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Piceatannol-mediated alteration of amyloidogenesis in SH-SY5Y neuroblastoma cells treated with amyloid-beta

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Abstract
High glucose condition impairs neuronal integrity and function by regulating amyloidogenesis and neuroinflammation. Here, we pursue to investigate whether high glucose condition leads impairments in neuronal integrity/function by inducing amyloidogenesis and neuroinflammation, and whether piceatannol (PIC) restores high glucose condition-induced deteriorations in neuronal cells. High glucose condition was induced by maintaining SH-SY5Y neuroblastoma cells in 25 mM glucose. In addition, amyloid-beta was applied to make the cells having amyloidogenesis-mediated deleterious alterations. Cells were maintained in low glucose (2.5 mM glucose) or high glucose (25 mM glucose) condition. And, two doses (10 and 20 μM) of PIC and/or 10 μM of amyloid-beta were treated in a subset of cells for 24 h. There were no significant differences in cell morphology and cell viability among groups. PIC treatment did not show cell toxicity based on cell viability assessed by PrestoBlue assay. High concentration of glucose and amyloid-beta treatments increased amyloid-beta concentrations in cell lysates and conditioned media, which were reduced by PIC. 10 μM PIC treatment for 24 h decreased amyloid precursor protein (APP), beta-site amyloid precursor protein cleaving enzyme-1, presenilin (PS) 1 and tau. Among analyzed inflammatory markers, interleukin (IL)-1 beta, IL-6, and tumor necrosis factor (TNF)-alpha were reduced in conditioned media by 10 μM PIC administration for 24 h. These data indicate that high glucose condition may result in excessive levels of amyloidogenesis and neuroinflammation as well as subsequent changes in neuronal integrity/function, and that PIC treatment may ameliorate the deleterious consequences from high glucose and amyloid-beta treatments.

Conflict of Interest
There is no conflict of interest.