (*p*H 7·0). After drying, the