

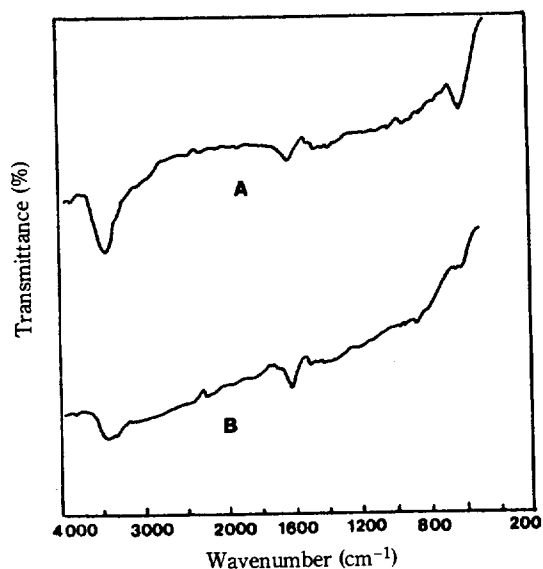


Figure 2. IR spectra of iodine-doped poly(dipropargyl Ether) [A, $(C_6H_5O)_1(I_2)_{0.30}$] and poly(dipropargyl sulfide) [B, $(C_6H_5S)_1(I_2)_{0.29}$]

tron transfer from polymer chain to the dopant.⁹ For iodine doped PDPE and PDPS, the infrared spectra show no C-I stretching bands in their normal region ($465-600\text{ cm}^{-1}$).¹⁰ This indicates that iodine atoms are not attached to carbon atoms by normal sigma bonds. This finding seems to be consistent with the results reported in the literature for bromine- and chlorine-doped poly(phenylacetylene).¹¹

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External NADH Oxidation of Mitochondrion Produces More Heat

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In the preface for the report¹ by one communicator of this communication, he stated that some people suffer a mild fever by the oral intake of ginseng roots. But he did not explain the cause of the fever broadly. It is supposed that a component of ginseng makes an organelle produce heat. Cold exposure in cold-adapted rats activated the pathway of external electron transport of mitochondrion for exogenous NADH oxidation² probably for heat production. This pathway donate its electron to the electron shuttle of cytochrome *c* in the intermembrane space of the organelle³⁻⁵. Because we could not find a report of measuring these heat production, we intended to measure the heat production or temperature elevation *in vitro* in the mitochondrial electron-transport systems (the ex-

ternal transport *via* cytochrome *c* and internal transport) and to compare them.

Mitochondria were separated from bovine heart by the method of Smith⁶ and quantitative analysis⁷ of the organelle was in terms of protein. The activity of the mitochondrion was determined in terms of absorption decrease of added NADH at 340 nm. The mitochondria were used by dispersing them in a isotonic buffer⁸-0.225M sucrose, 10 mM sodium phosphate, 5 mM $MgCl_2$, 20 mM KCl, 20 mM triethanolamine (pH 7.4). The final concentration of NADH in the reaction systems was $1.5 \times 10^{-4}M$.

Ginseng saponin was separated after Namba⁹ and its concentration in the reaction system was from 1.5×10^{-7}

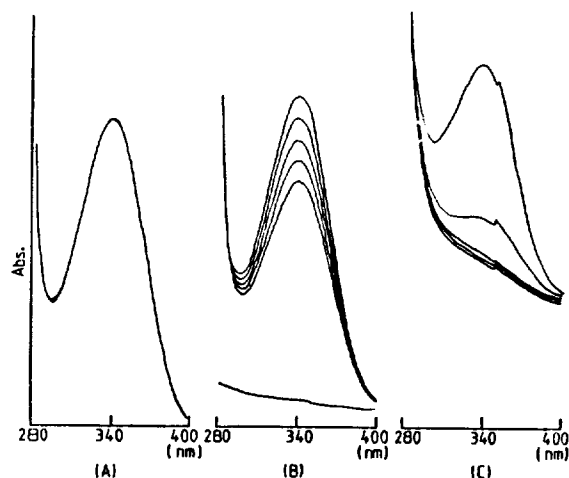


Figure 1. NADH oxidation in NADH- O_2 system without mitochondria (A) and in NADH- O_2 system with mitochondria. (B: 20 μ g/ml mitochondria, C: 100 μ g/ml mitochondria)

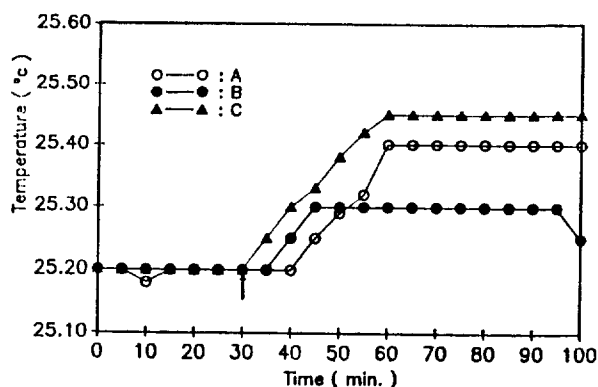


Figure 2. Time course of temperature change in the systems of intact mitochondrion conducting NADH oxidation. (A) external transport. (B) internal transport (shuttle system). (C) external transport with ginseng saponin. ($\uparrow$) expresses the time of adding substrates after the incubation of mitochondria at 25.2 $^{\circ}$ C.

through 1.5×10^{-6} M.

The contact of NADH to mitochondrial inner membrane for the internal transport was made with the assistance of malate-citrate shuttle system.¹⁰ ADP was added to both systems — external (*via* outer membrane to cytochrome oxidase) and internal (*via* inner membrane only) ones — for the phosphorylation with phosphate in the isotonic buffer.

The glass test tubes containing the reaction systems were placed inside the holes of polystyrene for insulation at the

room temperature of 25 $^{\circ}$ C.

The activity of the mitochondria in terms of the absorption of NADH assayed at the intervals of two minutes was increased with the increase of mitochondrial concentration for the external pathway (Figure 1). The system for this pathway showed increase in its temperature from 25.2 $^{\circ}$ C to 25.4 $^{\circ}$ C on adding NADH. Ten minutes was required for this elevation of temperature. The temperature increase for the internal system was 0.1 $^{\circ}$ C by 10 minutes. This temperature increase was definitely lower than the external system. Addition of 1.6×10^{-5} M ginseng saponin for the external system resulted in higher temperature elevation of 0.25 $^{\circ}$ C for the oxidation of NADH (Figure 2).

External electron transport does produce more heat than internal electron transport. The production of more heat is in short because of electron transport with less ATP synthesis. Ginseng saponin does facilitate the external transport. This facilitation might be from the elevation of the mobility of intermembranous cytochrome *c* of mitochondrion. Present result might not be parallel with the case *in vivo*; but a possibility of parallelism exists.

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