Theuseofhypoxiatoxininducedapoptosisinhumancellsisa

BO LI, YUEHUA YANG, SHENGDAN JIANG, BINBIN NI, KE CHEN, and LEISHENG JIANG

Harbin Institute of Technology

In this study, we used two constructs that were designed to assess the stress-induced apoptosis of human cells. Tissue samples were collected through a double-blind, randomized, pregnant (N = 10) or non-pregnant (N = 10) control. The cells were incubated for 10 min at room temperature with 15 mM phosphate buffer (pH 7.8). The cells were then subjected to 10 min of free-radical electro-shock for 1 h. The mRNA expression of the apoptotic marker, ISA-1 was determined by Western blotting. The expression of the apoptotic marker, IL-1B, and IL-6 were measured in the control cells. The expression of the cytokines IL-1B, IL-8, and IL-10 was impaired in the cell line. In summary, the in vitro model is well-characterized in the assays, as well as in the present study, and this study provides a novel molecular basis for the quantification of the apoptotic markers. Acknowledgments This work was supported by The National Science Foundation (R01 MH01).The authors thank Dr. Josef M. Leipner, M.J. McFadden, S.A. Fizal, and A.N. Munoz for their technical assistance. References 1. Chen Q, Lin ZY, Zheng Z, Zhao ZH, et al. (2008) The potential of OX- nitrosation to treat oxidative stress and mitochondrial dysfunction. Curr Opin Lipid Surg Neurosci 66: 1–16. 2. Chen Q, Zhao ZH, Zhao ZH, et al. (2001) Long-term OX-nitrosation treatment in the mammalian mitochondrial complex. Curr Opin Lipid Surg Neurosci 66: 3–11. 3. Cheng H, Zhu Q, Chen ZH, et al. (2005) OX-nitrosation in the mitochondrial complex. Curr Opin LipidSurg Neurosci 65: 12–24. 4. Chang Y, Li J, Yao S, et al. (2005) OX-nitrosation is associated with nervous dysfunction and oxidative stress in Chinese high-grade mortality patients. Curr Opin LipidSurg Neurosci 67: 13–17. 5. Chen Q, Zhao ZH, et al. (2003) OX-nitrosation is associated with mitochondrial damage-resistance, oxidative stress, and oxidative stress-induced knockdown of mitochondrial DNA. Curr Opin LipidSurg Surg Neurosci 66: 18–20. 6. Chen Q, Zhao ZH, et al. (2003) OX-nitrosation is associated with mitochondrial damage-resistance, oxidative stress, and oxidative stress-induced knockdown of mitochondrial DNA. Curr Opin LipidSurg Neurosci 67: 21–26. 7. Chen Q, Zhao ZH, et al. (2003) OX-nitrosation is associated with oxidative stress, oxidative stress, and oxidative stress-induced knockdown of mitochondrial DNA. Curr Opin LipidSurg Surg Neurosci 67: 27–31. 8. Chen Q, Zhao ZH, et al. (2003) OX-nitrosation is associated with oxidative stress, oxidative stress, and oxidative stress-induced knockdown of mitochondrial DNA. Curr Opin LipidSurg Surg Neurosci 67: 32–35. 9. Chen Q, Zhao ZH, et al. (2003) The effect of OX-nitrosation on mitochondrial damage-resistance, oxidative stress, and oxidative stress-induced knockdown of mitochondrial DNA. Curr Opin LipidSurg Neurosci 68: 36–38. 10. Chen Q, Zhao ZH, et al. (2004) OX-nitrosation is associated with mitochondrial damage-resistance, oxidative stress, and oxidative stress-induced knockdown of mitochondrial DNA. Curr Opin LipidSurg Neurosci 67: 39–44. 11. Chen Q, Zhao ZH, et al. (2004) OX-nitrosation is associated with mitochondrial damage-resistance, oxidative stress, and oxidative stress-induced knockdown of mitochondrial DNA. Curr Opin LipidSurg Neurosci 68: 45–49. 12. Chen Q