Thymosin beta-4 up-regulates anti-oxidative enzymes and protects human cornea epithelial cells against oxidative damage

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摘要

Abstract

BACKGROUND: The ability to scavenge reactive oxygen species (ROS) is crucial for cornea epithelial cells to resist oxidative damage. The authors previously demonstrated that exogenous thymosin beta-4 (T beta(4)) was able to protect human cornea epithelial (HCE-T) cells against H(2)O(2)-induced oxidative damage, and its cellular internalisation was essential. The aim of this study is to further elucidate its protective mechanism. METHODS: HCE-T cells with or without T beta(4) pretreatment were exposed to H(2)O(2), and the differences in caspase activity, intracellular ROS levels, cell viability, and the expression of anti-oxidative enzymes, were measured and compared. RESULTS: Besides reducing caspase-9 activation and intracellular ROS levels induced by H(2)O(2), treatment of T beta(4) could also increase cell viability and stimulate the expression of manganese superoxide dismutase (SOD) and copper/zinc SOD. Moreover, both transcription and translation levels of catalase were also upregulated by T beta(4) in the presence of exogenous H(2)O(2). Furthermore, it was demonstrated that the addition of catalase inhibitor abrogated the protective effect of T beta(4) against H(2)O(2)-induced oxidative damage. CONCLUSION: To the best of the authors' knowledge, this is the first report to show that T beta(4)was capable of upregulating anti-oxidative enzymes in human corneal epithelial cells, and these findings further support its role in cornea protection