

cell lines (WHO grade I and II). The expression and location of critical proteins in these 6 patient-derived cancer cell lines were examined using immunofluorescence analysis. Also, analysis by GTG-banding determined the existence of different patterns of chromosomal abnormalities in representative cancer cell lines.

**Results:** The GBM cell line showed positive expression of NESTIN, SOX2, VIMENTIN and GFAP. All of the meningioma-derived cancer cell lines showed homogenous VIMENTIN expression, whereas various expression patterns were examined in NESTIN depending on the patient. The GBM cell line showed the gain of chromosome 7 with loss of an X chromosome in 10 out of 35 metaphases (28.6%). A near-tetraploid karyotype (4n) was found in 11 out of 30 metaphases (36.7%) of a transitional meningioma cell line and the loss of a Y chromosome was examined in 4 out of 30 metaphases (13.3%) obtained from an atypical meningioma cell line.

**Conclusions:** The patient derived cancer cell lines established in this study might be used to study the mechanism of brain tumor and improve the success in the clinic by explaining the slightly different clinical behavior among the patients.

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## P-250

### Enhanced expression of immediate-early genes in mouse hippocampus after trimethyltin treatment

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**Introduction:** Immediate-early genes (IEGs) are transiently and rapidly activated in response to various cellular stimuli. IEGs mediate diverse functions during pathophysiologic events by regulating cellular signal transduction. We investigated the temporal expression of several IEGs, including c-fos, early growth response protein-1 (Egr-1), and activity-regulated cytoskeleton-associated protein (Arc), in trimethyltin (TMT)-induced hippocampal neurodegeneration.

**Materials and Methods:** Mice (7 weeks old, C57BL/6) were administered TMT (2.6mg/kg intraperitoneally).

**Results:** The mice presented severe neurodegenerative lesions

in the dentate gyrus (DG) and showed behavioral seizure activity on days 1-4 post-treatment, after which the lesions and behavior recovered spontaneously over time. c-fos, Egr-1, and Arc mRNA and protein levels significantly increased in the mouse hippocampus after TMT treatment. Immunohistochemical analysis showed that nuclear c-fos expression increased mainly in the DG, whereas nuclear Egr-1 expression was increased extensively in cornu ammonis (CA) 1, CA3, and the DG after TMT treatment. Increased Arc levels were detected in the cellular somata/dendrites of the hippocampal subregions after TMT treatment.

**Conclusions:** Therefore, we suggest that increased IEGs are associated with TMT-induced pathological events in mouse hippocampus.

## P-251

### Trimethyltin-induced hippocampal neurodegeneration

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**Introduction:** Trimethyltin (TMT), a toxic organotin compound, induces neurodegeneration selectively involving the limbic system and especially prominent in the hippocampus. Neurodegeneration-associated behavioral abnormalities, such as hyperactivity, aggression, cognitive deficits, and epileptic seizures, occur in both exposed humans and experimental animal models.

**Materials and Methods:** Previously, TMT had been used generally in industry and agriculture, but the use of TMT has been limited because of its dangers to people.

**Results:** TMT has also been used to make a promising in vivo rodent model of neurodegeneration because of its region-specific characteristics. Several studies have demonstrated that TMT-treated animal models of epileptic seizures can be used as tools for researching hippocampus-specific neurotoxicity as well as the molecular mechanisms leading to hippocampal neurodegeneration.

**Conclusions:** This review summarizes the in vivo and in vitro underlying mechanisms of TMT-induced hippocampal neurodegeneration (oxidative stress, inflammatory responses, and neuronal death/survival). Thus, the present review may be helpful to provide general insights into TMT-induced neurodegeneration and approaches to therapeutic interventions for neurodegenerative diseases, including temporal lobe epilepsy.