Journal of Alzheimer's Disease 28 (2012) 1–6
IOS Press

Supplementary Data

The Dying of the Light: Mitochondrial Failure in Alzheimer's Disease

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Accepted 14 October 2011

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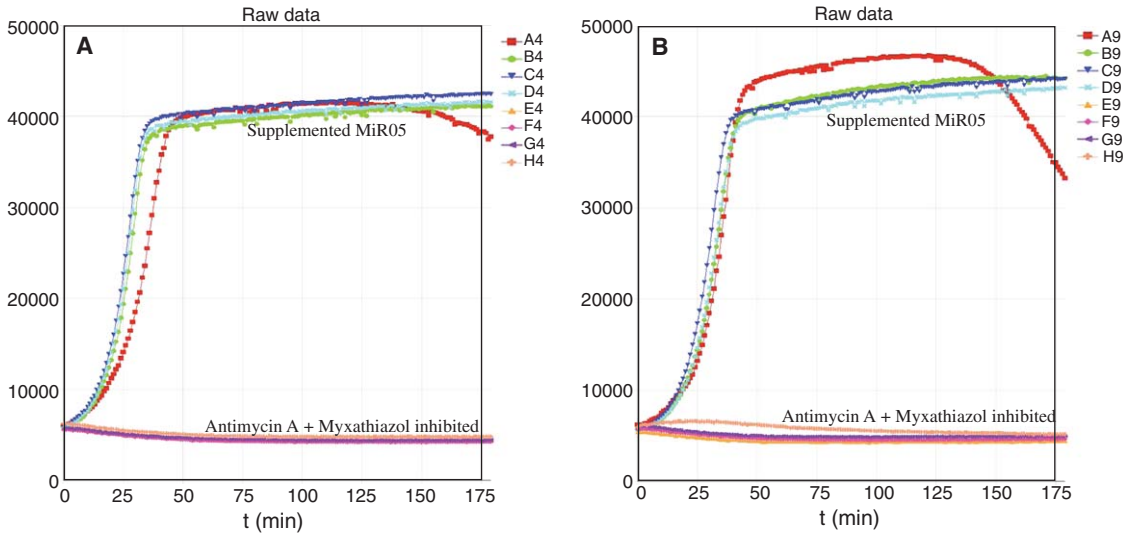
ISSN 1387-2877/12/$27.50

Supplementary Table 1
Average age of CTL subjects is 68.88 ± 4.658 ($n = 8$; 62.5% male), while the average age of AD subjects is 79.80 ± 2.962 ($n = 10$; 70% female). The postmortem interval (PMI) is 10.25 ± 1.763 h ($n = 8$) for CTL subjects versus 17.17 ± 3.593 h ($n = 9$) for AD subjects

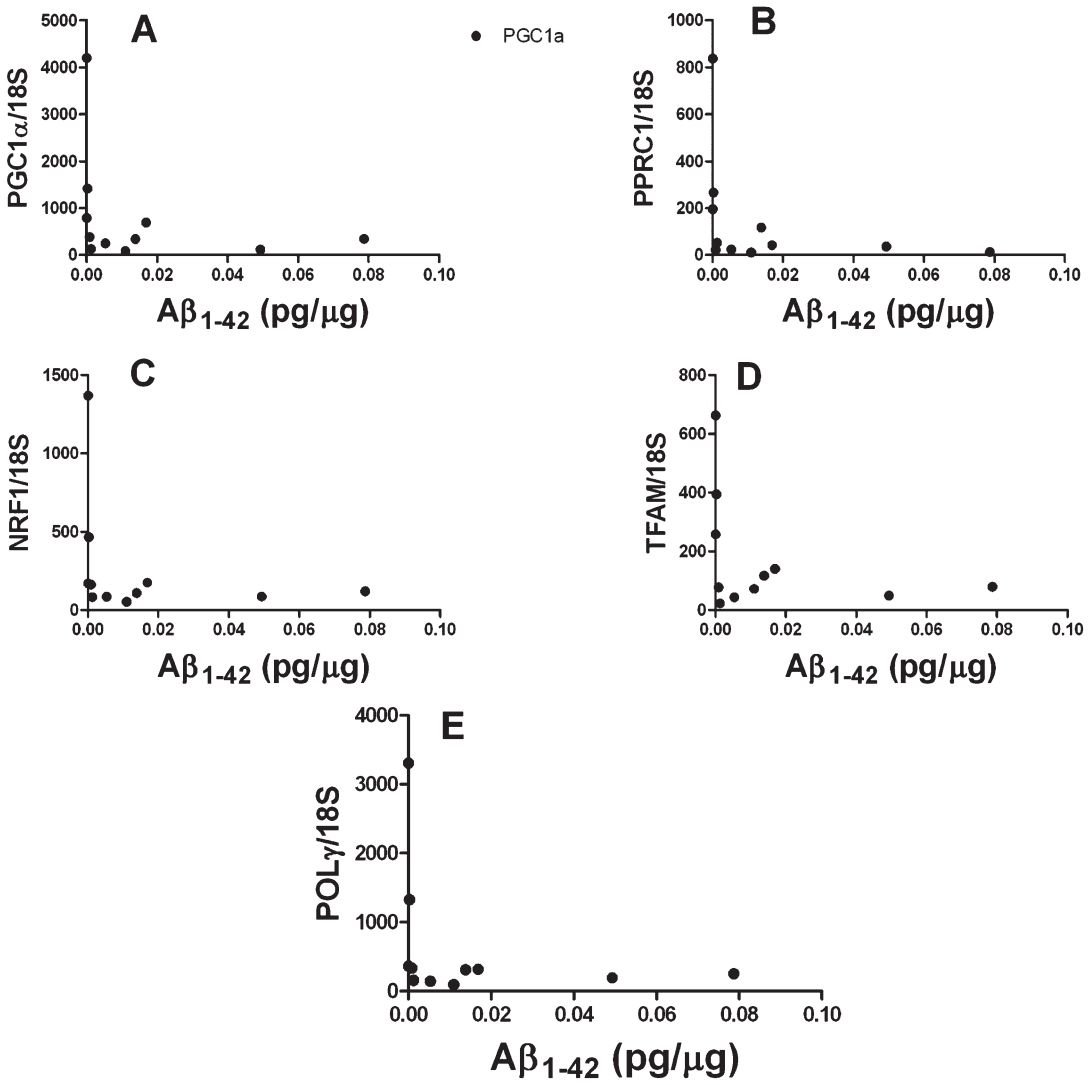
Brain	Age	Gender	Post-mortem interval (h)
CTL1	76	M	12
CTL2	88	M	5.5
CTL3	63	M	12
CTL4	59	M	2.5
CTL5	53	F	14
CTL6	61	M	6
CTL7	87	F	13
CTL8	64	F	17
AD1	77	F	9.5
AD2	92	F	3.5
AD3	71	F	37.5
AD4	73	F	8.5
AD5	81	F	11.5
AD6	88	F	20
AD7	84	F	29.5
AD8	87	M	18.5
AD9	61	M	16
AD10	84	M	NK

Supplementary Table 2
Primer and probe sequences for human mtDNA, biogenesis, and antioxidant genes

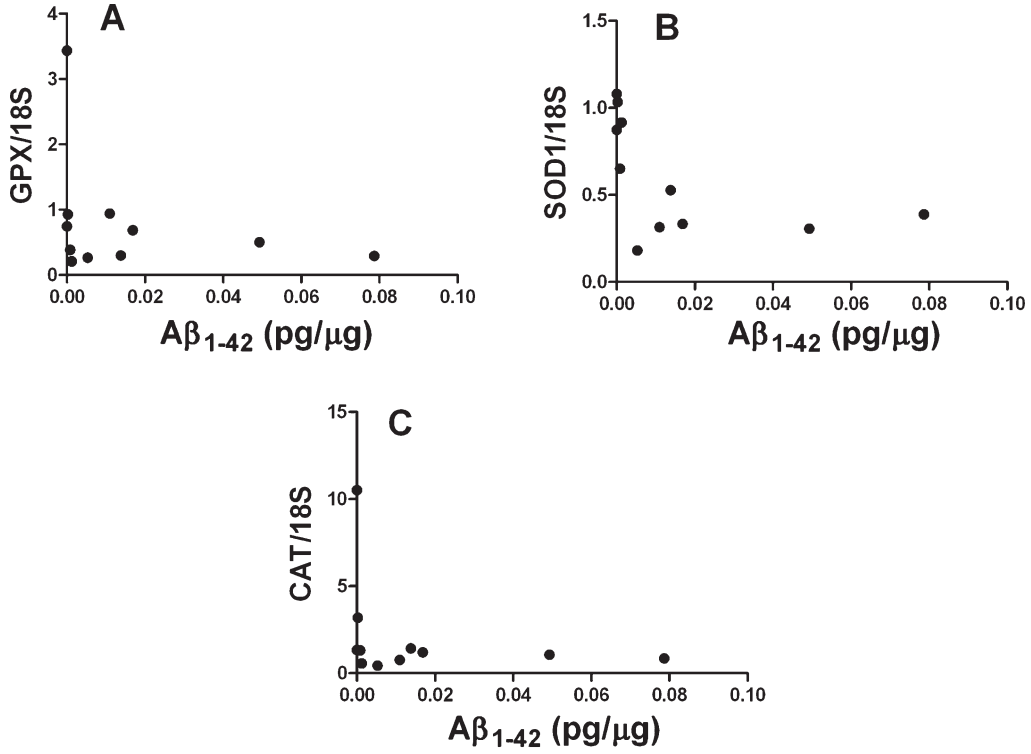
Gene	Sequence
ND2, ND4, and CO3 sense and antisense primers	[40]
PGC1α sense primer	AGCTAAGTTATAATGAATGC
PGC1α antisense primer	CTTTTGCTGTTGACAAAT
PGC1α probe (Tx Red)	TCTTCGCTTTATTGCTCCA
TFAM sense primer	GCACAATAAAGAAACAAC
TFAM antisense primer	ATTTTACGATAGCTTCAG
TFAM probe (FAM)	TCCTGTGCCTATCCATTG
NRF1 sense primer	GTATTTGAGTCTAATCCAT
NRF1 antisense primer	AGATACAGAGGACAATAG
NRF1 probe (TET)	CACGAGTAGTATATTCATCTAACG
PPRC1 sense primer	AGCTACATGGATG
PPRC1 antisense primer	AGAGCCGTGAG
PPRC1 probe (FAM)	CACTTCATTCTGGTCCTCC
POLγ sense primer	GACTGGCAGGA
POLγ antisense primer	AGCCCTGAGATG
POLγ probe (TET)	AATGTTTCCTTTGACCGAG
SOD1 sense primer	CCAAAGGATGAAGAGAGG
SOD1 antisense primer	GAGAGTGAGATCACAGAAT
SOD2 sense primer	TGTTAAGCTCTTTATGACTGTTT
SOD2 antisense primer	GTGACTAAGCAACATCAAGAA
GPX1 sense primer	ATGGGTGCTGGTCCTGTT
GPX1 antisense primer	CTGACACCCGGCACTTTA
GSS sense primer	TACTGATTGCTCAAGAGAAGG
GSS antisense primer	CGTCGGATCACATGGATG
GSR antisense primer	GTGAATGTTGGATGTGTA
GSR antisense primer	GACTTGGTGAGATTGTTT
CAT sense primer	ACATTTAATCAGGCAGAAA
CAT antisense primer	CAACCTCAGCAAAGTAAT
SirT3 sense primer	TATTAAAGGTGGAAGAAGGT
SirT3 antisense primer	GAATCAGCTCAGCTACAT



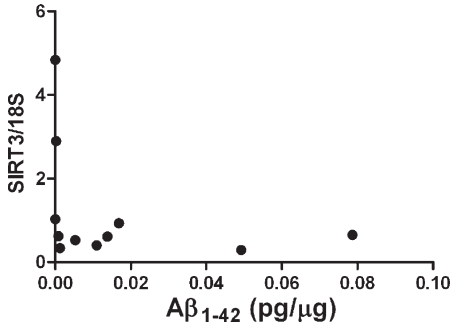
Supplementary Figure 1. The respirosomes of fresh and frozen mouse brain mitochondria function in a similar pharmacologically-dependent manner. Mice were sacrificed and had their brain mitochondria isolated immediately (fresh, A) or after 16 h at −20°C (frozen, B). Mitochondrial isolates were resuspended in MiR05 supplemented with malate, pyruvate, succinate, ADP, and cytochrome c and added to a 96-well BD Biosensor plate in quadruplicate and is reported as maximum slope (A: 2088 ± 85.1 RFU/min, $n = 4$; B: 2037 ± 62.1 RFU/min, $n = 4$), which is observed during the first hour of measurement. The oxygen consumption observed was inhibited by antimycin A and myxathiazol, two inhibitors of Complex III.



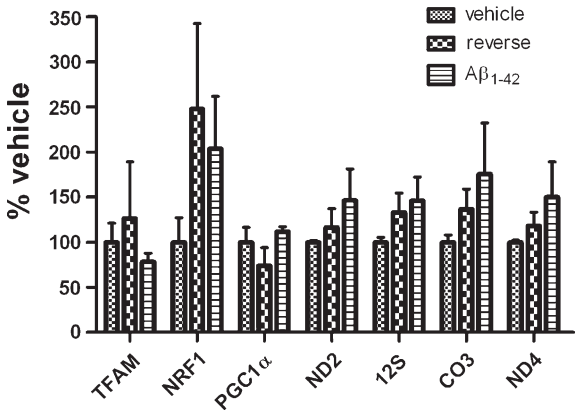
Supplementary Figure 2. $A\beta_{1\text{-}42}$ levels in human brain mitochondria do not correlate with expression of genes responsible for mitochondrial biogenesis. Expression of genes (A-E) responsible for mitochondrial biogenesis was compared to mitochondrial $A\beta_{1\text{-}42}$ concentration.



Supplementary Figure 3. Antioxidant gene expression does not correlate with $A\beta_{1\text{-}42}$ levels in human brain mitochondria. Expression of ROS scavenger genes (including GPX, SOD1, and CAT, A-C) was compared to mitochondrial $A\beta_{1\text{-}42}$ levels.



Supplementary Figure 4. Expression of SirT3, a gene responsible for epigenetic metabolic regulation, does not correlate with human brain mitochondrial $A\beta_{1\text{-}42}$. SirT3 expression was compared to mitochondrial $A\beta_{1\text{-}42}$ levels.



Supplementary Figure 5. mRNA expression of DAN cells treated with $A\beta_{1\text{-}42}$ did not significantly differ from those treated with $A\beta_{42-1}$. mRNA for PGC1α, NRF1, TFAM, ND2, 12S, CO3, and ND4 was measured for DAN cells treated with $A\beta_{1\text{-}42}$ and its reverse peptide $A\beta_{42-1}$.