OBJECTIVE: On the new founding of our former research that the premature brain has a stronger resistance to brain damage induced by prolonged seizure or status epilepticus, to explore the molecular biological base of internal protective response in premature brain which reducing the process of apoptosis and necrosis of neurons.

DESIGN: Megimide was injected in healthy adult rats and baby rats respectively to evoke prolonged seizures and SE. Adult rats and baby rats were sacrificed at 1, 2, 4, 12, 24, 48, 72 hours and 7 days after prolonged seizure stopped. Hippocampus, dentate gyrus and parietal cortex of their brain were taken for immunocytochemistry studies for apoptosis associated genes bcl-2 and P53 expression.

RESULTS: (1) There was a much stronger and higher rate (90%) of P53 expression in adult rats' brain, and such state continually keep even at 72h after seizure stopped; however bcl-2 expression was weak and low (57%), and no difference was found on bcl-2 expression only in several hours comparing with normal control. (2) In baby rats' brain there was more than 85% neurons with a strongest expression of bcl-2; and the strongest bcl-2 expression sustained in 72h after SE, however P53 expression lasted less than 24h.

CONCLUSION: Significant difference between premature and mature brain was shown on the genes’ expression associated apoptosis after seizure. Anti apoptosis gene (bcl-2) expression was much higher and longer than apoptosis gene (P53) in baby rats’ brain, and it was reversed in adult rats group. So the strongest expression of bcl-2 after status epilepticus could be the important molecular biological basis for the special resistance to neuron injury in premature brain.