Analysis of expression of vitamin E-binding proteins in H2O2 induced SK-N-SH neuronal cells supplemented with α-tocopherol and tocotrienol-rich fraction

ABSTRACT

Natural α-tocopherol (α-TCP), but not tocotrienol, is preferentially retained in the human body. α-Tocopherol transfer protein (α-TTP) is responsible for binding α-TCP for cellular uptake and has high affinity and specificity for α-TCP but not α-tocotrienol. The purpose of this study was to examine the modification of α-TTP together with other related vitamin E-binding genes (i.e., TTPA, SEC14L2, and PI-TPNA) in regulating vitamin E uptake in neuronal cells at rest and under oxidative stress. Oxidative stress was induced with H2O2 for an hour which was followed by supplementation with different ratios of α-TCP and tocotrienol-rich fraction (TRF) for four hours. The cellular levels of vitamin E were quantified to determine bioavailability at cellular levels. The expression levels of TTPA, SEC14L2, and PI-TPNA genes in 0% α-TCP were found to be positively correlated with the levels of vitamin E in resting neuronal cells. In addition, the regulation of all the above-mentioned genes affect the distribution of vitamin E in the neuronal cells. It was observed that, increased levels of α-TCP secretion occur under oxidative stress. Thus, our results showed that in conclusion vitamin E-binding proteins may be modified in the absence of α-TCP to produce tocotrienols (TCT), as a source of vitamin E. The current study suggests that the expression levels of vitamin E transport proteins may influence the cellular concentrations of vitamin E levels in the neuronal cells.