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(spin trapping/lipid peroxidation)

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sured by superoxide dismutase-inhibitable reduction of 30

Table 1. Oxygen consumption by endothelial cell suspensions

μM succinoylated cytochrome *c*, monitored at 550 nm (ε_{max} = 21 $mM^{-1}cm^{-1}$) according to the method of Kuthan et al.

nmol O_2 consumed ·

An EPR spectrum characteristic of DMPO-OH (Fig. 1) might suggest that hydroxyl radical was produced by menadione-treated endothelial cells. However, this is probably not the case, since DMPO-OOH rapidly decomposes into

droxyl radical (21, 22). Approximately 3% of DMPO-OH arises via this latter pathway (22). Confirmation that DMPO-

vielding an autoxidizable semiquinone (25). Dicoumarol inhibition of the two-electron reduction of menadione by DT-diaphorase [NAD(P)H:(quinone-acceptor)oxidoreductase (EC 1.6.99.2); see ref. 25] diminishes extracellular superox-

hydroxynaphthalene available for extracellular diffusion and subsequent reduction of menadione to the semiquinone.

Table 2. Reduction of succinylated cytochrome *c* by endothelial

droxynanthalene available for extracellular diffusion (Table

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