

Nitroxide Tempo Accelerates Repair of Soft Palate Surgery

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Reduction or cessation of nutritive blood flow to tissue, with subsequent hypoxia, leads to cellular damage. Cyclic nitroxides are stable free radicals, protecting against oxidative damage in several cellular models. The cell-permeable compound 2,2,6,6-tetramethylpiperidine-1-oxyl (Tempo) was used to investigate the action of nitroxides on the oxidative status and tissue regeneration after palatal surgery. Male Wistar rats were submitted to soft palate surgery, with release of tissue through a punch scalpel. Animals received a unique dose of Tempo by intraperitoneal (10 mg/body weight) or topic administration (0.1% m/v) during 3 consecutive days. Control groups were treated with vehicles in identical conditions. At 3rd, 6th, or 12th days, palatal tissue was removed to macroscopic and histological analyses. Lipid oxidative status was determined by HPLC through malonaldehyde (MDA) measuring, using a fluorescence detector (λ_{ex} 532nm, λ_{em} 553nm). The content of reduced (GSH) and oxidized (GSSG) glutathione, was assessed after derivatization with monobromobimane (λ_{ex} 360nm, λ_{em} 470nm), and HPLC separation without or with previous glutathione reductase and NADPH incubation. Protein carbonyl groups were determined by reactions with 2,4-dinitrophenylhydrazine ($\lambda_{370\text{nm}}$: 22.000 M⁻¹cm⁻¹). Macroscopic and histological analyses showed that after 3 and 6 days after surgery, Tempo ameliorates palate laceration. MDA and carbonyl groups levels were significantly lower in nitroxide-treated groups than in respective controls, particularly in animals subject to topical treatment. GSH contents were minimally augmented in animals exposed to both Tempo administrations. However, at 12th day, no difference was found between groups. These results suggested that Tempo may be used to accelerate surgery cicatrization and to control oxidative stress due to isque mic-reperfusion injury. Supported by: UNIFAL-MG, CNPq-INCT Redoxoma, Fapemig, PET/SESu.

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