medium of T (Texas) but not of N (Normal) mitochondria, a study was initiated to investigate the various details of the pathways of electron transport and associated activities in N and T mitochondria. On the basis of the details of the pathways in Jerusalem artichoke mitochondria developed by Coleman and Palmer (1972, Eur. J. of Biochemistry 26:499), the effects of the race-T pathotoxin on various steps in this network of enzyme reactions was investigated. The race T pathotoxin causes an increase in the activity of cytochrome oxidase and succinate cytochromic reductase, possibly due to a disturbance of the mitochondrial membranes which allows increased substrate accessibility acting as an uncoupler.

The first ATP-coupled site of the electron transport chain, which includes the endogenous NADH dehydrogenase, was studied using malate as a substrate in the absence of exogenous NAD^+. In T mitochondria, the pathotoxin strongly inhibited the oxidation of malate by intact mitochondria. Malate oxidation via endogenous NADH dehydrogenase in N mitochondria was unaffected by similar concentrations of pathotoxin. Upon the addition of NAD^+, however, there is a marked stimulation of malate oxidation in intact T mitochondria. Thus, the presence of an intermembrane malate dehydrogenase activity coupled to NAD^+ reduction leads to an initial and immediate stimulation of malate oxidation via the exogenous NADH dehydrogenase. This confirms that the inhibition of malate and oxoglutarate oxidation in T mitochondria by pathotoxin is almost certainly at the endogenous NADH dehydrogenase complex of the inner membrane.

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4. Location of pg^m of the En system.

pg^m (Peterson, 1960 Genetics 45:115) has been found to be allelic with a pg^m isolated by Neuffer in mutagen treatments. This is uncovered by TB-3b, which places pg^m on chromosome 3S.

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