An attempt to establish an assay system for protective agents against glutamate-induced neurotoxicity using QNR/D cells, a cell line derived from quail embryotic neruroretinas

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Glaucoma is one of the most common causes of blindness in Japan. In the retina of the patients of glaucoma, retinal ganglion cells (RGC) are selectively degenerated, and glutamate-induced toxicity may be involved in the underlying mechanisms. Previously, RGC-5 was proposed to be a cell line derived from rat retinal ganglion cells and used as a useful model for the assay for glutamate-induced RGC toxicity. However, recent studies have revealed that RGC-5 is not rat RGC, but murine photoreceptor cells. Recently, QNR/D cells, a cell line derived from quail embryotic neruroretinas, were reported and the cell line can be obtained from ATCC. In the present study, we tried to establish an assay system for protective agents against glutamate-induced toxicity using QNR/D cells. QNR/D cells were cultured in DMEM supplemented with 10% FBS in 5% CO2/95% O2 at 39 degree Celsius. Cell survival was determined by WST-8 assay. Under the presence of L-buthionine-(S,R)-sulfoximine (2 mM), which depletes cellular glutathione levels, glutamate (4-25 mM) concentration-dependently induced cell death. This cell death was reduced by vitamin E (100 µM), but not by MK-801, an N-methyl-D-aspartic acid (NMDA) receptor antagonist, nor L-nitro-L-arginine methyl ester, an NO synthase inhibitor. NMDA did not induce cell death. These results suggest that protective agents not against excitotoxicity, but against oxidative glutamate toxicity can be assayed using the present experimental system.