Cytochalasin E, a Potential Agent for Anti-Glioma Therapy, Efficiently Induces U87 Human Glioblastoma Cell Death

Junyang LI, Bing GU, Gong CHEN, Banyou MA, Jing XU, Guofeng ZHANG, Dong WEI, Peiyuan GU, Biao XING, Meng LI & Weixing HU *

Department of Neurosurgery, The First Affiliated Hospital of Nanjing Medical University, 300 Guangzhou Road, Nanjing, 210029, P.R.China.

SUMMARY. Glioblastoma is one of the most malignant brain tumors. Current treatments for glioblastoma usually make poor responses, and novel treatment strategies are extremely imperative. Cytochalasin E was reported to inhibit angiogenesis and tumor growth in some studies, but its effects on gliomas are still unknown. In this study, we found cytochalasin E inhibits U87 human glioblastoma cell growth in a very low concentration range of 10^{-8} to 10^{-6} M in a time and concentration dependent manner, and the IC50 were 1.17 \pm 0.32 \times 10^{-7} M for 48 h treatment, 6.65 \pm 1.12 \times 10^{-8} M for 72 h and 3.78 \pm 1.30 \times 10^{-8} M for 96 h. We also found cytochalasin E induces cell-cycle G2/M phase arrest (72 h-treatment of 10^{-6} M cytochalasin E caused 56.2 \pm 6.1 \% cells arrest in G2/M phase) and cell apoptosis (96 h-treatment of 10^{-6} M cytochalasin E induced 24.1 \pm 4.2 \% cells apoptosis). Thus, cytochalasin E is proposed as a potential agent for glioblastoma chemotherapy.

KEY WORDS: Actin, apoptosis, Cytochalasin E, Glioma, Proliferation.

* Author to whom correspondence should be addressed. E-mail: hwx66@126.com