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3 Imprinted genes in a founder population

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5 Parent of origin gene expression in a founder population identifies two new

6 imprinted genes at known imprinted regions.

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22 ## Abstract

23 Genomic imprinting is the phenomena that leads to silencing of one copy of a gene
24 inherited from a specific parent. Mutations in imprinted regions have been involved in diseases
25 showing parent of origin effects. Identifying genes with evidence of parent of origin expression
26 patterns in family studies allows the detection of more subtle imprinting. Here, we use allele
27 specific expression in lymphoblastoid cell lines from 306 Hutterites related in a single pedigree
28 to provide formal evidence for parent of origin effects. We take advantage of phased genotype
29 data to assign parent of origin to RNA-seq reads in individuals with gene expression data. Our
30 approach identified known imprinted genes, two putative novel imprinted genes, and 14 genes
31 with asymmetrical parent of origin gene expression. We used gene expression in peripheral
32 blood leukocytes (PBL) to validate our findings, and then confirmed imprinting control regions
33 (ICRs) using DNA methylation levels in the PBLs.

34 ## Author Summary

35 Large scale gene expression studies have identified known and novel imprinted genes
36 through allele specific expression without knowing the parental origins of each allele. Here, we
37 take advantage of phased genotype data to assign parent of origin to RNA-seq reads in 306
38 individuals with gene expression data. We identified known imprinted genes as well as two
39 novel imprinted genes in lymphoblastoid cell line gene expression. We used gene expression in
40 PBLs to validate our findings, and DNA methylation levels in PBLs to confirm previously
41 characterized imprinting control regions that could regulate these imprinted genes.

42 ## Introduction

43 Imprinted genes have one allele silenced in a parent of origin specific manner. In humans,
44 approximately 105 imprinted loci have been identified, many of which play important roles in

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45 development and growth[1-3]. Dysregulation of imprinted genes or regions can cause diseases
46 that show parent of origin effects, such as Prader-Willi or Angelman syndrome, among others
47 [2]. Imprinted regions have also been associated with complex traits, such as height and age of
48 menarche [4,5], as well as common diseases such as obesity and some cancers [2]. More than
49 80% of imprinted genes in humans are clustered in genomic regions that contain both maternally
50 and paternally expressed genes, as well as genes that encode non-coding RNAs[2,6]. Parent-
51 specific expression of the genes within a cluster are maintained by complex epigenetic
52 mechanisms at cis-acting imprinting control regions (ICRs) [3], which show parent of origin
53 specific DNA methylation patterns and chromatin modifications[7].

54 Using RNA-seq and allele specific expression (ASE) we can map genes to parental
55 haplotypes and identify those that are expressed when inherited from only the father or only from
56 the mother, a hallmark feature of imprinted loci. Parent of origin effects and imprinted genes
57 have been most elegantly studied in mice, where two inbred strains are bred reciprocally to
58 identify parent of origin effects on gene expression in progeny that have the same genotypes but
59 different patterns of inheritance [8]. Additionally, uniparental inheritance of imprinted regions in
60 mice were associated with abnormal developmental phenotypes [9] before it was shown that
61 imprinting defects are associated with human disease[10,11]. One approach to identifying
62 imprinted loci in humans has been to test for parent of origin effects on gene expression and
63 phenotypes in pedigrees [4,12]. For example, Garg et al. used gene expression in LCLs from
64 HapMap trios to identify 30 imprinting eQTLs with parent of origin specific effects on
65 expression [13]. A study from the GTEx Consortium used RNA-seq data and allele specific
66 expression to identify allelic imbalance in 45 different tissues. By considering genes with
67 monoallelic expression that was evenly distributed to both the reference and alternate alleles

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68 across individuals as evidence for imprinting, they identified 42 imprinted genes, both known
69 and novel, and used family studies to confirm imprinting of 5 novel imprinted genes [14].
70 Santoni et al. identified nine novel imprinted genes using single-cell allele-specific gene
71 expression and identifying genes with mono-allelic expression in fibroblasts from 3 unrelated
72 individuals and probands of 2 family trios, and then used the trios to confirm parent of origin of
73 the alleles [15].
74 Here, we perform a parent of origin ASE study in a large pedigree to characterize parent
75 of origin specific gene expression in the Hutterites, a founder population of European descent,
76 for which we have phased genotype data [16]. We use RNA-seq from lymphoblastoid cell lines
77 (LCLs) to map transcripts to parental haplotypes and identify known and two not previously
78 reported imprinted genes. We validated the two putative imprinted genes by showing the same
79 patterns of parent of origin expression PBLs from different Hutterite individuals, and show DNA
80 methylation signatures of imprinting in the PBLs at these regions.
81

82 Results

83 Mapping transcripts to parental haplotypes

84 For each of 306 individuals, the total number of transcripts at each gene was assigned as
85 maternally inherited, paternally inherited, or unknown parent of origin. The last group included
86 transcripts without heterozygote SNPs or transcripts with SNPs without parent of origin
87 information. Transcripts were assigned to the parentally inherited categories using SNPs in the
88 reads and matching alleles to either the known maternally or paternally inherited alleles. All the
89 genes analyzed had some transcripts of unknown origin (average 97.8%, range 8.3-100%). For
90 each gene we assigned parental origin to an average of 1.8% of transcripts (range: 0-34.7%), and

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91 for each individual we assigned parental origin to an average of 1.4% of transcripts (range: 0-
92 1.7%). On average, about 40 SNPs per gene were used to assign the transcripts of a gene to
93 parent (range 1-1839 SNPs).

94 **Table 1. Summary Statistics for Parental Origin of Transcripts.**

	Mean	Standard Deviation	Range
Proportion of transcripts from each gene assigned to transcripts of unknown origin	0.978	0.031	(0.083, 1)
Proportion of transcripts from each gene assigned to parental origin	0.018	0.019	(0, 0.347)
Proportion of transcripts for each individual assigned to parental origin	0.014	0.0015	(0, 0.017)

95

96 After quality control (see Methods), transcripts in 15,889 genes were detected as
97 expressed in 306 individuals. Some transcripts for 14,791 of those genes could be assigned to a
98 parent. Of these, 75 genes were only expressed on the paternally-inherited allele in at least one
99 individual and not on the maternally inherited allele in any individuals. Similarly, 64 genes were
100 only expressed on the maternally-inherited allele in at least one individual and not on the
101 paternally inherited allele in any individuals (S1 Table).

102 **Imprinted Genes in Lymphoblastoid Cell Lines (LCLs)**

103 Among the 139 genes with only paternally inherited expression or only maternally
104 inherited expression, there are three known imprinted genes (*CDKN1C*, *NDN*, *SNRPN*) and one
105 previously predicted to be imprinted (*IFITM1*) [17]. *CDKN1C* showed patterns opposite of what
106 has been reported [18,19], which could be due to the small sample (only three individuals
107 showed expression from one parent) or to the different cell types used here (LCLs) and in
108 previous studies (developing brain and embryonal tumors for *CDKN1C*).

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109 We expect some imprinted genes to have 'leaky' expression, such that there is some
110 expression from the parental chromosome that is mostly silenced. To detect these genes, we used
111 a binomial test to find patterns of gene expression asymmetry by parental transcript levels. This
112 analysis identified 28 genes with an FDR <5% (Table 2). The 11 genes that showed the most
113 asymmetry are known imprinted genes: *ZDBF2*, *PEG10*, *SNHG14*, *NHP2L1*, *L3MBTL1*,
114 *ZNF331*, *LPAR6*, *FAM50B*, *KCNQ1*, *NAP1L5*, and *IGF1R*. Parent of origin expression for
115 *ZDBF2* and *KCNQ1* are shown in **Fig 1A** and **1B**, respectively. We identified two additional
116 genes that showed asymmetry in parental expression from mostly one parent (*PXDC1, PWAR6*),
117 which we consider potentially new imprinted genes. The remaining fourteen genes showed
118 significant patterns of asymmetry but had expression from both maternal and paternal
119 chromosomes. These genes are likely not imprinted but could have asymmetry in expression due
120 to an expression quantitative trait loci (eQTL).

121 **Table 2. Results for Gene with Parent of Origin Expression Asymmetry.** Genes listed by
122 category of imprinting status: (A) Known Imprinted, (B) Conflicting Evidence for Imprinted
123 Status, (C) New Imprinted Genes, (D) Genes with Asymmetrical Parent of Origin Expression.
124 Genes are ordered by significance within each category.

125

Gene	p-value	Number of individuals with more maternal expression than paternal expression	Number of individuals with more paternal expression than maternal expression	References
A. Known Imprinted				
ZDBF2	1.59e-41	2	148	geneimprint.com, Baran et al.[14], and Babak et al.[8]
PEG10	5.51e-38	2	136	geneimprint.com, Baran et al.[14], and Babak et al.[8]

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SNHG14	1.64e-36	2	131	Baran et al. [14]
NHP2L1	1.24e-33	23	189	Babak et al. [8] and Docherty et al.[20]
L3MBTL1	6.72e-31	2	107	geneimprint.com and Li et al.[21]
ZNF331	4.05e-25	36	184	Daelemans et al. [22]and Baran et al. [14]
LPAR6	2.65e-23	0	76	Baran et al. [14]
FAM50B	5.29e-23	0	75	geneimprint.com, Baran et al. [14]
KCNQ1	1.34e-22	79	1	geneimprint.com, Baran et al. [14]
NAP1L5	3.76e09	0	29	geneimprint.com
IGF1R	1.11e-05	14	49	Geneimprint.com, Sun et al. [23,24], Boucher et al. [25], Al Adhami et al. [26]
B. Conflicting Evidence for Imprinting Status in the literature				
PRIM2	5.53e-05	30	71	geneimprint.com, Santoni et al. [15]
C. New Imprinted Genes				
PXDC1	9.83e-14	12	81	-
PWAR6	2.27e-13	0	43	-
D. Genes with Asymmetrical Parent of Origin Expression				
SNHG17	6.2e-08	113	45	-
ZNF813	8.7e-07	63	132	-
DAAM1	1.78e-05	66	126	-
RP11-379H18.1	2.09e-05	52	106	-
HMGN1P38	2.43e-05	32	6	-
MTX2	3.05e-05	0	16	-
ZNF714	4.61e-05	35	79	-
MAF1	4.45e-05	17	51	-
IL16	5.71e-05	61	115	-
CPNE1	5.56e-05	111	58	-

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ATP6V0D1	7.03e-05	32	7	-
FAHD1	9.34e-05	68	29	-
CNN2	1.18e-04	127	72	-
HSP90AB3P	1.16e-04	7	31	-

126

127 Two genes showed gene expression signatures consistent with imprinting but have not
128 previously been recognized as imprinted genes. The first potentially new imprinted gene is
129 *PXDC1*, which is in the same region and next to (<100kb) a known imprinted gene, *FAM50B*.
130 The second potentially novel imprinted gene is *PWAR6*, or Prader Willi Angelman Region
131 RNA6, a gene encoding a regulatory class of RNA. Although this gene is located within the
132 intron of a known imprinted gene, *SNHG14*, this noncoding RNA has not previously been
133 recognized as having parent of origin specific expression (**Fig 1C**).

134 The remaining fourteen genes show significant asymmetry using the binomial test but do
135 not have expression from mostly one parental chromosome. One of these genes, *SNHG17*, is a
136 noncoding RNA. Another gene with parent of origin asymmetry, *ZNF813*, is next to a known
137 imprinted gene, *ZNF331*. The remaining genes with asymmetrical parent origin expression have
138 expression from both parental chromosomes, unlike imprinted genes. These genes include
139 *DAAM1*, which is involved in cytoskeleton, specifically filopodia formation [27,28], and has a
140 suggested role for cytoskeleton organization during Mammalian testis morphogenesis and
141 gamete progression [29]; *RP11-379H18.1*, a noncoding RNA gene; *HMGN1P38* [30]; *MTX2*, a
142 nuclear gene that interacts with mitochondrial membrane protein metaxin 1 and is involved in
143 mitochondrial protein import and metabolism of proteins in mice; *MAF1*, a negative regulator of
144 RNA polymerase 2; *ZNF714*, *CPNE1*, *IL16*, *ATP6V0D1*, *FAHD1*, *HSP90AB3P*, and *CNN2* are

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145 the remaining genes that show parent of origin asymmetry but not with a pattern consistent with
146 imprinting (**S1 Figure**).

147 **Validation of Imprinted Genes in PBLs**

148 Using the same methods described above, we assigned parent of origin to transcripts in
149 PBLs from 99 Hutterite individuals not included in the LCL studies. Maternal and paternal
150 expression in PBLs for all 28 genes identified in LCLs showed similar trends of asymmetry as in
151 LCLs (**Fig 2**).

152 **Methylation at Imprinting Control Regions.**

153 One of the mechanisms underlying parent of origin effects on expression at imprinted
154 loci is differential methylation at cis-acting imprinting control regions (ICRs). DNA methylation
155 from the Illumina HumanMethylation 450K array was available in PBLs from the same
156 individuals included in the validation study described above. To determine the expected patterns
157 of methylation at known imprinted loci, we first looked at previously characterized methylated
158 regions at known imprinted regions from Court et al. and Joshi et al. [31,32].

159 The methylation patterns at the two potentially novel imprinted genes identified in this
160 study, *PXDC1* and *PWAR6*, lie in or near known imprinted regions that contain previously
161 characterized ICRs. These previously characterized ICRs show about 50% methylation (beta
162 value of between 0.25 and 0.75) in our DNA methylation data, which likely reflect methylation
163 at only one parental chromosome in all the cells in the sample. Methylation patterns in PBLs at
164 these two ICRs fall within this hemi-methylation range, further suggesting that these two genes
165 are indeed imprinted (**Fig 3**).

166

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167 # Discussion

168 Dysregulation of imprinted genes can have a large impact on mammalian development
169 and has been associated with significant diseases in humans. Studies aimed at identifying
170 imprinted genes at genome-wide levels have used allele specific expression and imbalance to
171 infer parent of origin. Here we used a large pedigree with assigned parent of origin alleles to map
172 transcripts to chromosomes with known parent of origin and identify imprinted genes.

173 Using this approach, we found genes with expression primarily from either the maternal
174 or paternal haplotype. Because gene silencing at imprinted loci may be incomplete, we used a
175 binomial test on parent of origin gene expression and identified 11 known imprinted genes and
176 two potentially novel imprinted genes. Both of these novel genes, *PWAR6* and *PXDC1*, lie in
177 known imprinted regions but have not themselves been characterized as imprinted. The
178 remaining genes that have significant parent of origin asymmetry in gene expression do not show
179 clear imprinting expression patterns. To validate these findings, we mapped gene expression in
180 PBLs from Hutterite individuals not included in the LCL study. The same genes showed similar
181 patterns of asymmetry in these different cell sources (transformed B cells and peripheral blood
182 leukocytes) from different individuals.

183 We also characterized methylation patterns near genes showing asymmetry. Using results
184 from studies that had previously characterized ICRs in patients with uniparental disomy at many
185 imprinted regions [31,32], we estimated regions for defining hemi-methylation near the genes
186 identified in our study. Using this approach, we were able to provide additional supportive data
187 for the two potentially new imprinted genes to be true imprinted genes regulated by previously
188 characterized ICRs.

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189 Although our study is the largest pedigree-based study to date to search genome-wide for
190 imprinted genes, it has limitations. First, we are able to determine the parent of origin for a many
191 transcripts in the Hutterites but we could not assign every RNA sequencing read to a parent due
192 to lack of heterozygous sites or missing parent of origin information for alleles. Second, we
193 conducted these studies in lymphoblastoid cell lines, and therefore could only study genes
194 imprinted in this cell type and would miss the many imprinted genes that are tissue-specific
195 and/or developmentally regulated[33]. Third, while we can verify previously characterized ICRs,
196 our study is not designed to identify novel ICRs because DNA methylation values from an array
197 cannot be assigned to parental haplotype. Lastly, although we characterized the gene expression
198 and methylation patterns for two potentially novel imprinted genes, replication of these genes in
199 a different population and in different tissues, and functional characterization of these genes are
200 required to confirm their status as imprinted genes. Similarly, some of the other genes with
201 parent of origin asymmetry in the blood cells examined in this study may show more clear-cut
202 evidence for imprinting in other tissues or at specific periods of development.

203 In summary, we have identified two new imprinted genes using gene expression from a
204 founder population. The genes with asymmetrical parental expression had similar patterns of
205 asymmetry in a different source of blood cells and in different individuals, and we were able to
206 replicate the methylation patterns in known ICRs near the known and novel imprinted genes in
207 this study. Our method and study population allowed us to map reads to parental haplotypes and
208 uncovered *PWAR6* and *PXDC1* as new imprinted genes that could potentially impact disease risk
209 and development.

210

211

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212 # Methods

213 **Genotypes**

214 Hutterite individuals (n=1,653) were genotyped using one of three Affymetrix genotype
215 arrays, as previously described [16], of which 121 underwent whole genome sequencing by
216 Complete Genomics, Inc (CGI) (n=98) or Illumina whole genome sequencing (n=27). A total of
217 10,235,233 variants present in the sequenced individuals were imputed and phased to the
218 remaining 1,532 genotyped individuals using PRIMAL [16]. Parent of origin was assigned to
219 89.85% of the alleles with call rate 81.6842% after QC. For this study, we included individuals
220 with genotyped parents in the primary analyses in LCLs. Written consents for these studies were
221 obtained from the adult participants and parents of children under 18; written assents were
222 obtained from all children. This study was approved by the University of Chicago Institutional
223 Review Board.

224 **RNA-seq in Lymphoblastoid Cell Lines (LCLs).**

225 RNA-seq was performed in LCLs as previously described [34]. For this study,
226 sequencing reads were reprocessed as follows. Reads were trimmed for adaptors using Cutadapt
227 (reads less than 5 bp discarded) then remapped to hg19 using STAR indexed with gencode
228 version 19 gene annotations [35,36]. To remove mapping bias, reads were processed and
229 duplicate reads removed using WASP [37]. We used a custom script modified from WASP to
230 separate reads that overlap maternal alleles or paternal alleles. Reads without informative SNPs
231 (homozygous, or no parent of origin information) were categorized as unknown where the
232 unknown, maternal, and paternal make up the total gene expression. Gene counts were quantified
233 using STAR for each category. VerifyBamID was used to identify sample swaps [38]. Genes

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234 mapping to the X and Y chromosome were removed; genes with a CPM log transformed value
235 less than 1 in less than 20 individuals were also removed.

236 **RNA-seq in Peripheral Blood Leukocytes (PBLs)**

237 RNA-seq was performed in whole blood as previously described [39]. For this study,
238 sequencing reads were reprocessed as described above for the studies in LCLs. For these
239 analyses, we excluded 32 individuals who were also in the LCL study.

240 **Identifying Imprinted Genes**

241 We used a binomial test to detect asymmetry in parent of origin gene expression. Using
242 the paternally and maternally assigned reads, we generated a binomial Z-score for each
243 individual for each gene (Z_i) and excluded those where Z_i=0. For each gene, the number of
244 subjects with Z_i >0 can be modeled by a Binomial distribution with probability ½, under the null
245 hypotheses of symmetric expression . For imprinted genes that show patterns of asymmetry, we
246 expect a distribution of Z-scores that are skewed to one direction corresponding to asymmetric
247 expression. Because we are only asking whether there are more individuals with more maternal
248 expression or more paternal expression and not gene expression measures there is no need to
249 model over-dispersion.

250 **DNA methylation profiling and processing in PBLs**

251 One milliliter of whole blood from 145 Hutterites was drawn into TruCulture (Myriad
252 RBM; Austin, Texas) tubes containing proprietary TruCulture media. DNA was extracted using
253 AllPrep DNA/RNA Mini Kits (Qiagen). DNA samples were bisulfite converted and hybridized
254 to the Illumina HumanMethylation 450K array at the University of Chicago Functional
255 Genomics Center. Samples were processed using default parameters using the R package minfi
256 [40], normalized using SWAN (subset within-array normalization [41]) and quantile normalized

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257 similar to previous methylation studies [42]. Probes were removed if: (1) mapped non-uniquely
258 to a bisulfite-converted genome; (2) mapped to sex chromosomes; (3) had a probe detection p-
259 value >0.01 in at least 25% of samples; and (4) contained common SNPs within the probe
260 sequence, as previously described [43]. Principal components analysis (PCA) was used to
261 identify significant technical covariates, and the ComBat function [44] within the R package sva
262 [45] was used to correct for chip effect. Analyses of DNA methylation levels were conducted
263 using beta values, which were converted from M-values using the lumi R package [46].
264

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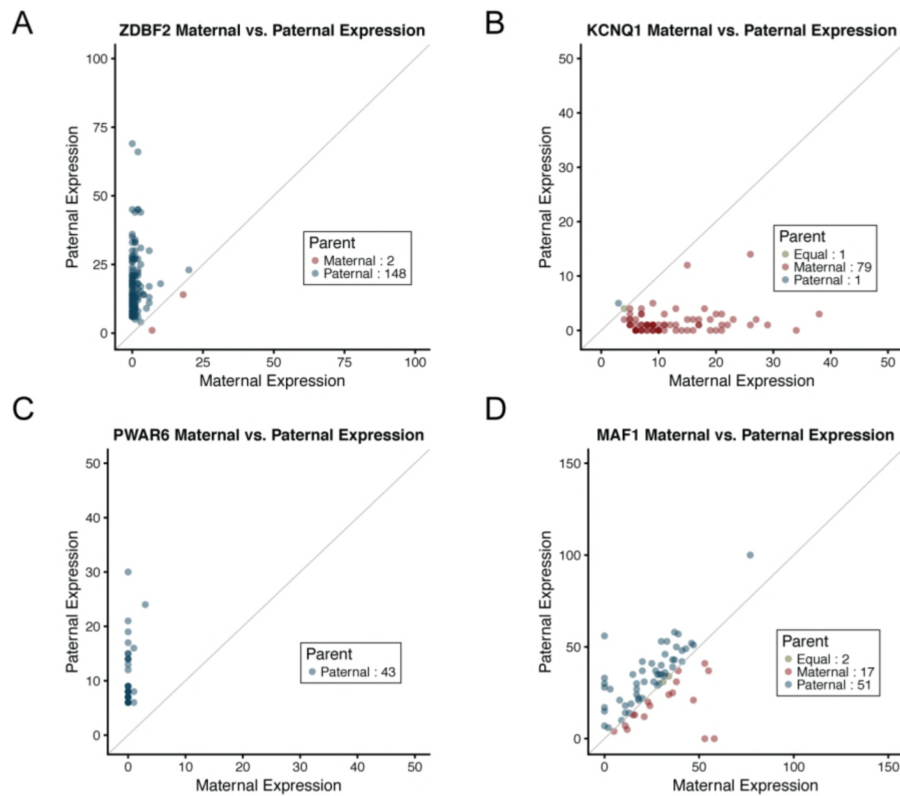
420 # Figures



421

422 **Figure 1.** Plot of maternal (x-axis) and paternal (y-axis) gene expression for four genes. **(A)**
423 maternally imprinted gene *ZDBF2* (paternally expressed), **(B)** paternally imprinted gene *KCNQ1*
424 (maternally expressed), **(C)** novel maternally imprinted gene *PWAR6* (paternally expressed), **(D)**
425 gene with asymmetry in parental expression *MAF1*. Each point represents one individual.
426 Numbers in the legend represent the number of individuals with equal maternal and paternal
427 expression, more maternal expression, or more paternal expression.

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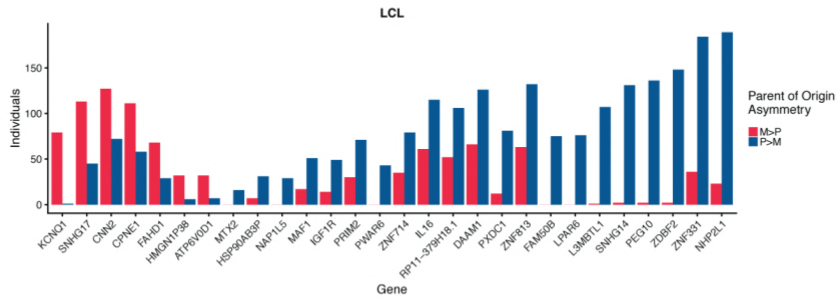

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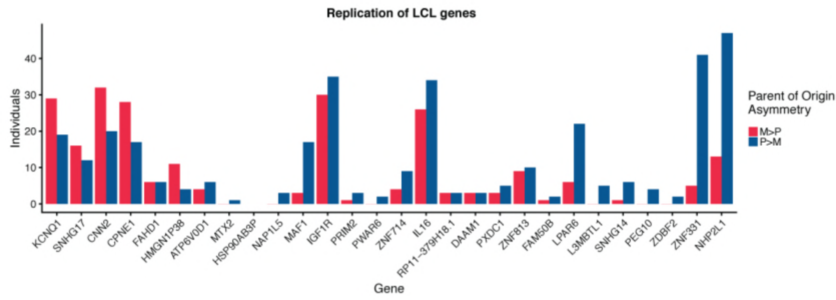

428

429 **Figure 2.** Histogram showing the number of individuals with more maternal expression (M>P)
430 or more paternal expression (P>M) for the 28 genes showing parent of origin asymmetry in (**A**)
431 LCLs and (**B**) PBLs. Genes are ordered by the magnitude of the difference in the number of
432 individuals with more maternal expression than paternal expression in LCLs.

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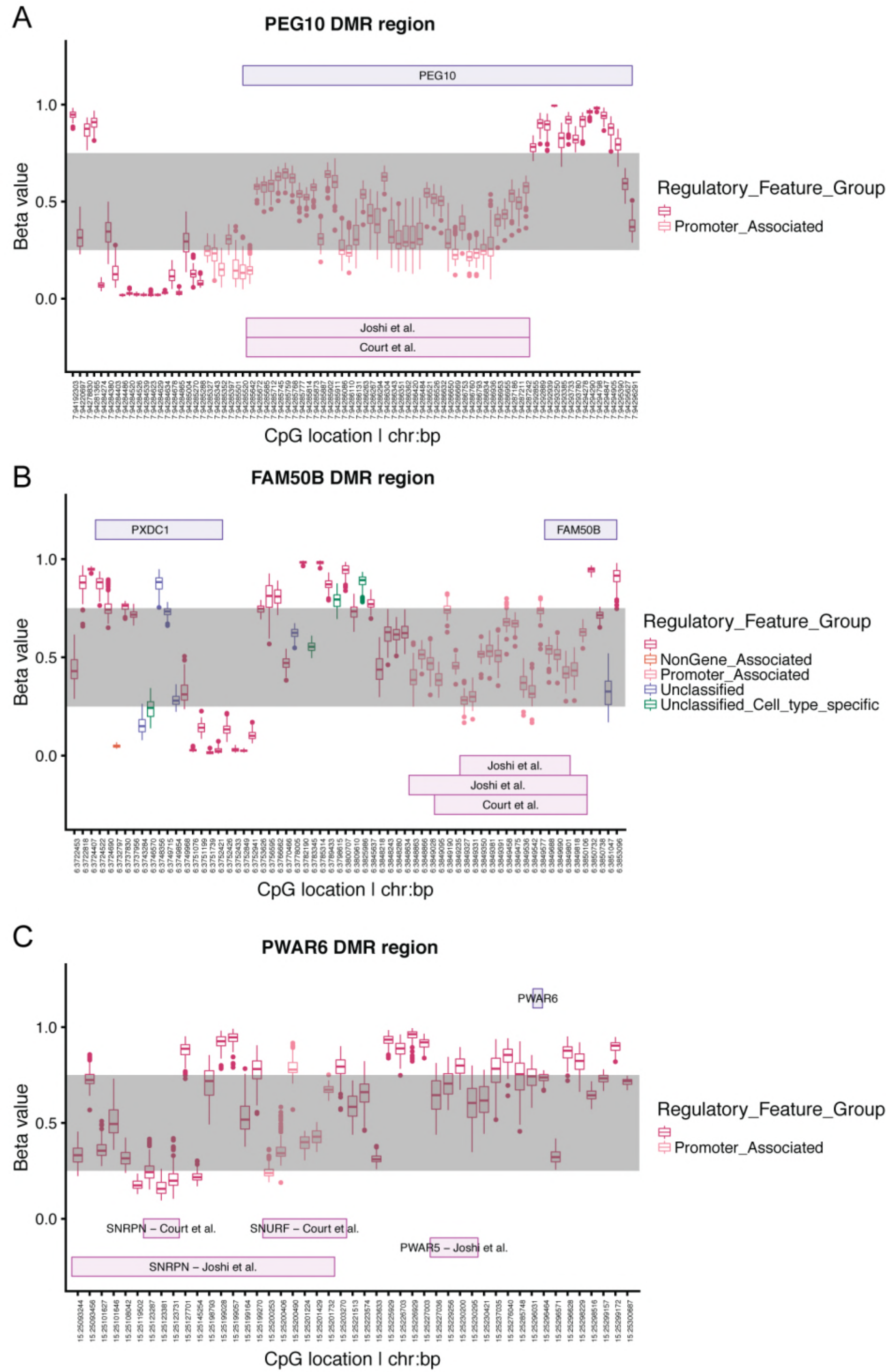

433

434 **Figure 3.** DNA methylation levels near known and novel imprinted genes previously defined by

435 Joshi et al. and Court et al. (**A)** *PEG10*, (**B)** *PXDC1* and *FAM50B*, (**C)** *PWAR6.*

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437 # Supporting Information

438 **S1 Figure.** Plots of maternal and paternal expression for remaining genes with parent of origin
439 asymmetry.

440 **S2 Figure.** Density plot for Differentially Methylated Regions (DMRs) for imprinted genes from
441 Joshi et al and Court et al.

442 **S1 Table.** Genes expressed only on maternal or only on paternal haplotypes in LCLs.

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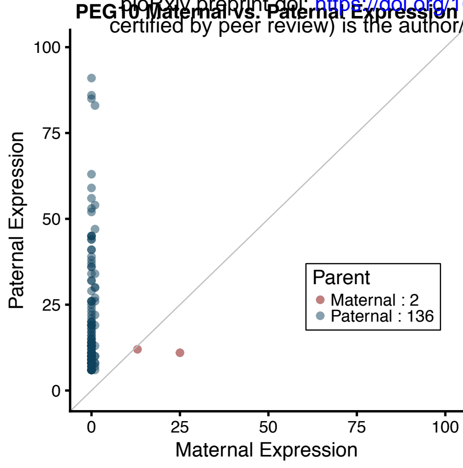

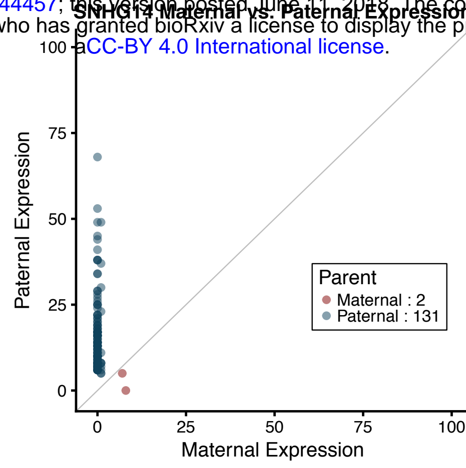

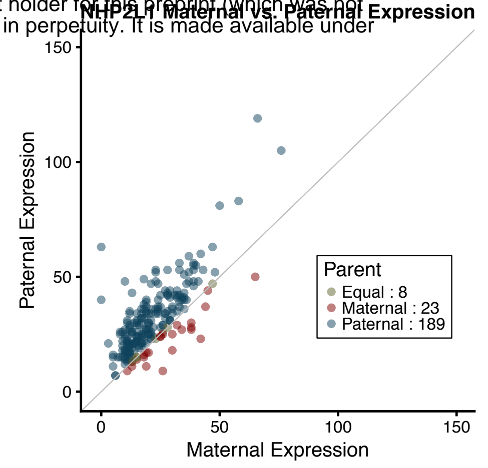

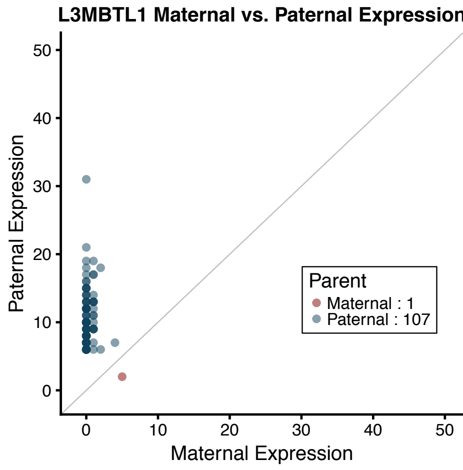

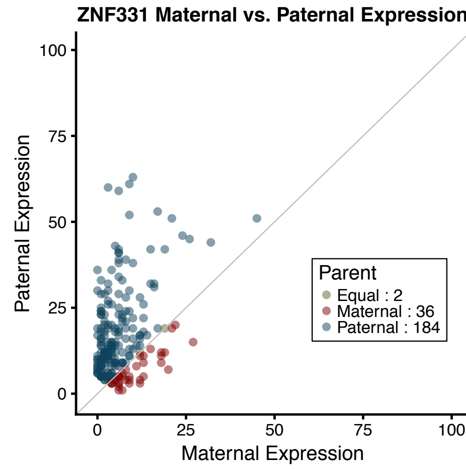

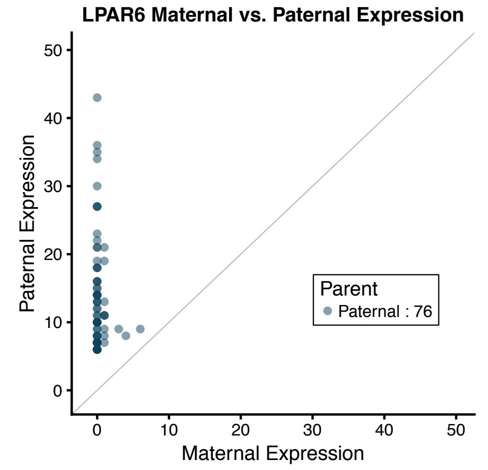

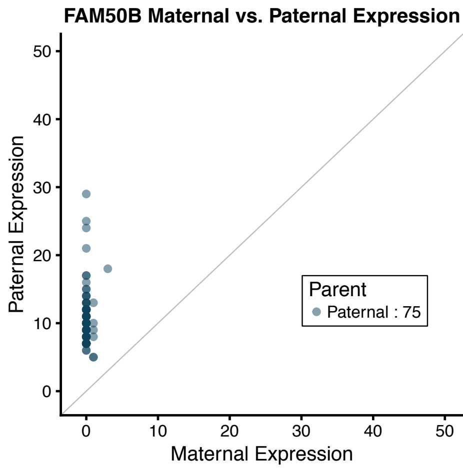

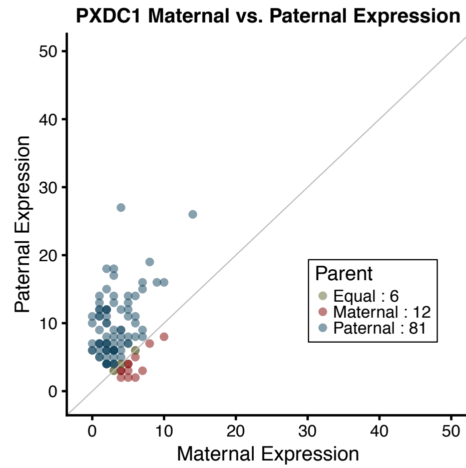

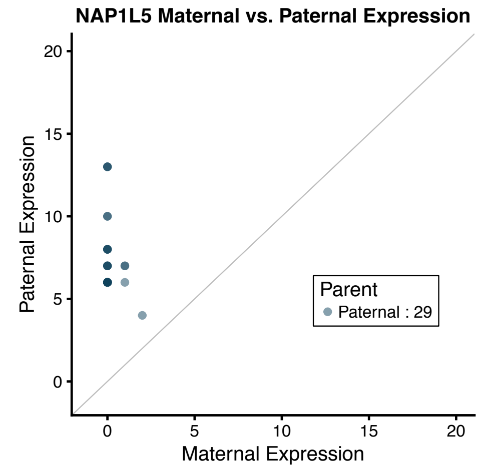

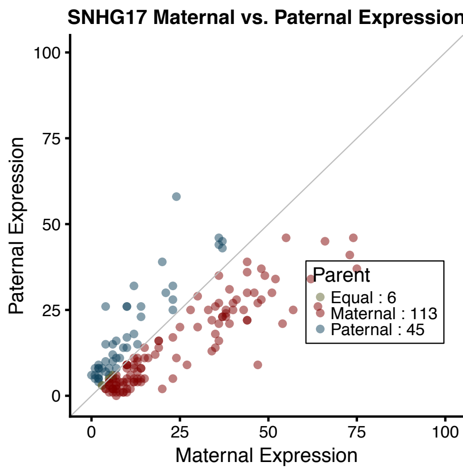

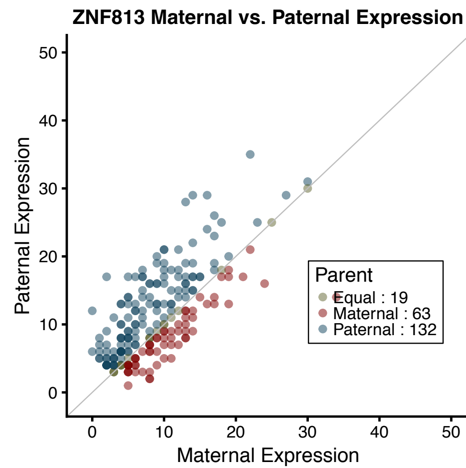

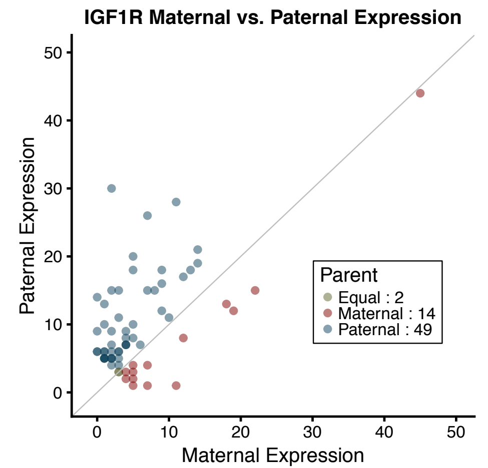

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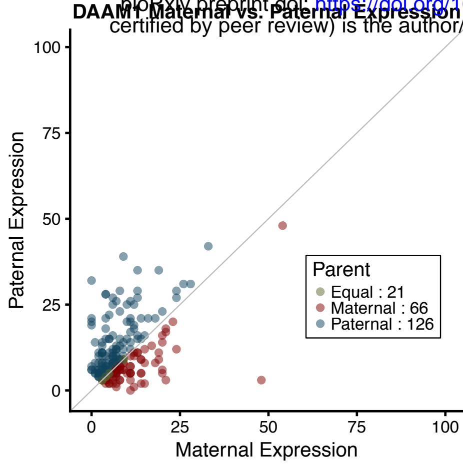

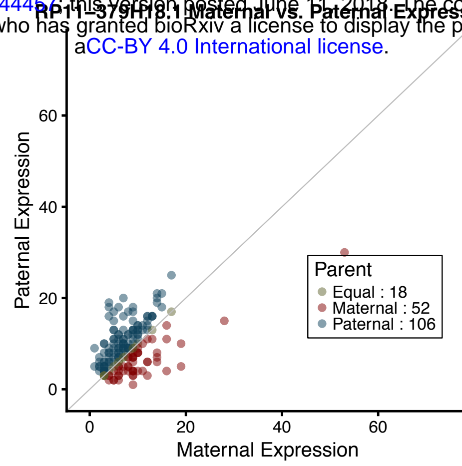

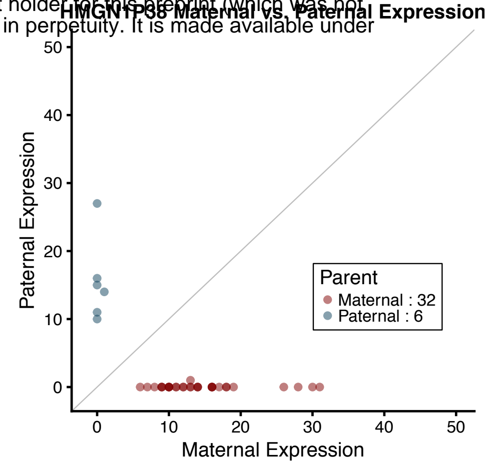

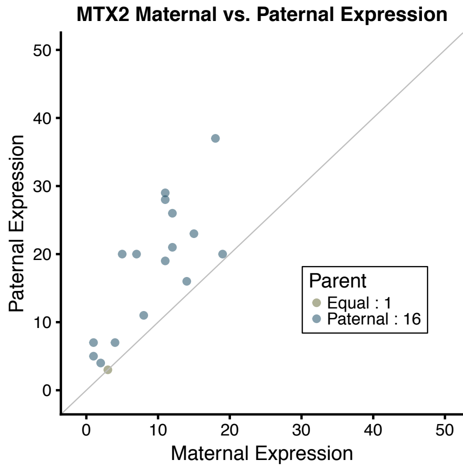

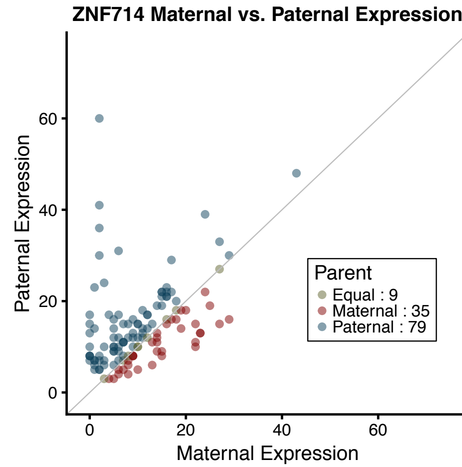

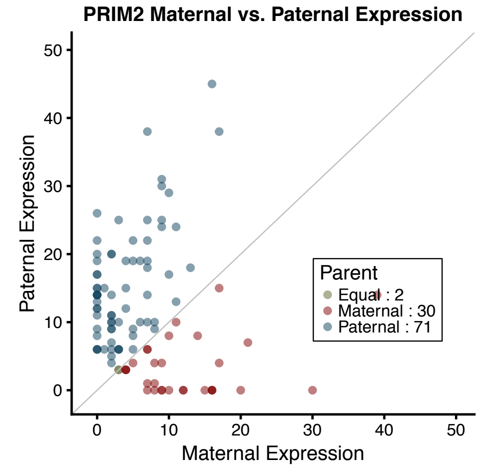

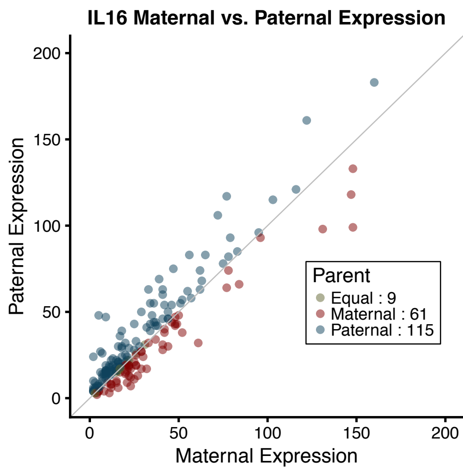

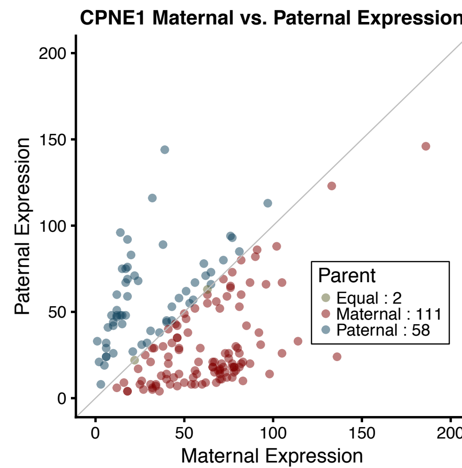

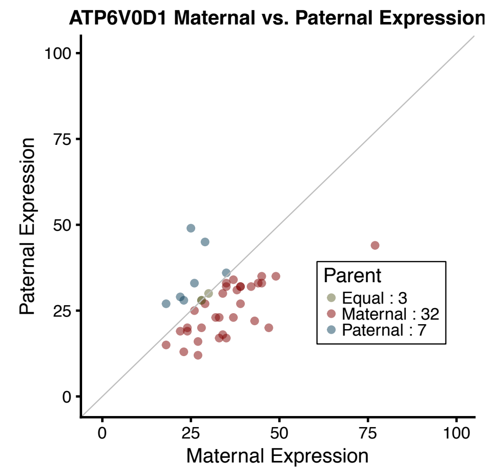

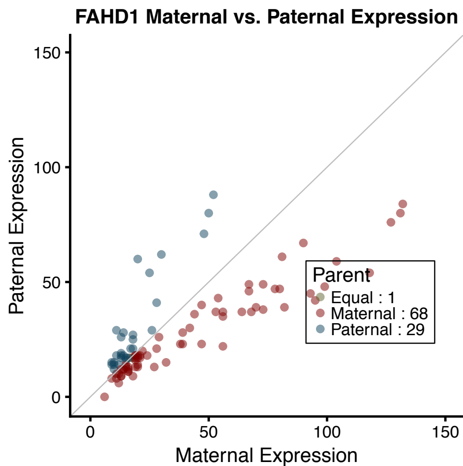

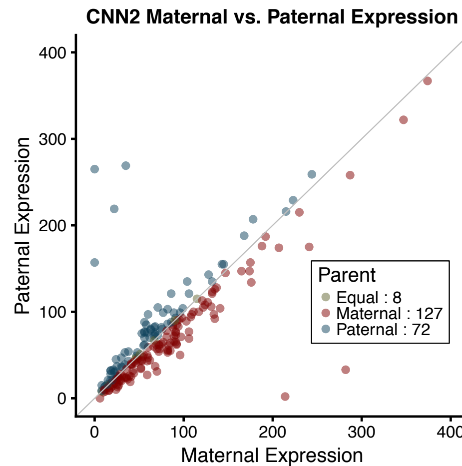

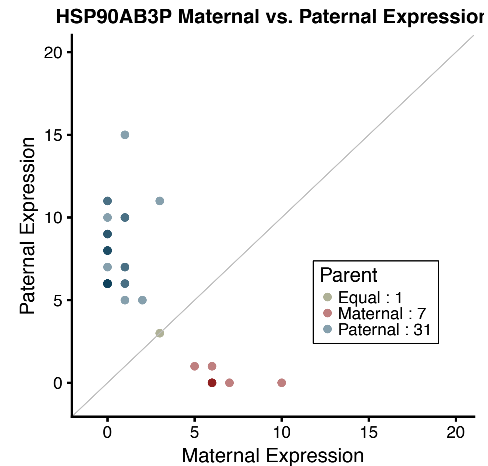

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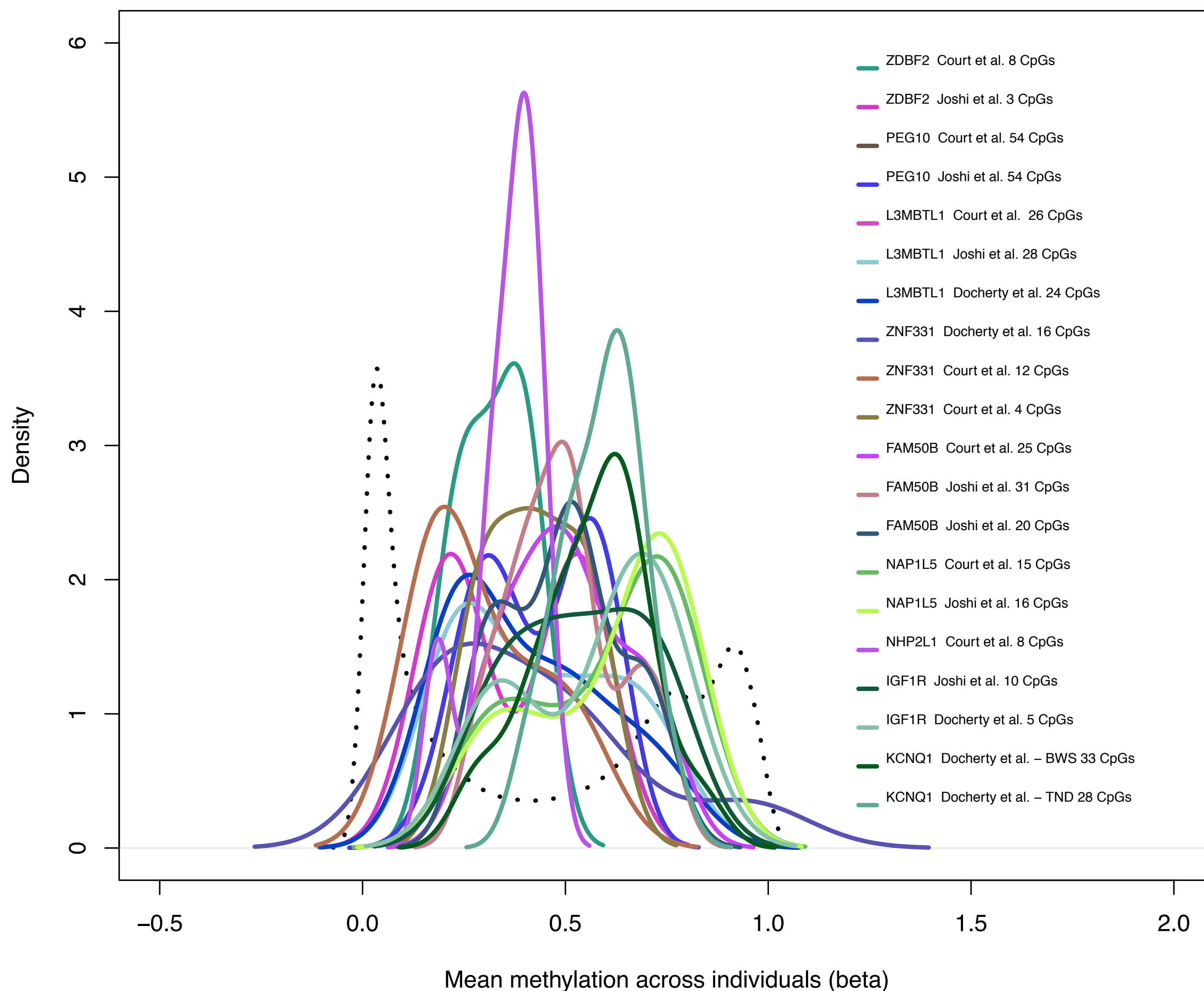

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Supplementary Table 1. Genes with only Maternal/only Paternal gene expression

Gene	Number of Individuals	Gene Name	Imprinted Status	Observed Expression Pattern in LCLs	Pattern consistent with known expression?
ENSG00000182636	22	NDN	Known	Paternal	Consistent
ENSG00000259261	11	IGHV4OR15-8		Maternal	
ENSG00000128739	9	SNRPN	Known	Paternal	Consistent
ENSG00000185044	9	RP11-435B5.4		Maternal	
ENSG00000089876	6	DHX32		Paternal	
ENSG00000262333	5	HNRNPA1P16		Paternal	
ENSG00000196378	5	ZNF34		Maternal	
ENSG00000198744	5	RP5-857K21.11		Maternal	
ENSG00000233757	5	AC092835.2		Maternal	
ENSG00000272933	5	RP11-47A8.5		Maternal	
ENSG00000154611	4	PSMA8		Paternal	
ENSG00000228109	4	MFI2-AS1		Paternal	
ENSG00000103269	4	RHBDL1		Maternal	
ENSG00000129654	4	FOXJ1		Maternal	
ENSG00000129757	3	CDKN1C	Known	Paternal	Inconsistent
ENSG00000137936	3	BCAR3		Paternal	
ENSG00000148926	3	ADM		Paternal	
ENSG00000204482	3	LST1		Paternal	
ENSG00000211669	3	IGLV3-10		Paternal	
ENSG00000223509	3	RP11-632K20.7		Paternal	
ENSG00000260442	3	RP11-22P6.3		Paternal	
ENSG00000270441	3	RP11-694I15.7		Paternal	
ENSG00000160828	3	STAG3L2		Maternal	
ENSG00000165886	3	UBTD1		Maternal	
ENSG00000181284	3	TMEM102		Maternal	
ENSG00000214269	3	LGMNP1		Maternal	
ENSG00000258561	3	RP11-72M17.1		Maternal	
ENSG00000075089	2	ACTR6		Paternal	
ENSG00000105518	2	TMEM205		Paternal	
ENSG00000115457	2	IGFBP2		Paternal	
ENSG00000137821	2	LRRC49		Paternal	
ENSG00000158517	2	NCF1		Paternal	
ENSG00000183604	2	RP11-347C12.2		Paternal	
ENSG00000211637	2	IGLV4-69		Paternal	
ENSG00000211940	2	IGHV3-9		Paternal	
ENSG00000233426	2	EIF3FP3		Paternal	
ENSG00000240041	2	IGHJ4		Paternal	
ENSG00000240731	2	RP5-890O3.9		Paternal	

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ENSG00000272145	2	NFYC-AS1	Paternal
ENSG00000025156	2	HSF2	Maternal
ENSG00000108298	2	RPL19	Maternal
ENSG00000133216	2	EPHB2	Maternal
ENSG00000133328	2	HRASLS2	Maternal
ENSG00000134864	2	GGACT	Maternal
ENSG00000158481	2	CD1C	Maternal
ENSG00000169019	2	COMMD8	Maternal
ENSG00000175701	2	LINC00116	Maternal
ENSG00000196465	2	MYL6B	Maternal
ENSG00000198155	2	ZNF876P	Maternal
ENSG00000215030	2	RPL13P12	Maternal
ENSG00000232640	2	RP1-266L20.2	Maternal
ENSG00000233493	2	TMEM238	Maternal
ENSG00000235400	2	RP4-641G12.4	Maternal
ENSG00000240652	2	RP11-832N8.1	Maternal
ENSG00000243364	2	EFNA4	Maternal
ENSG00000255135	2	RP11-111M22.3	Maternal
ENSG00000267152	2	CTD-2528L19.6	Maternal
ENSG00000033122	1	LRRC7	Paternal
ENSG00000096080	1	MRPS18A	Paternal
ENSG00000100442	1	FKBP3	Paternal
ENSG00000100632	1	ERH	Paternal
ENSG00000109083	1	IFT20	Paternal
ENSG00000111875	1	ASF1A	Paternal
ENSG00000116819	1	TFAP2E	Paternal
ENSG00000121089	1	NACA3P	Paternal
ENSG00000122218	1	COPA	Paternal
ENSG00000128011	1	LRFN1	Paternal
ENSG00000129673	1	AANAT	Paternal
ENSG00000140459	1	CYP11A1	Paternal
ENSG00000148187	1	MRRF	Paternal
ENSG00000150456	1	N6AMT2	Paternal
ENSG00000151366	1	NDUFC2	Paternal
ENSG00000154640	1	BTG3	Paternal
ENSG00000158716	1	DUSP23	Paternal
ENSG00000158806	1	NPM2	Paternal
ENSG00000163634	1	THOC7	Paternal
ENSG00000165121	1	RP11-213G2.3	Paternal
ENSG00000167286	1	CD3D	Paternal
ENSG00000173715	1	C11orf80	Paternal
ENSG00000173762	1	CD7	Paternal
ENSG00000175550	1	DRAP1	Paternal

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ENSG00000179603	1	GRM8		Paternal	
ENSG00000181038	1	METTL23		Paternal	
ENSG00000181852	1	RNF41		Paternal	
ENSG00000183506	1	PI4KAP2		Paternal	
ENSG00000197568	1	HHLA3		Paternal	
ENSG00000198356	1	ASNA1		Paternal	
ENSG00000204472	1	AIF1		Paternal	
ENSG00000211594	1	IGKJ4		Paternal	
ENSG00000211595	1	IGKJ3		Paternal	
ENSG00000211965	1	IGHV3-49		Paternal	
ENSG00000215548	1	RP11-764K9.4		Paternal	
ENSG00000225329	1	RP11-325F22.5		Paternal	
ENSG00000226121	1	AHCTF1P1		Paternal	
ENSG00000233912	1	AC026202.3		Paternal	
ENSG00000239819	1	IGKV1D-8		Paternal	
ENSG00000239830	1	RPS4XP22		Paternal	
ENSG00000243312	1	RP11-397E7.1		Paternal	
ENSG00000244055	1	AC007566.10		Paternal	
ENSG00000253998	1	IGKV2-29		Paternal	
ENSG00000257261	1	RP11-96H19.1		Paternal	
ENSG00000259699	1	HMGB1P8		Paternal	
ENSG00000260219	1	RP11-347C12.10		Paternal	
ENSG00000260655	1	CTA-250D10.23		Paternal	
ENSG00000264473	1	hsa-mir-4538		Paternal	
ENSG00000268568	1	AC007228.9		Paternal	
ENSG00000106211	1	HSPB1		Maternal	
ENSG00000118514	1	ALDH8A1		Maternal	
ENSG00000126709	1	IFI6		Maternal	
ENSG00000131773	1	KHDRBS3		Maternal	
ENSG00000135914	1	HTR2B		Maternal	
ENSG00000136104	1	RNASEH2B		Maternal	
ENSG00000136463	1	TACO1		Maternal	
ENSG00000148444	1	COMMD3		Maternal	
ENSG00000156873	1	PHKG2		Maternal	
ENSG00000163249	1	CCNYL1		Maternal	
ENSG00000164794	1	KCNV1		Maternal	
ENSG00000172586	1	CHCHD1		Maternal	
ENSG00000174871	1	CNIH2		Maternal	
ENSG00000178922	1	HYI		Maternal	
ENSG00000183426	1	NPIPA1		Maternal	
ENSG00000185885	1	IFITM1	Predicted	Maternal	Consistent
ENSG00000197279	1	ZNF165		Maternal	
ENSG00000199753	1	SNORD104		Maternal	

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ENSG00000215302	1	CTD-3092A11.1	Maternal
ENSG00000226085	1	UQCRFS1P1	Maternal
ENSG00000227053	1	RP11-395B7.4	Maternal
ENSG00000232573	1	RPL3P4	Maternal
ENSG00000237973	1	hsa-mir-6723	Maternal
ENSG00000240356	1	RPL23AP7	Maternal
ENSG00000240449	1	RP4-584D14.5	Maternal
ENSG00000253485	1	PCDHGA5	Maternal
ENSG00000254681	1	PKD1P5	Maternal
ENSG00000254887	1	CTC-378H22.1	Maternal
ENSG00000261504	1	RP11-317P15.4	Maternal
ENSG00000262691	1	CTC-277H1.7	Maternal
ENSG00000266208	1	CTD-2267D19.3	Maternal
ENSG00000268030	1	AC005253.2	Maternal
ENSG00000272468	1	RP1-86C11.7	Maternal