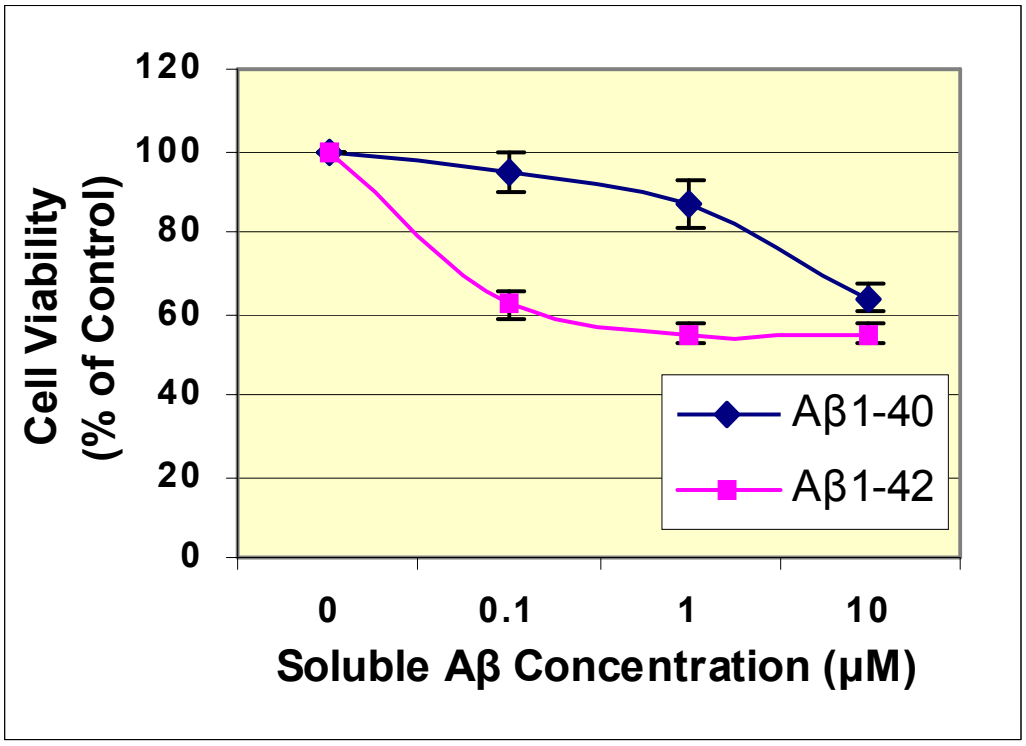
Supplementary data: Figure-1



Cytotoxicity of Aβ1-40 and Aβ1-42 in mice primary neuronal cells: Mice primary cortical neuronal cells were exposed to various concentrations (0.0/0.1/1.0/10.0 μM) of soluble (non-aggregated) Aβ1-40 or Aβ1-42 obtained from commercial source (American peptide company, Sunnyvale, CA). After 48 hours of exposure, cell viability was tested by MTT assay using Promega (Madison, WI) kit. Results revealed that both the peptides were toxic to the cells even at lower concentrations and Aβ1-42 was more cytoxic than Aβ1-40. Data shown were Mean ± S.E. derived from 3-4 independent observations.

Supplementary data: Table 1

Gene ID	Description	Fold increase (Pb/Control)	CpG rich
U77942	Syntaxin 7 (STX7)	2.672	No
J04046	Calmodulin	1.867	Yes
D21243	Heme oxygenase 2 (HO2)	1.822	Yes
D30648	Flavoprotein subunit of complex II;	1.775	Yes
M22430	Membrane-associated phospholipase A2 precursor	1.742	Yes
D78579	Neuron-derived orphan receptor 1 (NOR1)	1.584	Yes
M28215	ras-related protein RAB5A	-1.558	Yes
D63475	Clathrin coat assembly protein AP50	-1.567	Yes
U71127	ras-related protein RAB-32	-1.634	Yes
X69550	rho GDP dissociation inihibitor 1 (RHO-GDI 1)	-1.653	Yes
U18420	ras-related protein RAB-5C	-1.739	Yes
M36430	Guanine nucleotide-binding protein beta subunit 1 (GNB1)	-1.976	Yes
D13988	RAB GDP dissociation inihibitor beta (RAB GDI beta)	-2.062	Yes
L36983	dynamin 2	-2.326	Yes
L33075	ras GTPase-activating-like protein IQGAP1; p19; KIAA0051	-2.404	Yes
U07364	Inward rectifier potassium channel 4	-2.427	Yes
X85030	Calpain p94 large (catalytic) subunit	-2.558	Yes
L25119	mu-type opioid receptor (MOR-1)	-2.786	Yes
D21260	Clathrin heavy subunit 1 (CLH-17); KIAA0034	-2.915	Yes
U07882	delta-type opioid receptor (DOR-1)	-3.861	Yes
D10995	5-hydroxytryptamine 1B receptor (5-HT-1B)	-3.968	Yes
X55758	D1A dopamine receptor (DRD1A)	-4.167	No

Microarray screening of neurobilogy related genes: Microarray array analysis of about 588 neurobiology-related human genes was conducted to identify the genes that are altered due to infantile exposure to Pb in the frontal association cortex of 23-year old cynomolgus monkeys. The results showed that the expression profile of only a few genes (22) was changed due to early life exposure to Pb. Most of the altered genes belonged to neurotransmitter, growth receptors and signal transduction pathways. We further conducted searches on various databases (Ensembl and NCBI nucleotides) to determine if the regulatory regions of these genes were rich in CpG dinucleotides (>60%). We found that most of these altered genes (with the exception of two) were abundant in CpG dinucleotides in their 5'untranslated regions (5'UTR). These findings provide the initial evidence for the association of epigenetic pathway to explain such latent effects; however, these studies require validation by high throughput methylation sequence profiling.