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Detection of mitochondrial electron chain carrier redox status by transhepatic light intensity during rat liver reperfusion.

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The aim of the study was to investigate mitochondrial electron transfer during rat liver reperfusion after cold storage and hypothermic machine perfusion. Livers from male Brown Norway rats were preserved (UW) for 10h either by cold storage (CS) or by hypothermic oxygenated perfusion extracorporeal (HOPE). Transhepatic photometric analysis allowed determination of the redox status of mitochondrial cytochromes during preservation, rewarming and reperfusion. Mitochondrial electron chain carriers were inhibited at different sites with rotenone and cyanide in some experiments. reversed transcriptional polymerase chain reaction (RT-PCR) was performed after reperfusion concerning transcription of TNFalpha, caspase 9, and c-jun kinase (JNK). Increased superoxide anion formation as well as transcription of TNFalpha, caspase 9, and JNK during reperfusion after cold storage (CS) were related with completely reduced cytochromes before and during reperfusion. In contrast, hypothermic oxygenated livers (HOPE) showed oxygenated cytochromes as well as decreased superoxide anion formation and no detectable transcription of TNFalpha, caspase 9, and JNK. A similar low level of superoxide anion formation was found when electron chain transfer of cold stored livers was inhibited during reperfusion with rotenone but not with cyanide. After hypothermic oxygenation (HOPE) inhibition of mitochondrial electron chain with rotenone showed no change in formation of superoxide anion formation whereas inhibition with cyanide showed increased superoxide anion formation. Thus mitochondrial cytochrome redox status is suggested to be related: (i) with the release of reactive oxygen substances as well as (ii) with the expressions of TNFalpha, caspase 9, and JNK during reperfusion and may thus be usable as predictive marker of liver grafts.

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