Six New Heterocyclic Stilbene Oligomers from Stem Bark of *Shorea hemslleiana*.
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From the stem bark of *Shorea hemslleiana* (Dipterocarpaceae), four new stilbenoids with one or two dihydrofuran ring(s)
(hemslleianols C, D, hemslleianosides E, F and (-)-ampelophosin H) and a new stilbenoid with a carbonyl group (hemslleianol E)
were isolated. These structures including the relative configuration were elucidated on the basis of spectroscopic evidence.

Four New Glucosides of Stilbene Oligomers From the Stem of *Gnetum gnemonoides*.
Ibrahim IILYA, Toshiyuki TANAKA,* Miyuki FURUSAWA, Zulfiqar ALI, Ken’ichi NAKAYA,
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Four new stilbene oligomeric glucoside, gnemonoside A, B, C and D, were isolated from the stem of *Gnetum gnemonoides*
along with four known stilbenoids, trans-resveratrol, gnetins C, D and E. The structures were elucidated on the basis of spectral
evidence.

Administration of FGF-2 to Embryonic Mouse Brain Induces Hydrocephalic Brain Morphology and
Aberrant Differentiation of Neurons in the Postnatal Cerebral Cortex.
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FGF-2 was injected into mouse cerebral ventricles at E 14 in utero. High doses of FGF-2 (200 or 300 ng) caused encephalic
alterations such as deformation of the calvarium, enlargement of the ventricular spaces, and thinning of the cerebral cortex. There
was no gross abnormality in the alignment of the cerebral neuronal layers, however, both cell number and cell density of the upper
layers (II/III) and the lower layers (IV-VI) of the cerebral cortex were increased. BDNF, tyrosine hydroxylase, nestin, and
microtubule-associated protein 2 were aberrantly or ectopically expressed in the deep areas of the cerebral cortex. These observations
demonstrate that a subpopulation of neurons in the cortical deep layer abnormally differentiated or partly sustained their immature
state following a single administration of FGF-2. Developmental analysis of localization of BDNF-positive cells suggested that the
abnormality started around P5. FGF-2 seems to play predominant roles in the proliferation of neuronal precursors and in neuronal
differentiation in the developing mouse cerebral cortex even at relatively late stages of brain neurogenesis.

Difference in Toxicity of \( \beta \)-Amyloid Peptide with Aging in Relation to Nerve Growth Factor Content
in Rat Brain.
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Amyloid \( \beta \)-peptide (A \( \beta \)) is the major constituent of the senile plaques in the brains of patients with Alzheimer’s disease. We
have demonstrated previously that memory impairment, dysfunction of the cholinergic and dopaminergic neuronal system and
morphological degeneration are produced after the continuous infusion of A \( \beta \) into the cerebral ventricle in 8-week-old rat. In the
present study, we investigated the toxicity of A \( \beta \) in infant (10 days old), adult (8 weeks old) and aged (20 months old) rats in
relation to NGF content in various regions of the brain. After a 2-week-infusion, choline acetyltransferase activity was significantly
decreased in the hippocampus of adult, but not infant or aged rats. NGF levels in the hippocampus were increased only in adult rats.
These results suggest that A \( \beta \) is toxic only in the matured adult brain, and that the mechanism of toxicity is related to NGF synthesis.