Supplemental table 1. The information of the patients that were used in the study.

Patient#	Age	Gender	Cold PMI[1]	Frozen PMI[1]	Neuropathological Diagnosis
T92	68	M	2:09	11:29	no abnormality recognized
T99	44	M	23:10	49:15	no abnormality recognized
T111	33	M	3:40	34:50	no abnormality recognized
T136	64	W	7:50	11:30	no abnormality recognized
T139	57	M	unknown	unknown	no abnormality recognized
T147	92	W	1:55	6:55	no abnormality recognized
T148	66	M	10:25	21:55	no abnormality recognized
T159	54	W	3:40	15:40	no abnormality recognized
T164	41	M	1:46	7:01	no abnormality recognized
T168	57	M	5:09	6:29	no abnormality recognized
T230	87	M	5:10	6:40	Alzheimer changes, mild[2]
T243	77	W	unknown	11:50	Parkinson disease, moderate[3]
T247	90	W	6:40	8:45	Alzheimer's Disease[4]
T267	73	M	5:30	22:45	Alzheimer's Disease[4]
T278	86	M	2:05	50:25	Alzheimer's Disease[4]
T279	83	M	3:45	5.35	Alzheimer's Disease[4]
T293	89	W	13:50	15:45	Alzheimer's Disease[4]
T313	83	W	6:30	8:15	Alzheimer's Disease[4]
T314	85	W	4:13	6:23	Alzheimer's Disease[4]
T324	85	W	4:15	8:35	Alzheimer's Disease[4]
T344	89	W	5:00	7:30	Alzheimer's Disease[4]
T360	74	M	5:15	23:45	Alzheimer's Disease[4]
T567	84	M	4:24	6:19	Alzheimer's Disease[4]
T704	83	M	11:35	13:05	Alzheimer's Disease[4]
T725	65	W	4:35	5:45	Alzheimer's Disease[4]
T3799	98	W	2:55	14:09	Alzheimer changes, mild[2]

[1] post-mortem interval

Cold PMI is computed according to the following formula:
Cold PMI (in hours) = Time when the body, or brain is put in cold – Time of death

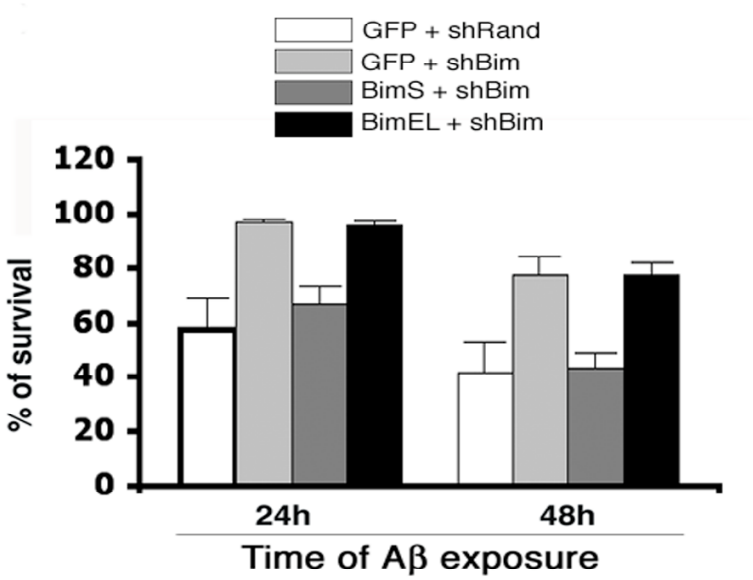
Frozen PMI (or PMI) is computed according to the following formula:
Frozen PMI (in hours) = PMI = (Time starting processing + 1 h) – Time of death

[2] These individuals were having occasional neuritic plaques or tangles or both in the brain without neurologic or psychiatric disorder, but with normal cognition.

[3] Encephalopathy, degenerative, moderate, consistent with Parkinson disease

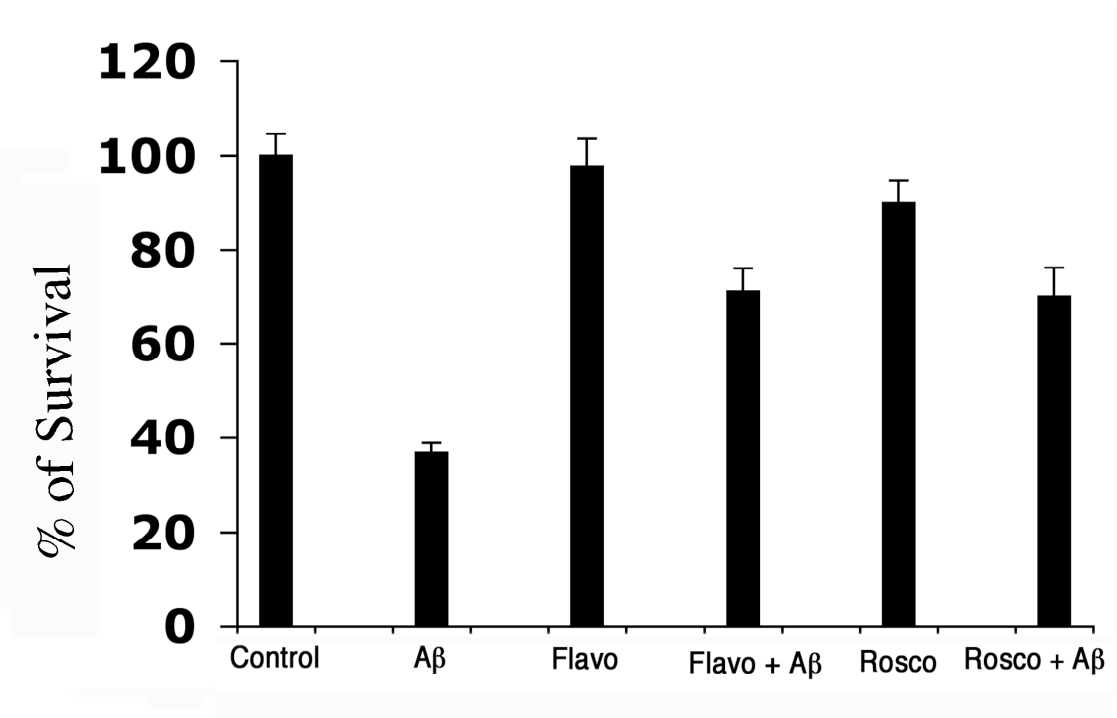
[4] Encephalopathy, degenerative, marked, consistent with Alzheimer disease

Supplemental figure 1.



Bim shRNA does not provide protection against death evoked by Aß in presence of the short isoform of Bim (BimS) that is not targeted by shBim. Cortical neurons were transfected with PCMS-eGFP (GFP), PCMS-BimS-eGFP (BimS) or PCMS-BimEL-eGFP (BimEL) with pSIREN-Bim-shRNA-ZsGreen (shBim) or pSIREN-Random-shRNA- ZsGreen (shRand), maintained for 48h in presence of a pan caspase inhibitor (25 μM ZVAD-fmk) and then washed twice with medium and treated with 1 μM oligomeric Aß as indicated. Numbers of surviving transfected (green) cells were counted just after treatment with Aß and after 24h and 48h. Data represent means ± SEM of 3 replicates. The results show that Bim shRNA provides significant protection of cultured cortical neurons from death evoked by Aß even in presence of exogenous BimEL but does not protect them in presence of BimS (shBim does not target BimS) after Aß exposure.

Supplemental Figure 2.



Cdk inhibitors protect neurons from apoptotic death evoked by Aß. Cortical neurons were cultured in 24-well dishes for 5 d, and then treated with 1 μM oligomeric Aβ in presence and absence of 1 μM flavopiridol (Flavo) or 20 μM roscovitine (Rosco). After 48 hr the cells were lysed, and the numbers of intact nuclei were counted. Cell survival was expressed as a percentage of the number of living cells in the treated cultures compared with the controls. Please note that cdk inhibitors, falovopiridol and roscovitine protected cultured cortical neurons from death evoked by Aß.