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高压氧对疲劳大鼠肝损伤的保护作用 [点击下载全文](#)

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摘要:

**摘要目的:** 探讨高压氧暴露对疲劳大鼠肝脏损伤的保护作用及机制。**方法:** 4周龄健康雄性(Sprague Dawley, SD)大鼠30只, 随机分为3组: 对照组、疲劳模型组、疲劳高压氧暴露组, 每组10只。建立大鼠游泳致力竭疲劳模型并通过高压氧暴露进行恢复; 8周实验后取每只大鼠两块肝脏组织, 其中一块取每只大鼠的肝脏相同部位, 光镜观察肝脏组织的病理变化; 另一块取肝右叶5g, 制备成10%的肝组织匀浆, 检测匀浆液的丙二醛(MDA)、超氧化物歧化酶(SOD)含量的变化。结果: ①疲劳高压氧暴露组大鼠游泳时间明显长于疲劳模型组( $P < 0.05$ )。②对照组、疲劳模型组和疲劳高压氧暴露组大鼠肝脏组织MDA含量存在显著性差异( $P < 0.01$ ), 进一步两两比较显示, 疲劳模型组MDA含量显著高于其他两组( $P < 0.01$ ); 并且三组肝脏组织SOD活性存在显著性差异( $P < 0.01$ ), 进一步两两比较发现, 疲劳模型组SOD活性显著低于其他两组( $P < 0.01$ )。③对照组肝脏组织结构正常, 疲劳模型组肝索断裂明显, 肝窦变窄, 中央静脉结构紊乱, 肝小叶内轮廓不清, 肝细胞肿胀, 核变大, 胞浆空淡明显可见, 部分肝小叶中出现散在的点灶状坏死肝细胞, 经高压氧治疗后, 疲劳高压氧暴露组中央静脉仍然扩张并伴有淤血, 但结构完整, 肝索断裂呈散在分布, 较疲劳模型组稍有缓解。结论: 高压氧暴露能有效加长大鼠游泳时间, 降低疲劳大鼠肝脏MDA含量, 提高SOD活性, 改善肝脏组织的病理变化, 从而对疲劳大鼠肝脏损伤起到保护作用。

**关键词:** [运动疲劳](#) [高压氧](#) [肝脏](#) [丙二醛](#) [超氧化物歧化酶](#)

A study on the protective role of hyperbaric oxygen on liver injury in fatigue rats [Download Fulltext](#)

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Fund Project:

Abstract:

**Abstract Objective:** To explore the protective role and mechanism of hyperbaric oxygen (HBO) exposure on liver injury in fatigue rats. **Method:** Thirty healthy male SD rats at 4 weeks of age, were randomly divided into 3 groups: control group, fatigue model group, fatigue model HBO exposure group, each group of 10. Rat models of exhaust swimming to fatigue were established and applied to HBO exposure. After 8 week experiment two blocks of liver tissue were taken from each rat. Of which one block was taken from the same part of rats' livers and pathological changes of liver tissues were observed under light microscopic. The other block was taken from the right lobe of liver and was prepared for 10% liver homogenate to detect the content of MDA and the activity of SOD. **Result:** ①The swimming time of rats in HBO exposure group was significantly longer than that in fatigue model group ( $P < 0.05$ ); ②The content of MDA of rats liver tissue in three groups had significant differences ( $P < 0.01$ ). Further comparison showed that the content of MDA in fatigue model group was significantly higher than that in the other two groups ( $P < 0.01$ ). The activity of SOD in three groups had also significant differences ( $P < 0.01$ ), and activity of SOD in fatigue model group was also significantly lower than that in the other two groups ( $P < 0.01$ ); ③The hepatic tissue structure was normal in control group, while in fatigue model group, the hepatic cord ruptured, hepatic sinusoidal narrowed, central venous disordered, hepatic lobule contour were not clear, liver cell swelled, nuclear enlarged, cytoplasm became pale obviously, and scattering points of focal necrosis of liver cells appeared in a part of liver lobules. After HBO therapy, although central venous still dilated and associated with congestion, the structures remained intact and hepatic cord rupture were scattered. So morphological structure of hepatic tissue in HBO group improved compared with the fatigue model group. **Conclusion:** HBO exposure could effectively lengthen the rat's swimming time, reduce the content of MDA and elevate the activity of SOD of fatigue rat's liver, improve pathological changes of liver tissue. This proved that HBO exposure could effectively protect the damaged liver of fatigue rat.

**Keywords:** [exercise-induced fatigue](#) [hyperbaric oxygen](#) [liver](#) [malondialdehyde](#) [superoxide dismutase](#)

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