

Supplemental Tables

microRNA	FC	padj
hsa-miR-3909	1.9443348	0.0002312
hsa-miR-30d-5p	1.5094323	0.0062502
hsa-miR-6803-3p	0.6747345	0.0122642
hsa-miR-30a-5p	1.6681270	0.0206559
hsa-miR-92a-3p	1.3888891	0.0423676
hsa-miR-1249-3p	0.6133912	0.0480158
hsa-miR-222-3p	1.4345961	0.0480158

Table S1. Differentially expressed microRNAs in the music-performance versus control comparison from DESeq2 (Love, Huber & Anders, 2014) analysis. The columns 1-3 show the DE microRNA, fold change and adjusted p-value for music-performance versus control study comparison.

Intragenic DE microRNAs

DE microRNA	Precursor miRNA	Mature miRNAs	Family	Family Conservation	Clustered miRNAs (10kb from the DE microRNA)	Total no. of predicted targets from Targetscan 7.2	Band	Chromosome	Start	End	Host gene
hsa-miR-3909	hsa-mir-3909	hsa-miR-3909	miR-3909	poorly conserved; well annotated	hsa-mir-3909 and hsa-mir-6069	3547 transcripts	22q12.3	chr22	35335640	35335758	TOM1
hsa-miR-6803-3p	hsa-mir-6803	hsa-miR-6803-5p, hsa-miR-6803-3p	miR-6803-3p	other miRBase annotation	hsa-mir-6803 and hsa-mir-6802	1035 transcripts	19q13.42	chr19	55245186	55245250	PPP6R1
hsa-miR-1249-3p	hsa-mir-1249	hsa-miR-1249-3p, hsa-miR-1249-5p	miR-1249-3p	conserved		16 transcripts	22q13.31	chr22	45200954	45201019	KIAA0930

Intergenic DE microRNAs

DE microRNA	Precursor miRNA	Mature miRNAs	Family	Family Conservation	Clustered miRNAs (10kb from the DE microRNA)	Total no. of predicted targets from Targetscan 7.2	Band	Chromosome	Start	End	
hsa-miR-30a-5p	hsa-mir-30a	hsa-miR-30a-5p, hsa-miR-30a-3p	miR-30-5p	broadly conserved	hsa-mir-30d: hsa-mir-30d and hsa-mir-30b	1566 transcripts	6q13	chr6	71403551	71403621	
hsa-miR-30d-5p	hsa-mir-30d	hsa-miR-30d-5p, hsa-miR-30d-3p	miR-30-5p	broadly conserved		1566 transcripts	8q24.22	chr8	134804876	134804945	
hsa-miR-222-3p	hsa-mir-222	hsa-miR-222-5p, hsa-miR-222-3p	miR-221-3p/222-3p	broadly conserved	hsa-mir-222 and hsa-mir-221	496 transcripts	Xp11.3	chrX	45747015	45747124	
hsa-miR-92a-3p	hsa-mir-92a-1	hsa-miR-92a-1-5p, hsa-miR-92a-3p	miR-25-3p/32-5p/92-3p/363-3p/367-3p	broadly conserved	hsa-mir-17, hsa-mir-18a, hsa-mir-19a, hsa-mir-20a, hsa-mir-19b-1, hsa-mir-92a-1	1037 transcripts	13q31.3	chr13	91351314	91351391	
hsa-miR-92a-3p	hsa-mir-92a-2	hsa-miR-92a-3p, hsa-miR-92a-2-5p	miR-25-3p/32-5p/92-3p/363-3p/367-3p	broadly conserved	hsa-mir-106a, hsa-mir-18b, hsa-mir-20b, hsa-mir-19b-2, hsa-mir-92a-2, hsa-mir-363	1037 transcripts	Xq26.2	chrX	134169538	134169612	

Table S2. Genomic location of DE microRNA transcript from HGNC (Yates et al., 2017) and miRIAD database (Hinske et al., 2014), conservation status, microRNA family information and number of predicted target genes of DE microRNAs from TargetScan Release 7.2 (Agarwal et al., 2015) and microRNA cluster information from miRBase (Kozomara & Griffiths-Jones, 2014) are given.

DE microRNA Tissue expression data and functions from literature

miR-222	<p>hsa-miR-222-3p is located on Xp11.3 close to Xp11.4, a locus for an X-linked disorder characterised by deafness (Berger et al., 1992) and near to Xp11.23, which shows linkage for non-musical creativity (Oikkonen et al., 2016a). miR-222 is expressed in adult human brain (Burmistrova et al., 2007), human and macaque prefrontal cortex and cerebellum (Hu et al., 2011), mouse brain (Babak et al., 2004), mammalian brains, neurons and cranial ganglia of fore and mid brain (Wienholds et al., 2005). mir-221 (member of mir-222 cluster) is also expressed in mammalian (mouse and rat) primary cortical neurons (Kim et al., 2004) and new born mouse cochlea and vestibule (Friedman et al., 2009; Friedman & Avraham, 2009). miR-222-3p shows differential expression patterns between hippocampus, cerebellum, and prefrontal cortex across various stages of human brain development including early and late childhood and adolescence (Ziats & Rennert, 2014). miR-222 is also differentially expressed between the sensory epithelium of cochlea and vestibule with roles in the regulation of cellular fate during development and in the maintenance of their functions and morphology (Elkan-Miller et al., 2011; Rudnicki et al., 2014). Increased levels of miR-222 was observed during oligodendrocyte maturation (Lau et al., 2008). miR-221, member of the same cluster as the miR-222, is enriched in distal axons (Natera-Naranjo et al., 2010) and expressed during hippocampal neuronal development (Spronsen et al., 2013). miR-222 targets the cell cycle inhibitor <i>CDKN1B</i> which encodes p27 protein and shows pro-proliferative functions (Sage et al., 2007; Frenquelli et al., 2010), whereas, in the neuroblastoma cells, miR-222 is activated by p53 and inhibit proliferation (Rihani et al., 2015); hence shows context-dependent functions and represents a switch hub for apoptosis, survival and proliferation; similar to its transcriptional regulator, p53 protein (Rihani et al., 2015). miR-222 is involved in the sensory perception of sound (Zhang et al., 2013). miR-222 is up-regulated in schizophrenia compared to healthy subjects (Perkins et al., 2007) with possible roles in MAPK pathway. In Huntington's disease, a hereditary neurodegenerative disease, miR-222 is found down-regulated in the Brodmann's area 4 (Packer et al., 2008), frontal cortex and striatum (Martí et al., 2010). In AD, miR-222 is found down-regulated in the brain and blood whereas p27 was up-regulated (Wang et al., 2015). miR-221, which belongs to the same cluster as miR-222, is found to be analogous to miR-125a-5p whose increased expression in human brain endothelial cells enhanced the blood brain barrier and helped to form an extensive, continuous junctional complex (Reijerkerk et al., 2013). This is of significance in inflammatory diseases like multiple sclerosis where reduced levels of miR-125a-5p contributed to pro-inflammatory TNFα mediated intracellular cell adhesion molecule (ICAM) and monocyte migration across the blood brain barrier. According to SHIELD database (Shen et al., 2015), miR-222 is expressed in adult cochlear inner and outer hair cells and takes part in cellular response to amino acid stimulus and Wnt receptor signaling pathway. miR-222 was also found down-regulated when exposed to various types of environmental stress inducers including thermal stress (Wilmink et al., 2010) and cigarette smoke (Izzotti et al., 2009).</p>
miR-30d	<p>Located on 8q24.22, near to 8q24 which has been implicated in absolute pitch (Theusch, Basu & Gitschier, 2009), auditory neuropathy (Rance, 2005) and Alzheimer's disease (AD) (Sillén et al., 2011). 8q24 is also the locus for <i>AGO2</i> and <i>MYC</i>, upstream regulators of the candidate genes in music-performance (Kanduri et al., 2015a), <i>ARC</i>, a top candidate for musical ability (Oikkonen et al., 2016b) and <i>FAM49B</i>, one of the up-regulated genes from the <i>high COMB</i> group (Kanduri et al., 2015b). In music genetic studies, a duplication of the 8q24 region has been noted in individuals with low combined musical score (low <i>COMB</i>) (Ukkola-Vuoti et al., 2013). miR-30d is expressed in adult human brain (Burmistrova et al., 2007), prefrontal cortex and cerebellum of adult macaque, chimpanzee and human (Hu et al., 2011), mouse brain (Babak et al., 2004), rat cortical neurons (Kim et al., 2004) and during neuronal development in the hippocampal neurons (Spronsen et al., 2013). miR-30d is expressed in adult cochlear inner and outer hair cells and is involved in cellular response to amino acid stimulus (Shen et al., 2015) and sensory perception of sound (Zhang et al., 2013). miR-30d-5p is amongst the top 40 most expressed microRNA out of the 455 identified in mouse cochlear or vestibular sensory epithelium (Rudnicki et al., 2014) and is also found differentially expressed between hippocampus and cerebellum in adolescent human brain (Ziats & Rennert, 2014). All other members of miR-30 family are also expressed in the cochlear sensory epithelium with significant down-regulation of miR-30d in the cochlea after a day of acoustic over-stimulation or noise exposure and associated sensory cell degeneration (Patel et al., 2013). miR-30a-5p, a cluster member of up-regulated miR-30d is expressed in new borne mouse vestibular sensory epithelium (Friedman et al., 2009; Friedman & Avraham, 2009). miR-30d is increased during oligodendrocyte maturation (Lau et al., 2008) possibly suggestive of its role. miR-30 family, including miR-30d, target <i>RASA2</i>, which represses ERK activation in the mouse embryonic stages thereby regulating the ERK signalling pathway that has important functions in cellular homeostasis, cell identity, proliferation and plasticity (Pernaute et al., 2011). hsa-miR-30d-5p is down-regulated in dicer deficient human temporal lobe epileptic hippocampus which might be one of the contributing factors to hippocampal sclerosis (McKiernan et al., 2012). A possible functional role for miR-30 in the visual system is supported by its high expression in Zebra fish lens (Xu, 2009) and in dicer deficient mouse photoreceptor rod (Sundermeier et al., 2014) where it might support rod survival in retinal degeneration. In AD, miR-30d is up-regulated in the CSF (Cogswell et al., 2008) and miR-30e-5p (mir-30 family) is up-regulated in hippocampus (Satoh, 2010). Whereas, miR-30e and miR-30b is found down-regulated in the prefrontal cortex of schizophrenia patients (Perkins et al., 2007).</p>
miR-3909	<p>miR-3909 is located on 22q12.3, whose deletions (22q12.3-q13.1) are associated with neurocristopathy, and sensorineural hearing loss (Jelena et al., 2014). Its preceding region, 2q11.1-21 is the best linked region for musical aptitude (Oikkonen et al., 2015) and 22q11 is a locus for neuropsychiatric diseases including schizophrenia (Squarcione et al., 2013).</p>
miR-92a	<p>miR-92a-3p is transcribed from 2 locations, Xq26.2 and 13q31. Microdeletion of 13q31.3-q32.1 region has been linked to problems associated with intelligence (Valdes-Miranda et al., 2014). 13q31 is also a locus susceptible for epilepsy (Tauer et al., 2005; EPICURE Consortium et al., 2012), dysplastic ear and psychomotor delays (Grigori et al., 2011). 13q31.3 has been identified as a risk locus for multiple sclerosis (Comabella et al., 2008), a disease of the nervous system characterised by loss of myelin. Expressed in the cochlear and vestibular sensory epithelia (cochlea and vestibule) (Weston et al., 2006; Rudnicki et al., 2014) and in human and mouse brains with roles in neuron differentiation (Sempere et al., 2004). Mammalian miR-92 shows wide expression in most parts of the developing and adult central nervous system with higher intensity in the developing vestibulocochlear ganglion, a region associated with sound perception balance perception from the inner ear signals (Deo et al., 2006). Also expressed in the prefrontal cortex and cerebellum of adult macaque, chimpanzee and human (Hu et al., 2011). Co-expressed along with other cluster members from mir-17 cluster (mir-17, mir-18a, mir-19a, mir-20a, mir-19b-1, hsa-mir-92a) in the developing brain (Miska et al., 2004). miR-92 was profoundly decreased in the brain tissues after traumatic brain injury where a concurrent increase in the β-APP (β-amyloid precursor) and IL-1β are noted (Pinchi et al., 2018).</p>
miR-30a	<p>Expressed in the cochlear and vestibular sensory epithelia (cochlea and vestibule) (Weston et al., 2006; Rudnicki et al., 2014), embryonic and adult cortex, striatum and hippocampus (Landgraf et al., 2007) and developing mammalian brain (Lagos-Quintana et al., 2002) including that of humans with roles in neuronal differentiation (Sempere et al., 2004). miR-30a found to be under the regulation of Dicer and was up-regulated during myelination in the CNS and the PNS (Bremer et al., 2010). The database SHIELD also shows miR-30a expression in adult cochlear inner and outer hair cells (Shen et al., 2015). Also expressed in the prefrontal cortex and cerebellum of adult macaque, chimpanzee and human (Hu et al., 2011). hsa-miR-30a-5p is located on 6q13 in the 6q13-q16.1 region, one of the loci for otosclerosis (OMIM:611572) and sensorineural hearing loss (Thys et al., 2007). It is also located adjacent to <i>PHIP</i> (location: 6q14.1), one of the top 40 genes linked to musical abilities from the convergent genomic analysis of musical traits (Oikkonen et al., 2016b). Deletion of 6q13-6q14.1 region has been associated with delays in psychomotor activities and language development (Lespinasse et al., 2009; Mascheretti et al., 2017; Parmeggiani et al., 2017).</p>

Table S3. Candidate microRNAs associated with music-performance of professional musicians, their expression patterns and relevant functional information.

Comparison

Target genes of down-regulated microRNAs and singing stimulated genes from songbirds (Wada et al., 2006; Hilliard et al., 2012; Whitney et al., 2014)

Target genes of DE microRNAs that show overlap with the compared studies

ACTR8, BCL2L13, CEBPD, DOK6, EPDR1, FAF2, FZD7, GGA1, H2AFY2, HMG20B, HNRNPK, LRRC10B, MAFK, MAMLD1, MAPRE2, MLEC, MYO1F, NAB2, OTUD7A, PDCL, PRKRIP1, PTCH1, RAB11FIP4, RAB35, RANBP1, RASD1, SERPINF1, SLC25A25, SLC4A2, SLC6A6, SLMO1, SNPH, SPOPL, SRRM3, STAU2, SYTL4, TGFBRAP1, TMEM104, TRIB2, TRIOBP, TXNRD3, UBE2QL1, WDR26, WDR33, ZFAND6

Target genes of up-regulated microRNAs and singing inhibited genes from songbirds (Whitney et al., 2014)

ACACA, AIG1, AJAPI, ANKRD17, ARID4A, ARID4B, ARL5B, ARPP19, ATL2, ATRNL1, ATXN1, B3GALT2, BRMS1L, BTF3L4, CADM2, CAMK2D, CDC42, CNOT1, CRY1, CSMD1, CTCF, CTDSPL2, DBT, DCTN6, DDX3X, DGKI, DIRAS2, DNAJB11, DNAJC21, DUSP6, DYNC111, EBAG9, EDEM3, ELK4, ELMO1, EPC1, EXOC6, FAMI3C, FAMI81B, FARP1, FARSA, FGF14, FKBP1A, FOS, FUBP1, GABRA5, GK, GLB1L2, GRAMD1B, GRM7, HECA, HERC2, HNRNPA3, HNRNPH3, HSPA5, ING5, JHDM1D, JMY, KCTD8, KIAA2026, LRRC10B, MAN1B1, MAPK6, MATR3, MOCS2, MRPL50, MTF1, MYO10, NAA25, NAP1L1, NETO1, NEUROD6, NLGN1, NR2F2, NUS1, PCDH10, PDCD10, PDIA3, PDP2, PLK2, POLR1D, PPP2R2A, R3HDM1, RBM5, RIMS1, RPP30, RWDD1, SAMD12, SCN2A, SERTAD4, SLC15A2, SMC6, SRPR, STXBP5, TAF1, TAGLN3, TBL1XR1, TIMP3, TNRC6B, TOP1, TRIOBP, TULP4, UBXN4, URM1, WDR70, YTHDC1, ZNRF1

Table S4. Comparative analyses of the target genes of DE microRNAs with singing regulated genes in songbirds.

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