

**Lanoue K, J Watts, C Koch. Adenine nucleotide transport during cardiac ischemia. *Am J Physiol* 1981;24:H663-H671.**

The suggestion that long-chain acyl coenzyme A (CoA) derivatives may inhibit mitochondrial adenine nucleotide transport in heart cells during ischemia has been reevaluated. The effectiveness of media palmitoyl-CoA as an inhibitor is a function of mitochondrial protein and media adenine nucleotide concentrations. Extrapolation to the protein and adenine nucleotide levels of the cardiac cell suggest that physiological concentrations of cytosolic long-chain acyl-CoA would not inhibit adenosine 5'-triphosphate (ATP) transport. Palmitoyl-CoA was varied in the mitochondrial matrix by incubating the isolated mitochondria with and without palmitoyl carnitine. Intramitochondrial nucleotides were depleted by incubating the isolated mitochondria for various periods of time with arsenite and phosphate. Even at low substrate (matrix ATP) concentrations, no palmitoyl-CoA inhibition of ATP transport could be demonstrated. Further experiments showed that endogenous nucleotide levels are significantly depleted in mitochondria isolated from hearts made ischemic for 30-90 min. Since mitochondrial adenine nucleotide transport occurs by an exchange mechanism, this depletion of the internal pool of nucleotides from ischemic heart mitochondria may result in an irreversible diminution of ATP transport.

