Data in Brief 18 (2018) 902–912

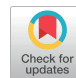
Contents lists available at ScienceDirect

Data in Brief

journal homepage: www.elsevier.com/locate/dib

Data Article

Dataset on gene expression in the elderly after Mindfulness Awareness Practice or Health Education Program



Hwee-Woon Lim [a], Woei-Yuh Saw [b,c], Lei Feng [d], Yuan-Kun Lee [e], Ratha Mahendran [f], Irwin Kee-Mun Cheah [g], Iris Rawtaer [d], Alan Prem Kumar [h,i], Ee-Heok Kua [d], Rathi Mahendran [d,j], Ene-Choo Tan [a,k,*]

[a] *KK Research Laboratory, KK Women's and Children's Hospital, Singapore*
[b] *Saw Swee Hock School of Public Health, National University of Singapore, Singapore*
[c] *Life Sciences Institute, National University of Singapore, Singapore*
[d] *Department of Psychological Medicine, Yong Loo Lin School of Medicine, National University of Singapore, Singapore*
[e] *Department of Microbiology and Immunology, Yong Loo Lin School of Medicine, National University Hospital, Singapore*
[f] *Department of Surgery, Yong Loo Lin School of Medicine, National University of Singapore, Singapore*
[g] *Department of Biochemistry, Yong Loo Lin School of Medicine, National University of Singapore, Singapore*
[h] *Medical Science Cluster, Yong Loo Lin School of Medicine, National University of Singapore, Singapore*
[i] *Cancer Science Institute of Singapore, National University of Singapore, Yong Loo Lin School of Medicine, National University of Singapore, Singapore*
[j] *Duke-NUS Medical School, Singapore*
[k] *SingHealth Duke-NUS Paediatrics Academic Clinical Program, Singapore*

ARTICLE INFO

Article history:
Received 16 January 2018
Received in revised form
15 March 2018
Accepted 20 March 2018
Available online 26 March 2018

Keywords:
Elderly
Gene expression
Mild cognitive impairment

ABSTRACT

It has been reported that relaxation techniques can improve physical health and cognitive function. A number of studies involving different types of relaxation practices showed changes in expression of genes. We investigated the gene expression pattern of a cohort of elderly subjects of Asian descent after weekly (for the first three months) and monthly (for the subsequent six months) intervention. Sixty consenting elderly subjects (aged 60–90 years) with mild cognitive impairment were assigned to either the Mindfulness Awareness Practice (MAP) or Health Education Program (HEP) group in a randomized controlled trial to assess the effectiveness of the programs in

* Corresponding author at: KK Research Centre, KK Women's and Children's Hospital, 100 Bukit Timah Road, Singapore 229899, Singapore.
E-mail address: tan.ene.choo@kkh.com.sg (E.-C. Tan).

https://doi.org/10.1016/j.dib.2018.03.086
2352-3409/© 2018 The Authors. Published by Elsevier Inc. This is an open access article under the CC BY license (http://creativecommons.org/licenses/by/4.0/).

Mindfulness Awareness Practice
Health education program

preventing further cognitive decline and evaluate the influence on neurological, cellular and biochemical factors. Blood samples were collected before the start of intervention and after nine months for gene expression profiling using Affymetrix Human Genome U133 Plus 2.0 arrays. The dataset is publicly available for further analyses.
© 2018 The Authors. Published by Elsevier Inc. This is an open access article under the CC BY license (http://creativecommons.org/licenses/by/4.0/).

Specifications Table

Subject area	Biology
More specific subject area	Gene expression
Type of data	Tables and figures
How data was acquired	Affymetrix GeneChip Human Genome U133 Plus 2.0 Arrays, Microarray Suite 5, Robust Multi-Array Average and statistical analysis
Data format	Raw (CEL.) and normalized (CHP.)
Experimental factors	Pre-intervention samples collected before the start of each program, post-intervention samples collected after 9 months of the program
Experimental features	Subjects were randomized to one of two groups. One group was taught Mindfulness Awareness Practice techniques (MAP). The second group underwent the Health Education Program (HEP) under which they received talks on healthy living topics.
Data source location	National University of Singapore and Research Laboratory, KK Women's and Children's Hospital, Singapore
Data accessibility	http://www.ncbi.nlm.nih.gov/geo/query/acc.cgi?acc=GSE108215

Value of the data

- Practices like yoga, Tai Chi, breathing exercises, and meditation are thought to evoke the relaxation response that counteracts stress response [1].
- A small study of experienced practitioners showed that mindfulness meditation could influence expression of genes related to histone modifications and pro-inflammatory genes [2]. There is also report that after listening to relaxation response-eliciting or health education recordings, healthy adults showed enhanced expression of genes associated with metabolism and telomere maintenance, while the expression of genes linked to inflammatory response and stress-related pathways was reduced [3].
- We investigated whether Asian elderly subjects with mild cognitive impairment would show gene expression changes after undergoing nine months of mindfulness practice program or health education program.
- The identification of genes and pathways can be used for the development of markers to assess the effectiveness of the mindfulness practice or other relaxation exercises.

1. Data

1.1. Isolation of RNA from blood and globin depletion

The average amount of RNA extracted was 14.88 μg while one sample collected before initiation of HEP intervention had insufficient RNA for further analysis. After going through GLOBINclear, the yield

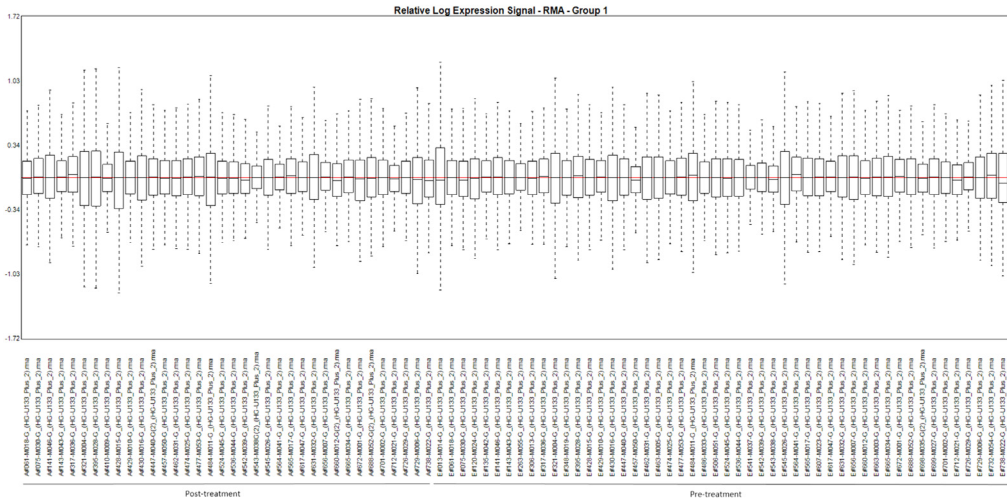


Fig. 1. Relative Log Expression plot based on RMA-normalized signal data. Each boxplot represents a sample. The boxplots presenting the distribution of the log-ratios are centered near 0 and have similar spread.

Table 1
Genes with differentially expressed probesets from unmatched samples analysis using MAS5 normalization.

Group	Gene symbol	Probesets	[*]*p* before FDR	[*]*p* after FDR	[a]Fold change
MAP	*DEC1*	220781_at	5.83E − 05	0.75	3.34
Pre: $n = 26$	*CCDC126*	228087_at	6.74E − 05	0.75	0.83
Post: $n = 17$	*HTR5A*	221362_at	9.18E − 05	0.75	2.08
	TMEM259	212575_at	1.01E − 04	0.75	2.69
	CKAP2	1554264_at	1.42E − 04	0.84	0.32
	FAM27E2/E3	1553590_at	1.84E − 04	0.91	2.22
	EFR3B	215328_at	3.44E − 04	0.99	0.39
	AP3S1	202442_at	4.44E − 04	0.99	0.89
HEP	*RAB3GAP2*	216057_at	1.53E − 04	0.99	0.28
Pre: $n = 24$	LOC284788	1557483_at	1.54E − 04	0.99	0.41
Post: $n = 19$	*C11orf30*	1569349_at	1.55E − 04	0.99	0.71
	FKBP14	235311_at	2.92E − 04	0.99	0.39

FDR = false discovery rate.

[*] *p* from ANCOVA after adjusting for batch and gender effects.

[a] Batch and gender-adjusted fold change.

was between 0.46 and 1.79 μg, with RNA integrity numbers of between 6.6 and 8.9, and 260/280 absorbance ratios of between 1.8 and 2.0 μg.

1.2. Microarray gene expression analysis

Relative Log Expression (RLE) of all the samples was at equal level, signal intensity across the samples were also comparable (Fig. 1). For the pre-MAP and post-MAP analysis using Microarray Suite 5 (MAS5) normalization with log2 transformed, the eight probesets with p-value < 0.0005 (before FDR correction) from unmatched samples analysis are listed in Table 1, and the gene expression heatmap in Fig. 2. The corresponding result for the matched samples analysis between pre-MAP and post-MAP is shown in Table 2. For the group with HEP intervention, the list of probesets which had p-value < 0.0005 (before FDR correction) from unmatched samples analysis is presented in Table 1,

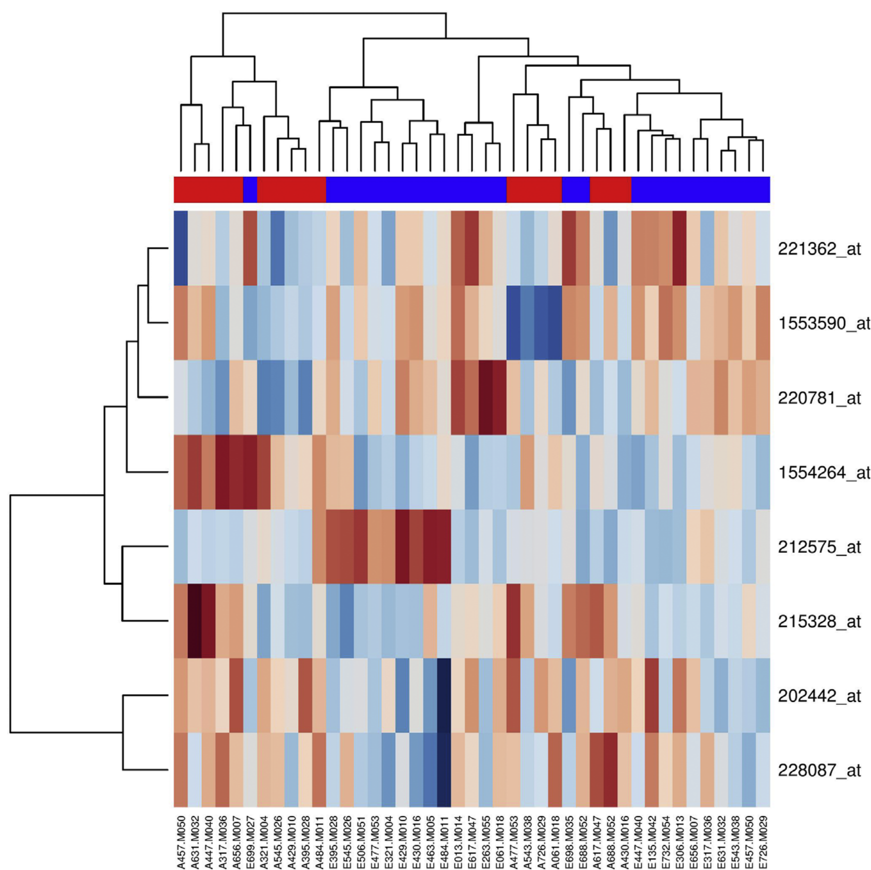


Fig. 2. Heatmap and dendrogram of the differential gene expression profiles for the eight gene markers generated from unmatched pre-MAP and post-MAP intervention samples using MAS5.0 normalization before FDR correction. The pre-intervention group is colored in blue and post-intervention group is in red. The relative expression level of each gene among all the tested samples have been assigned sequentially from dark red color to dark blue color in accordance with low to high expression level.

with the heatmap of the gene expression profiles shown in Fig. 3. The corresponding matched samples analysis result between pre-HEP and post-HEP samples is presented in Table 2.

The list of probesets with p-value < 0.0005 (before FDR correction) between pre- and post-intervention samples within each intervention group from the analysis using Robust Multi-array Average (RMA) normalization are presented in Table 3 for unmatched samples analysis whereas Table 4 shows the corresponding matched samples analysis.

Additional comparison was made between post-MAP and post-HEP samples, the 86 significant probesets with p-value < 0.05 (after FDR correction) or fold change cut-off (fc > 1.5 or fc < 0.66) are listed in Table 5. The heatmap for the corresponding gene expression profiles is shown in Fig. 4.

1.3. Pathway analysis

Analysis using *The Database for Annotation, Visualization and Integrated Discovery* (DAVID) version 6.8 showed that *RAB3GAP2, GRIK2, SCFD1* which are altered following HEP intervention are associated with disorders related to HDL cholesterol according to the data from the Genetic Association Database

Table 2
Genes with differentially expressed probesets from matched samples analysis using MAS5 normalization.

Group	Gene symbol	Probesets	[*]p before FDR	[*]p after FDR	[a]Fold change
MAP	*DEC1*	220781_at	4.70E − 06	0.14	3.77
Pre: $n = 17$	*TEF*	215673_at	8.61E − 05	0.99	0.24
Post: $n = 17$	*CKAP2*	1554264_at	2.79E − 04	0.99	0.30
	CCDC126	228087_at	3.69E − 04	0.99	0.82
	TTLL7	219882_at	3.92E − 04	0.99	2.85
	LRRC37A4P	229821_at	4.77E − 04	0.99	1.28
HEP	*GRIK2*	215655_at	1.73E − 04	0.99	0.19
Pre: $n = 18$	*PRKCZ*	1569748_at	1.82E − 04	0.99	3.16
Post: $n = 18$	*SLC2A5*	230705_at	2.42E − 04	0.99	0.37
	LOC284788	1557483_at	2.98E − 04	0.99	0.38
	SCFD1	233229_at	3.04E − 04	0.99	0.60

FDR = false discovery rate.
[*] p from ANCOVA after adjusting for batch effect.
[a] Batch-adjusted fold change.

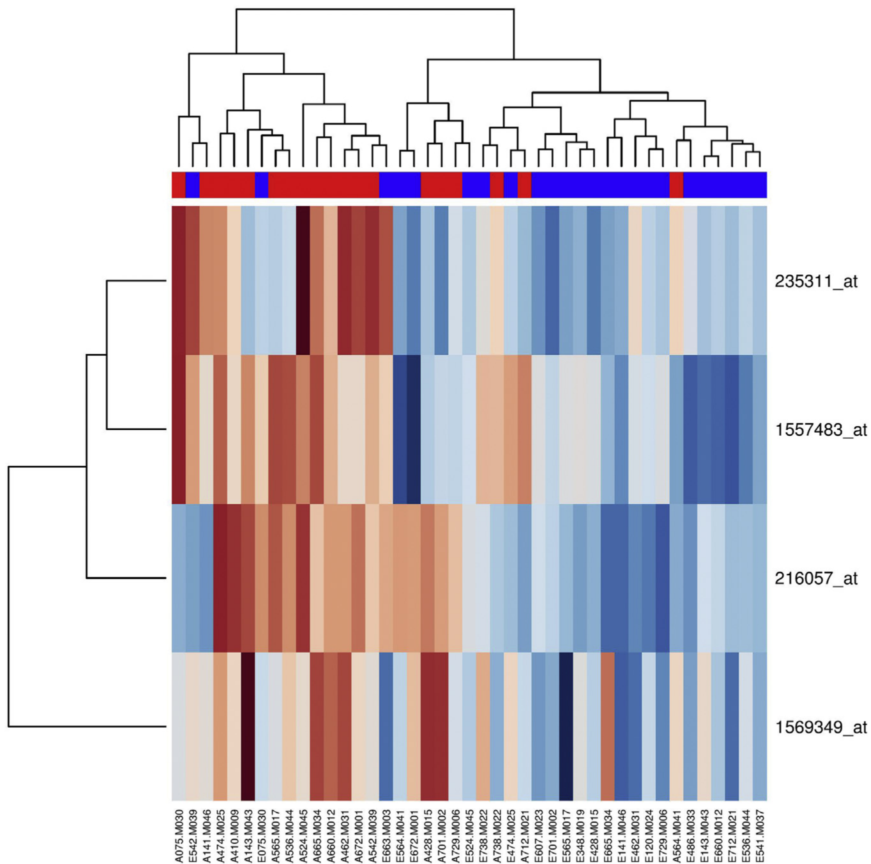


Fig. 3. Heatmap and dendrogram of the differential gene expression profiles for the four gene markers generated from unmatched pre-HEP and post-HEP intervention samples using MAS5.0 normalization before FDR correction. The pre-intervention group is colored in blue and post-intervention group is in red. The relative expression level of each gene among all the tested samples have been assigned sequentially from dark red color to dark blue color in accordance with low to high expression level.

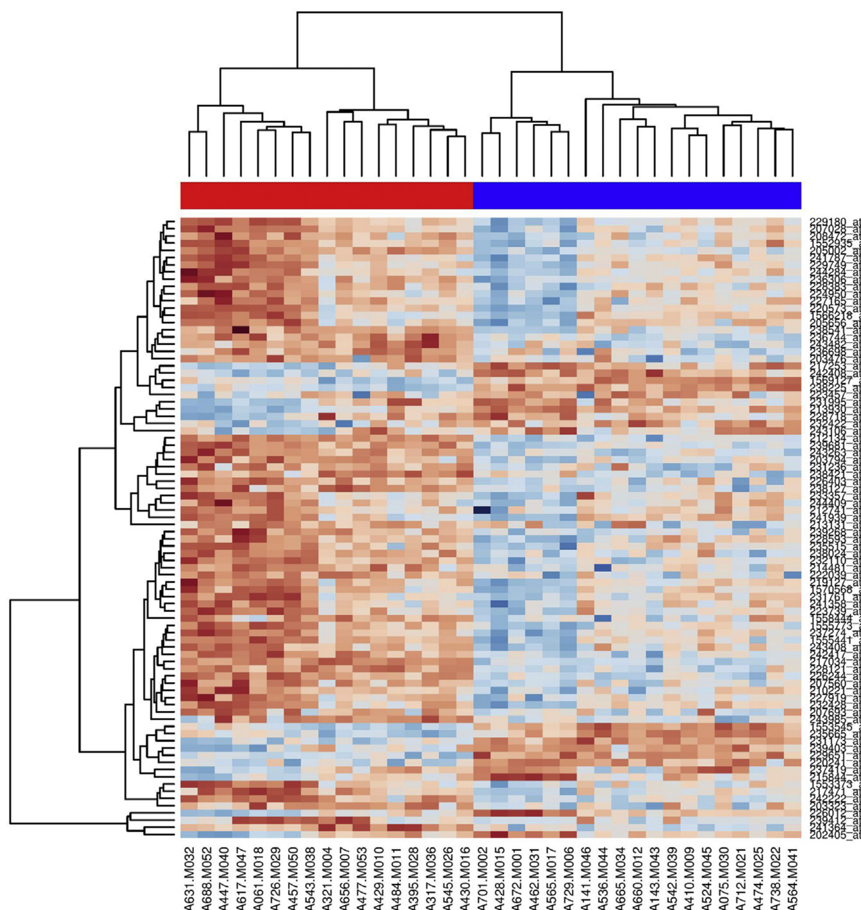


Fig. 4. Heatmap and dendrogram of the differential gene expression profiles for 86 gene markers with at least 1.5 fold up- or down- regulation, generated from post-intervention HEP and MAP samples using RMA normalization. The HEP group is colored in blue and MAP group is in red. The relative expression level of each gene among all the tested samples have been assigned sequentially from dark red color to dark blue color in accordance with low to high expression level.

Table 3
Genes with differentially expressed probesets from unmatched samples analysis using RMA normalization.

Group	Gene symbol	Probesets	[*]*p* before FDR	[*]*p* after FDR	[a]Fold change
MAP	LOC101928211	1559930_at	7.09E – 06	0.21	1.14
Pre: $n = 26$	*B3GNT2*	224154_at	2.31E – 04	0.99	1.17
Post: $n = 17$	LOC100996251	1568892_at	4.44E – 04	0.99	0.88
	CSNK1A1	205764_at	4.89E – 04	0.99	0.85
HEP	*SNAP91*	204953_at	1.00E – 04	0.99	1.14
Pre: $n = 24$	*SRSF4*	241245_at	1.28E – 04	0.99	0.86
Post: $n = 19$	*FABP6*	210445_at	4.00E – 04	0.99	1.20
	KCNB2	208123_at	4.32E – 04	0.99	1.15

FDR = false discovery rate.

[*] *p* from ANCOVA after adjusting for batch and gender effects.

[a] Batch and gender-adjusted fold change.

Table 4
Genes with differentially expressed probesets from matched samples analysis using RMA normalization.

Group	Gene symbol	Probesets	[*]p before FDR	[*]p after FDR	[a]Fold change
MAP	LOC101928211	1559930_at	5.01E − 05	0.99	1.16
Pre: $n = 17$	*PLA2G16*	235110_at	1.49E − 04	0.99	1.24
Post: $n = 17$					
HEP	*RPL23AP53*	222225_at	9.42E − 05	0.99	0.81
Pre: $n = 18$					
Post: $n = 18$					

FDR = false discovery rate.
[*] *p* from ANCOVA after adjusting for batch effect.
[a] Batch adjusted fold change.

(GAD), (Table 6). *GRIK2* is also found to be involved in other GAD diseases including echocardiography, triglycerides, tobacco use disorder, autism, schizophrenia, and other psychiatric disorders. None of the probesets is related to any disease in the Online Mendelian Inheritance in Man (OMIM) database. Kyoto Encyclopedia of Genes and Genomes (KEGG) pathway analysis indicates that *GRIK2* and *SLC2A5* which were upregulated for the HEP group are involved in neuroactive ligand-receptor binding (Table 7).

2. Experimental design, materials and methods

2.1. Study design

The study protocol was approved by the National University of Singapore Institutional Review Board. Sixty elderly who met the criteria below were enrolled:

- Between 60 and 85 years old.
- Living in the community.
- Fulfilled the operational criteria/definition of mild cognitive impairment including at least one age-education adjusted neuropsychological test Z score < -1.5, did not meet DSM-IV criteria for dementia, had memory or cognitive complaint which was corroborated by a reliable informant and had intact activities of daily living.
- Could function independently.
- Able to travel on their own to participate in the intervention programme.
- Did not have a neurological condition such as epilepsy or Parkinson's disease.
- Did not have a major psychiatric condition such as major depressive disorder.
- Did not suffer from a terminal illness at the time of enrollment.
- Had no marked upper and lower limb motor difficulties.
- Not participating in another interventional study at the same time.

2.2. Intervention

Participants in the MAP group were taught mindfulness awareness practice techniques modelled on McBee [4]. All participants gathered in a group once a week for 40 min for the first 12 weeks, followed by once a month for 45 min for the subsequent 6 months. They were required to practise these techniques at home daily and keep a record of their practice.

The HEP participants attended weekly talks on healthy living topics such as hypertension, diabetes, dementia, depression, medications, exercise, diet, sleep, home safety, falls and social support.

Table 5
Genes with differentially expressed probesets between post-MAP ($n = 17$) and post-HEP ($n = 19$) from unmatched samples analysis using RMA normalization.

Gene symbol	Probesets	[*]p before FDR	[*]p after FDR	[a]Fold change
LOC101928457	217034_at	1.51E − 15	4.49E − 11	0.57
AHDC1	205002_at	7.54E − 14	1.12E − 09	0.51
ENTHD2	239681_at	1.40E − 13	1.39E − 09	0.62
MIR6716///PHLDB1	212134_at	1.46E − 12	1.08E − 08	0.63
CLEC14A	226244_at	8.10E − 12	4.80E − 08	0.60
LINC00482	243263_at	6.88E − 11	2.27E − 07	0.66
NDUFS1	239268_at	1.12E − 10	3.02E − 07	0.65
C9orf172	236744_at	3.01E − 10	6.86E − 07	0.59
GALNT5	232110_at	3.30E − 10	7.00E − 07	0.58
LOC101928104	217471_at	3.76E − 10	7.44E − 07	0.61
TGFB2	228121_at	4.81E − 10	8.40E − 07	0.55
WWC1	229180_at	1.16E − 09	1.73E − 06	0.60
IKZF4	208472_at	1.74E − 09	2.15E − 06	0.64
KLK14	220573_at	2.64E − 09	2.80E − 06	0.56
LOC283278///PLEKHA7	242417_at	3.69E − 09	3.25E − 06	0.63
UBA6	1555441_at	4.88E − 09	3.92E − 06	0.64
CDC42BPA	203794_at	6.07E − 09	4.40E − 06	0.62
MYCNOS	207028_at	6.46E − 09	4.46E − 06	0.61
C21orf58	238541_at	6.37E − 09	4.46E − 06	0.63
GAS6-AS1	238127_at	6.81E − 09	4.52E − 06	0.51
BPIFC	1555773_at	7.86E − 09	4.95E − 06	0.65
TMC4	226403_at	8.09E − 09	4.95E − 06	0.61
LINC01106///LINC01123	242222_at	1.37E − 08	6.99E − 06	0.61
WDR64	1553373_at	1.98E − 08	9.77E − 06	0.65
ZCCHC5	1552935_at	2.37E − 08	1.00E − 05	0.62
SLC28A1	207560_at	2.37E − 08	1.00E − 05	0.64
SYNE4	235515_at	2.31E − 08	1.00E − 05	0.63
AC100830.4	238024_at	6.99E − 08	2.38E − 05	0.65
MTMR9LP	228593_at	8.74E − 08	2.76E − 05	0.66
KIAA2026	236306_at	9.68E − 08	2.90E − 05	0.64
LINC01208	237274_at	9.62E − 08	2.90E − 05	0.65
LOC101927703	241787_at	9.94E − 08	2.92E − 05	0.63
LOC100130502	1570568_at	2.27E − 07	5.26E − 05	0.61
CHRNA3	210221_at	2.45E − 07	5.47E − 05	0.64
UCA1	227919_at	3.51E − 07	6.92E − 05	0.64
PNPLA7	228383_at	3.67E − 07	7.02E − 05	0.65
RC3H2	238421_at	4.32E − 07	8.12E − 05	0.66
ANO4	229749_at	5.52E − 07	9.63E − 05	0.63
RP11-805I24.3	244284_at	6.59E − 07	1.12E − 04	0.62
PADI1	223739_at	7.83E − 07	1.28E − 04	0.61
MOGAT2	232428_at	8.68E − 07	1.37E − 04	0.64
KIF18B	222039_at	1.10E − 06	1.62E − 04	0.63
RP11-774O3.1///RP11-774O3.2	243408_at	1.12E − 06	1.63E − 04	0.65
PTGFRN	224950_at	1.29E − 06	1.83E − 04	0.61
FFAR1	231761_at	1.30E − 06	1.84E − 04	0.58
TMEM57	241364_at	1.86E − 06	2.39E − 04	0.54
HIST1H2AM	214481_at	2.39E − 06	2.76E − 04	0.63
EPS15L1	243482_at	2.48E − 06	2.82E − 04	0.61
SKA3	227165_at	3.09E − 06	3.22E − 04	0.65
WFIKKN2	241358_at	4.44E − 06	4.13E − 04	0.65
PRR15L	219127_at	5.80E − 06	4.99E − 04	0.61
ZFP57	231236_at	1.17E − 05	8.33E − 04	0.61
CCDC154	244409_at	1.35E − 05	9.29E − 04	0.66
PCDH17	205656_at	2.55E − 05	1.45E − 03	0.65
CAV2	203323_at	2.74E − 05	1.52E − 03	0.65
GTF2A2	243985_at	4.30E − 05	2.10E − 03	0.65
KRTAP5-2	1566218_at	1.04E − 04	3.85E − 03	0.66
DYNC1I2	236698_at	1.05E − 04	3.87E − 03	0.63
IRF5	239412_at	1.41E − 04	4.70E − 03	0.46

Table 5 (*continued*)

Gene symbol	Probesets	*p before FDR	*p after FDR	[a]Fold change
TPBG	203476_at	1.76E − 04	5.41E − 03	0.58
CACNB4	207693_at	1.90E − 04	5.75E − 03	0.65
TRIM67	233357_at	2.05E − 04	6.03E − 03	0.60
IQGAP3	241939_at	2.72E − 04	7.43E − 03	0.62
DQ592442	1558444_at	2.78E − 04	7.53E − 03	0.58
MAOA	212741_at	1.17E − 03	1.97E − 02	0.60
OLFM1	213131_at	4.26E − 03	4.62E − 02	0.56
MIR146A	238225_at	4.60E − 11	1.71E − 07	1.67
PYROXD1	231173_at	1.49E − 09	2.04E − 06	1.91
SEZ6	229651_at	2.07E − 09	2.33E − 06	1.53
ILDR1	1553545_at	2.90E − 09	2.87E − 06	1.59
ATG12	213930_at	3.00E − 09	2.87E − 06	1.76
TMCO3	220241_at	3.84E − 09	3.25E − 06	1.66
ANKRD11	226012_at	5.33E − 09	4.09E − 06	1.60
RP11-876N24.5	1569127_at	1.00E − 08	5.82E − 06	1.66
CCDC120	239403_at	1.29E − 08	6.83E − 06	1.55
STYX	242408_at	2.63E − 08	1.09E − 05	1.57
SH3BP2	217253_at	7.30E − 08	2.46E − 05	1.50
TIAL1	202405_at	2.68E − 07	5.70E − 05	1.69
AC018766.6	235665_at	1.07E − 06	1.61E − 04	1.57
TNPO2	215844_at	3.07E − 05	1.66E − 03	1.51
GGACT	232422_at	4.35E − 05	2.11E − 03	1.67
CAAP1	231995_at	3.16E − 04	8.15E − 03	1.55
RP11-722E23.2	237419_at	9.47E − 04	1.70E − 02	1.55
CLEC12A	243106_at	1.14E − 03	1.94E − 02	2.06
COPG2	223457_at	3.34E − 03	3.96E − 02	1.51
ZNF44	228718_at	3.58E − 03	4.12E − 02	1.50

FDR = false discovery rate.

* *p* from ANCOVA after adjusting for batch and gender effects.

[a] Batch and gender-adjusted fold change.

The sessions were led by an instructor who had experience conducting similar teaching sessions for the elderly. Participants were provided with hand-outs at each session and were also required to keep a record of activities to complete in-between sessions. The sessions were weekly for 40 min for the first 12 weeks, and then once a month for 6 months.

2.3. Samples and RNA preparation

Blood samples (1–3 ml) were collected in Tempus Blood RNA Tube from 51 subjects before the start of intervention (pre-intervention samples) and 36 subjects after nine months (post-intervention samples). The blood tubes were stored in the freezer at − 80 °C and processed in batches. Total RNA was extracted using Tempus Spin RNA Isolation Kit (Applied Biosystems, CA, USA). The purity and concentration of the total RNA were determined by UV–vis Spectrophotometer (Quawell, CA, USA) and Quantus Fluorometer (Promega, WI, USA). Globin mRNA depletion was performed with a minimum of 800 ng of total RNA using the GLOBINclear Kit (Ambion, TX, USA). The purity and concentration of the GLOBINclear RNA were determined by UV–vis Spectrophotometer and Quantus Fluorometer, respectively. The integrity of the GLOBINclear RNA was evaluated by RNA ScreenTape assay (Agilent Technologies, Waldbronn, Germany).

2.4. Microarray and quality control

Eighty-seven samples (51 pre-intervention, 36 post-intervention) were processed in batches of 4–12 samples. cRNA was synthesised from 100 ng of GLOBINclear RNA using GeneChip 3' IVT PLUS Reagent Kit according to the manufacturer's protocol. Fragmented and labeled cRNA (11 μg) was

Table 6
Genes involved in GAD disease.

GAD disease	Probesets	Gene	*p-value
Cholesterol, HDL	216057_at 215655_at 233229_at	*RAB3GAP2* *GRIK2* *SCFD1*	0.042
Tobacco Use Disorder	1569349_at 215655_at 202442_at 233229_at 230705_at	*C11orf30* *GRIK2* *AP3S1* *SCFD1* *SLC2A5*	0.188
Several psychiatric disorders	215655_at 221362_at	*GRIK2* *HTR5A*	0.208
Autism	215655_at 221362_at	*GRIK2* *HTR5A*	0.244
Echocardiography	215655_at 233229_at	*GRIK2* *SCFD1*	0.261
Triglycerides	215655_at 233229_at	*GRIK2* *SCFD1*	0.290
Schizophrenia	215655_at 221362_at	*GRIK2* *HTR5A*	0.464

Thresholds set at minimum count of 2 and maximum EASE score (*p*-value) of 0.5.
* *p*-value of a modified Fisher's exact test (EASE score) from DAVID.

Table 7
Genes involved in KEGG pathway.

KEGG pathway	Probesets	Gene	*p-value
Neuroactive ligand-receptor interaction	215655_at, 221362_at	*GRIK2* *HTR5A*	0.12

Thresholds set at minimum count of 2 and maximum EASE score (*p*-value) of 0.5.
* *p*-value of a modified Fisher's exact test (EASE score) from DAVID.

hybridised to Affymetrix GeneChip Human Genome U133 Plus 2.0 arrays according to the manufacturer's protocol. Arrays were scanned using the Affymetrix GeneChip 3000 7G with autoloader and the captured images were analyzed using Affymetrix GeneChip Command Console version 4.3.2. Metric analysis was carried out according to the instructions provided by manufacturer using the Affymetrix Expression Console 1.4.1.46. Relative Log Expression (RLE) generated from RMA normalization using Affymetrix Expression Console was used to ensure the compatibility across the all samples and identify the outliers.

All available data were used to compare the difference in gene expression between pre- and post-intervention for unmatched samples analysis of each intervention group. For matched samples analysis, only data from subjects with both pre- and post-intervention RNA samples were included. A total of 29,663 probesets with unique gene annotation were selected out of 54,676 raw probesets for analysis. Analysis of Covariance (ANCOVA) was performed for both unmatched samples analysis and matched samples analysis. Batch and gender effects were adjusted for the unmatched sample analysis

while only batch effect was adjusted in the matched sample analysis. Array data was normalized using (i) Microarray Suite 5 (MAS5) and subsequently log2 transformed, and (ii) Robust Multi-array Average (RMA) methods [5]. R software was used for normalization and differential gene expression analysis, and control of the false discovery rate (FDR) for multiple testing [6]. Gene expression is considered to show up-regulation in the post-intervention group if the adjusted fold change is more than 1.5 and downregulation if the adjusted fold change is less than 0.66. *P-values* < 0.05 (after FDR) were considered as statistically significant for differential gene expression (Tables 1–5).

Hierarchical clustering was applied to both samples and signature gene set for (i) before FDR for pre-/post-HEP, (ii) pre-/post-MAP, (iii) after FDR and fold change cut-off (FC > 1.5 or FC < 0.66) for post-HEP/post-MAP, shown in the format of dendrogram in combination with a heatmap of gene expression levels.

2.5. Pathway analysis

The differentially expressed genes from MAS5 normalization were used for enrichment analysis of pathways and diseases using version 6.8 of DAVID [7]. The list of probesets from Tables 1 and 2 was entered into the web application (david.ncifcrf.gov), with “AFFYMETRIX_3PRIME_IVT_ID” selected as the gene identifier and Homo sapiens selected as the species. Diseases were identified by the GAD and OMIM databases, and biochemical pathway by KEGG. Gene enrichment in annotation terms was measured by modified Fisher's exact test (EASE score). Functional annotation chart was visualized using minimum of 2 count and maximum EASE score (p-value) of 0.5. EASE score (p-value) of < 0.05 was considered as statistically significant (Tables 6 and 7).

Acknowledgements

This study was funded by the MIND-BODY Interest Group, National University of Singapore, Kwan Im Thong Cho Temple Elderly Depression Prevention Project (N-177-000-004-0001); and the National Research Foundation Singapore under NMRC/CG/006/2013 and NMRC/CG/M003/2017 administered by the Singapore Ministry of Health's National Medical Research Council. The funding sources have no role in the study design, in the collection, analysis and interpretation of the data, in the writing of the report, and in the decision to submit the article for publication.

Transparency document. Supplementary material

Supplementary data associated with this article can be found in the online version at http://dx.doi.org/10.1016/j.dib.2018.03.086.

References

[1] I. Buric, M. Farias, J. Jong, C. Mee, I.A. Brazil, What is the molecular signature of mind-body interventions? A systematic review of gene expression changes induced by meditation and related practices, Front. Immunol. 8 (2017) 670.
[2] P. Kaliman, M.J. Alvarez-Lopez, M. Cosin-Tomas, M.A. Rosenkranz, A. Lutz, R.J. Davidson, Rapid changes in histone deacetylases and inflammatory gene expression in expert meditators, Psychoneuroendocrinology 40 (2014) 96–107.
[3] M.K. Bhasin, J.A. Dusek, B.H. Chang, M.G. Joseph, J.W. Denninger, G.L. Fricchione, H. Benson, T.A. Libermann, Relaxation response induces temporal transcriptome changes in energy metabolism, insulin secretion and inflammatory pathways, PLoS One 8 (2013) e62817.
[4] L. McBee, Mindfulness-Based Elder Care: A CAM Model for Frail Elders and Their Caregivers, Springer, New York, 2008.
[5] R.A. Irizarry, B.M. Bolstad, F. Collin, L.M. Cope, B. Hobbs, T.P. Speed, Summaries of affymetrix GeneChip probe level data, Nucleic Acids Res. 31 (2003) e15.
[6] Y. Pawitan, S. Michiels, S. Koscielny, A. Gusnanto, A. Ploner, False discovery rate, sensitivity and sample size for microarray studies, Bioinformatics 21 (2005) 3017–3024.
[7] D.W. Huang, B.T. Sherman, R.A. Lempicki, Systematic and integrative analysis of large gene lists using DAVID bioinformatics resources, Nat. Protoc. 4 (2009) 44–57.