Supplementary Materials: Tocotrienol Affects Oxidative Stress, Cholesterol Homeostasis and the Amyloidogenic Pathway in Neuroblastoma Cells: Consequences for Alzheimer’s Disease

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Figure S1. No significant effect of α-tocopherol and α-tocotrienol on cell viability measured by LDH activity in medium (cytotoxicity solvent control: 1.8% ± 0.1%; cytotoxicity α-tocopherol: 2.5% ± 0.3%, p = 0.22 compared to the solvent, n = 4; cytotoxicity α-tocotrienol: 2.3% ± 0.2%, p = 0.41 compared to the solvent, p = 0.86 compared to α-tocopherol, n = 4). Statistical significance as described for Figure 1 (n.s., not significant).

Figure S2. Increased secretion of sAPPβ (136.8% ± 3.3%, p = 0.004, n = 3) by SH-SY5Y APP695 transfected cells after incubation with α-tocotrienol. Statistical significance as described for Figure 3 (** p ≤ 0.01).
**Figure S3.** Enhanced production of Aβ (133.6% ± 2.8%, p ≤ 0.001, n = 12) by SH-SY5Y C99 transfected cells after incubation with α-tocotrienol. 100% in the control cells corresponds to 10.0 ng/mL total Aβ. Statistical significance as described for Figure 3 (**p ≤ 0.001**).

**Figure S4.** Unchanged gene expression of the components of the β- and γ-secretase measured by real-time PCR. BACE1 = β-site APP cleaving enzyme 1: 93.2% ± 4.3%, p = 0.140, n = 8; PSEN1 = presenilin 1: 107.9% ± 5.5%, p = 0.17, n = 9; PSEN2 = presenilin 2: 102.1% ± 6.0%, p = 0.73, n = 9; NCSTN = nicastrin: 110.4% ± 6.6%, p = 0.13, n = 9; PSENEN = presenilin-enhancer 2: 95.7% ± 5.2%, p = 0.42, n = 9; APH1a = anterior-pharynx-defective 1 a: 105.4% ± 3.8%, p = 0.18, n = 9; APH1b = anterior-pharynx-defective 1 b: 85.2% ± 3.4%, p ≤ 0.001, n = 9). Statistical significance as described for Figure 3 (**p ≤ 0.001; n.s., not significant).
Figure S5. Unaltered protein level of BACE1 (97.8% ± 3.2%, p = 0.68, n = 7) and nicastrin (91.8% ± 3.9%, p = 0.13, n = 4) in the presence of α-tocotrienol. Statistical significance as described for Figure 3 (n.s., not significant).