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论著

人HepG2细胞低氧习服模型的建立

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摘要 目的：建立人HepG2细胞的低氧习服模型，以探讨细胞低氧习服的机制。 方法： HepG2细胞在1% O2低氧条件下培养24 h后，正常氧压条件下培养24 h，以此为一个周期，连续低氧处理6个周期，期间以MTT法、透射电镜观察法、Northern杂交检测细胞达到低氧习服后，细胞增殖能力、细胞超微结构以及细胞内EPO基因表达的变化。 结果： HepG2细胞在1%O2低氧条件下培养48 h后，细胞增殖受到抑制，超微结构受到损伤；而经过6周期的间断低氧处理后，再低氧48 h，细胞的增殖能力恢复到常氧对照组水平，提示细胞耐氧能力明显升高，细胞的形态、胞浆突起数量、线粒体数目和结构都接近于常氧对照组水平。急性低氧能够引起HepG2细胞内EPO基因表达增强，而低氧习服的HepG2细胞再低氧48 h，EPO基因表达量接近到常氧对照细胞水平。结论： HepG2细胞经过6个周期低氧处理后，细胞耐低氧能力增强，急性低氧促进EPO基因表达的作用受到抑制，细胞达到低氧习服状态。

关键词 缺氧； 低氧习服； 红细胞生成素； HepG2细胞

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Establishment of a cellular model of hypoxic acclimatization in human HepG2 cells

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Abstract

<FONT face=Verdana>AIM: To establish a cellular model of hypoxic acclimatization using HepG2 cells to explore the mechanism of cellular hypoxia acclimatization. METHODS: HepG2 cells were cultured in 1% O2 for 24 hours, then in 21% O2 for another 24 hours, which composed a hypoxic treating cycle. After 6 cycles, the activity of cell proliferation was estimated with MTT method. The morphologic features of HepG2 cells were observed with optical microscope and transmission electron microscope. EPO gene expression was detected by Northern blotting technique. RESULTS: Acute hypoxia inhibited cellular mitosis, impaired cellular ultrastructure and induced EPO gene expression. After 6 cycles of hypoxic treatment, proliferation ability of HepG2 treated with acute hypoxia for 48 h was resumed to the level of control cells cultured in 21% O2. The ultrastructure of HepG2 cells injured by hypoxia recovered and the level of EPO gene expression returned to that in control cells. CONCLUSION: After 6 cycles of hypoxic treatment, the ability of HepG2 to endure hypoxia is obviously enhanced and HepG2 cells might reach the status of hypoxic acclimatization.</FONT>

Key words Anoxia Hypoxic acclimatization Erythropoietin HepG2 cells

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