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An Intact Muscle Preparation Suitable for Studies of the Metabolic Effects of Electrical Stimulation

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Studies of the metabolic effects of stimulation on mammalian muscle have usually been carried out on whole hind limbs, either stimulated *in situ* or in perfused preparations. Changes in concentrations of metabolites in the plasma or perfusate from such systems can only indicate the response of a group of muscles, so that information on individual muscles is limited to that obtained by excision and analysis after stimulation. We have designed a system whereby an isolated muscle preparation may be electrically stimulated while allowing the incubation mixture to be readily sampled.

The system is basically that described by Pain & Manchester (1970) with modifications, and was developed for studies of the metabolism of the extensor digitorum longus muscle from young female rats. Each muscle is attached to an electrode and incubated at 37°C in 2ml of Krebs–Ringer bicarbonate buffer continuously gassed with O_2+ CO_2 (95:5). The other electrode is immersed in the medium. Constant tension is applied to the muscle during the passage of electric stimuli, the voltage, duration and frequency of which may be varied to suit a particular type of muscle. Additions to the medium or withdrawal of samples can easily be carried out throughout the incubation period.

When extensor digitorum longus muscles of 30mg or less were incubated in this system for 60min, tissue weights were unaltered, and the concentration of K^+ in the intracellular fluid was maintained at 88% (resting) and 76% (stimulated) of that in zero-time controls. With larger muscles we observed increases in tissue weight and extracellular fluid and decreases in K^+ and ATP concentrations, these changes being particularly pronounced in stimulated muscles. Probably the O_2 supply becomes critical in muscles of this size, and it is likely that this would also be the case for muscles with a higher proportion of aerobic red fibres.

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The Physiological Role of Taurine

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In a previous communication (Kaczmarek, Agrawal & Davison, 1970) we showed that taurine is present in nerve-ending particles isolated from rat brain, together with its synthesizing enzyme, cysteinesulphinate decarboxylase. In both distribution and properties this enzyme closely resembles glutamate decarboxylase, which is responsible for the synthesis of γ-aminobutyrate, a compound believed to act as a neuroinhibitor (Davison, 1956; Agrawal, Davison & Kaczmarek, 1971). We have now extended the work to a study of the uptake of taurine by cortical tissue, as well as its release into extracellular space on stimulation. Rat brain-cortex slices were first incubated for 30min in the presence of radioactive taurine. The slices were transferred to cells that were perfused with oxygenated taurine-free Krebs–Ringer medium. After an initial washout of taurine from the extracellular space a slow loss of radioactivity from the tissue was observed. On the application of rectangular electrical pulses to the tissue, the efflux of radioactivity was considerably enhanced, a result that has been found for γ-aminobutyrate by Srinivasan, Neal & Mitchell (1969). If such a process is of physiological significance and taurine is released from nerve endings to exert an inhibitory influence on the postsynaptic membrane, it should be accompanied by a mechanism for the termination of its action. This may be by re-uptake into the tissue, as has been proposed for the cessation of γ-aminobutyrate action (Iversen & Neal, 1968). We have found that slices of rat brain cortex will efficiently take up radioactive taurine at low concentrations (0.5μM) from Krebs phosphate medium. This work adds support to the suggestion from iontophoretic evidence (Curtis & Watkins, 1965) that taurine may be considered as a possible neurotransmitter, but much further work will be necessary to verify this hypothesis.

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Rapid Oxidation of Chondroitin Sulphates and other Glycols by Periodic Acid in Organic Solvents

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Oxidation of chondroitin sulphates, hyaluronate etc. by periodate in aqueous solution is slow but specific (Scott, 1968). Presumably periodate must interact with hydroxyl groups of the substrate, no matter what the detailed mechanism, and one would therefore expect that hydroxyl groups in the solvent would compete with oxidizable glycols for periodate, thus diminishing oxidation rates. Since hydroxylic solvents would also be expected to associate with hydroxyl groups of the substrate, this should also impede periodate–glycol interaction. On these arguments, water should be a particularly unfavourable solvent in which to use periodate as a glycol-splitting reagent. Using pinacol, a 'hindered' glycol that is slowly oxidized in water, we have investigated solvents in which the 'hydroxyl group concentration' is lowered or zero. Wherever possible, solvents were spectroscopic, A.R. or redistilled, as indicated. Consumption of periodate was followed at 222nm.

Rates of oxidation were very greatly increased (several thousandfold) in dioxan (spectroscopic). Inclusion of as little as 10% (v/v) of water in the dioxan drastically lowered the oxidation rate. Propan-2-ol (A.R.) and 2-methylpropan-2-ol, with 'hindered' hydroxyl groups, were effective solvents, allowing several hundredfold increases in rate as compared with water. Trifluoroethanol (spectroscopic) and methoxyethanol (redistilled), in which electron-attracting groups are adjacent to the hydroxyl group, also permit rapid oxidation, in general agreement with the concept that hydroxyl groups with increased electron availability at the oxygen atom interact better with periodate (Buist, Bunton & Miles, 1957). The results suggest that an unhindered, highly basic, oxygen atom in the solvent molecule is associated with a low rate of oxidation by periodate, presumably because of possible hydrogen-bonding to the glycol, and strong periodate–oxygen atom interaction. Thus in both *NN*-dimethylformamide and dimethyl sulphoxide oxidation is slow, whereas in sulpholane (a poor hydrogen acceptor) (Parker, 1962) oxidation is quite rapid.

Whereas many hours' oxidation in aqueous solution are needed to produce sufficient aldehyde groups in chondroitin sulphate and other polyuronides (Scott, 1968), a strong Schiff reaction is given by oxidizing them on glass-fibre paper within 1min in dioxan. Keratan sulphate and commercial heparin do not react, as would be expected from the formulae, and can thus be easily distinguished. It is possible that the very rapid oxidation observed in organic solvents may avoid the side reactions, e.g. overoxidation, and degradation that are a consequence of lengthy reaction times in aqueous solvents.

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