decreased type I and type II hydrogen approximately equally. 11-Deoxy-17-hydroxycorticosterone, progesterone and testosterone decreased type I hydrogen, but had hardly any effect on type II hydrogen.

Such analyses may provide a means for demonstrating how steroids can modify the production of tissue hydrogen. The present results support the notion of two types of NADPH hydrogen, since each type can be affected differently.

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Differential Effects of Reduced Nicotinamide-Adenine Dinucleotide Phosphate on Cytoplasmic-Mitochondrial Interactions

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The study of interactions between oxidation in the cytoplasm and the mitochondria is facilitated by using intact tissue sections. It was shown (Butcher, 1969) that mitochondrial hydrogen transport from the oxidation of succinate is greatly enhanced if NADPH is oxidized simultaneously in the cytoplasm. The NADPH may be generated in the sections by the addition of NADP⁺ and either glucose 6-phosphate or 6-phosphogluconate, or it may be added exogenously.

Sections of rat liver (10 μ m) were incubated at 37°C for 30 min in air in media containing neotetrazolium (as hydrogen acceptor) and polyvinyl alcohol (20%, w/v, as stabilizer) in glycylglycine buffer, pH 8.0. To some media (a) only succinate was added; to others (b) a source of NADPH; others received both these components to test whether the resultant activity was greater than the sum of (a) and (b) tested separately. The same tests were done in the presence of either pregnenolone or cortisone (0.1 mM). Pregnenolone decreased the activity caused by oxidation of glucose 6-phosphate by about 30% but had no effect on that found when 6-phosphogluconate was the substrate. Cortisone had no inhibitory effect. Neither of these steroids affected the simple oxidation of succinate. Moreover, the inhibition of glucose 6-phosphate dehydrogenase activity, although small, resulted in 90–100% inhibition of the enhanced mitochondrial hydrogen transport, i.e. the interaction between

oxidation in the cytoplasm and the mitochondria. In contrast, decreasing the glucose 6-phosphate oxidation in the absence of pregnenolone had little effect.

The amount of hydrogen produced from glucose 6-phosphate oxidation in the presence of phenazine methosulphate, i.e. type I+type II hydrogen as described by Altmann & Chayen (1970), was diminished by pregnenolone to give the same value as was obtained in the absence of phenazine methosulphate (type I hydrogen). It seems possible that pregnenolone could be inhibiting type II hydrogen generated from glucose 6-phosphate dehydrogenation specifically and that it is this fraction of hydrogen, or of NADPH, that is responsible for the cytoplasmic-mitochondrial interactions. Alternatively the interaction could require NADPH formed from 6-phosphogluconate dehydrogenation, or added exogenously, and that pregnenolone is inhibiting the formation of 6-phosphogluconate from glucose 6-phosphate (Altmann & Robertson, 1970). Either way these results strongly suggest that NADPH is not treated as a single entity in tissues.

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Inhibition of Tryptophan Oxygenase in Rat Liver By Steroid Hormones

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Pregnant women and normal human subjects treated with oestrogens show evidence of an increased capacity for the conversion of L-tryptophan into nicotinic acid ribonucleotide. This has been attributed to an increased activity of liver tryptophan oxygenase (EC 1.13.1.12) brought about either by the direct action of oestrogens, or indirectly via elevated corticosteroid concentrations (Rose, 1966, 1969). Tryptophan oxygenase activity is elevated in the liver of pregnant rats (Auricchio, Rigillo & Di Toro, 1960), but treatment with a combination of oestrone and progesterone failed to produce an increase in the activity of the enzyme (Greengard, Kalinsky & Manning, 1968). In a further study tryptophan oxygenase activity was determined in supernatant fractions prepared from