Calcium homeostasis is considered to be important in both expected antineoplastic and neurotoxic adverse effects of cisplatin. Voltage-activating calcium channels are thought to be responsible for these neurotoxic side effects of cisplatin. Because cisplatin cannot cross the blood-brain barrier, central nervous system neurons are not directly exposed to toxic levels of the drug. But the dorsal root ganglia are supplied by fenestrated capillaries and thus, sensory neurons are exposed to high levels of the drug. This study was aimed to investigate the role of Ca$^{2+}$ in cisplatin neurotoxicity by using nimodipine (an L voltage-sensitive calcium channel antagonist), MK-801 (an NMDA channel antagonist) and thapsigargin (an inhibitor of efflux of calcium from intracellular stores) in cultured rat dorsal root ganglion (DRG) neurons.

DRG cells were used for the determination of cisplatin (at the doses of 10-200 µM) neurotoxicity. Primary cultures of DRG were prepared from 1-day old Sprague Dawley rats. The toxic effects of cisplatin were evaluated by incubating the cells with cisplatin alone and with cisplatin plus nimodipine (1 µM), MK-801 (1 µM) or thapsigargin (100 µM) for 24 hours. MTT assay was used to detect the toxicity of DRG cells. Results were evaluated by using ELISA test system at a wavelength of 450 nm. Student's t test was used for statistical analysis.

The neurotoxicity of cisplatin was significant when used in high concentrations (100 and 200 µM). Nimodipine (1 µM) prevented the neurotoxic effects of cisplatin, whereas a beneficial effect was not observed with MK801 or thapsigargin.

The results of the present study may suggest that voltage-dependent calcium channels is likely to play a role in cisplatin neurotoxicity.