Reactive oxygen species

Intracellular redox homeostasis is crucial for a series of physiological processes. Reactive oxygen species (ROS) play important roles in redox processes. ROS can maintain cell reproduction and survival at moderate levels while promoting the initiation and progression of tumors at high levels.

Reactive oxygen species (ROS) are chemically reactive molecules containing oxygen. Examples include peroxides. superoxide, hydroxyl radical, and singlet oxygen. ROS are formed as a natural byproduct of the normal metabolism of oxygen and have important roles in cell signaling and homeostasis.

However, during times of environmental stress (e.g., UV or heat exposure), ROS levels can increase dramatically.

This may result in significant damage to cell structures. Cumulatively, this is known as oxidative stress. ROS are also generated by exogenous sources such as ionizing radiation.

Shan et al. established a gene signature associated with ROS to explore its influence on prognosis and immune microenvironment in gliomas.

The ROS-related gene expression profile dichotomized patients into two groups with different clinicopathological features and prognoses. A 19-gene ROS-related signature was used to robustly predict prognosis in both training and validation datasets. Functional analysis indicated an association between ROS levels and the immune microenvironment. The expression of immune checkpoints and M2-type markers was upregulated in the high-risk group, which suggested the immunosuppressive function of ROS.

ROS-related signature is an independent glioma prognosis factor and could potentially exert immunosuppressive effects on the tumor microenvironment [1].

Traumatic brain injury (TBI) is accompanied by the overload of reactive oxygen species (ROS), which can result in secondary brain injury. Although procyanidins (PCs) have a powerful free radical scavenging capability and have been widely studied in the traumatic brain injury treatment, conventional systemic drug therapy cannot make the drug reach the targeted area in the early stage of TBI and will cause systemic side effects because of the presence of the blood-brain barrier (BBB). To address this issue, they designed and fabricated a ROS-scavenging functional hydrogel-loaded PC (GelMA-PPS/PC) to deliver the drug by responding to the traumatic microenvironment. In situ injection of the GelMA-PPS/PC hydrogel effectively avoided the BBB and was directly applied to the surface of brain tissue to target the traumatic area. Hydrophobic poly(propylene sulfide)60 (PPS60), a ROS quencher and H2O2-responsive substance, was covalently bound to GelMA and exposed in response to the trauma microenvironment. At the same time, the H2O2 response of PPS60 further caused the structure of the hydrogel to degrade and release the encapsulated PC. Then PC could regulate the oxidative stress response in the cells and synergistically deplete ROS to play a neurotrophic protective role. This work suggests a novel method for secondary brain injury treatment by inhibiting

the oxidative stress response after TBI [2].

The signaling of reactive oxygen species (ROS) is essential for the maintenance of normal cellular function. However, whether and how ROS regulate stem cells are unclear.

Zhao et al. demonstrate that, in transgenic mice expressing the human manganese superoxide dismutase (MnSOD) gene, a scavenger of ROS in mitochondria, the number and function of mouse hematopoietic stem/progenitor cells (HSPC) under physiological conditions are enhanced. Importantly, giving MnTnBuOE-2-PyP5+(MnP), a redox- active MnSOD mimetic, to mouse primary bone marrow cells or to C57B/L6 mice significantly enhances the number of HSPCs. Mechanistically, MnP reduces superoxide to hydrogen peroxide, which activates intracellular Nrf2 signaling leading to the induction of antioxidant enzymes, including MnSOD and catalase, and mitochondrial uncoupling protein 3. The results reveal a novel role of ROS signaling in regulating stem cell function, and suggest a possible beneficial effect of MnP in treating pathological bone marrow cell loss and in increasing stem cell population for bone marrow transplantation [3].

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