



Over-reduction of mitochondrial complex I during brain ischemia results in a dissociation of reduced flavin (FMNH₂) from the enzyme. Reperfusion-induced reoxidation of FMNH₂ by molecular oxygen is associated with generation of ROS and contributes to oxidative stress in the mitochondrial matrix. Recovery of complex I function at the early stage of reperfusion is followed by oxidation of critical thiol residue(s) at later stages.