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CORRIGENDA

Linkage and association analysis of *CACNG3* in childhood absence epilepsy

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Due to minor changes in the clinical information, which have come to our attention, the data have been re-analysed. The corrected results for the linkage analysis are as follows: $HLOD_{max} = 3.14$ ($\alpha = 0.6$); $NPL_{max} = 2.68$ ($P < 0.003$).

Twenty-three tag SNPs were used for initial association analysis. Of these, SNPs 3, 7 and 8 showed evidence for transmission disequilibrium. These three SNPs still show significant evidence for transmission disequilibrium upon re-analysis although the values are slightly altered (Table 1).

A 'sliding window' approach was used for haplotype association analysis of SNPs 2–8 which were in linkage disequilibrium (LD). Table 2 shows the corrected data

Table 1 SNPs showing statistically significant disease association ($P \leq 0.01$) in at least one PDT test statistic in the entire resource

				Sum PDT		*Ave PDT*	
SNP	*Minor allele*	*Trans*	*Not trans*	*Z*	*P-value*	*Z*	*P-value*
3	2 (G)	291	250	2.45	0.014	2.00	0.046
7	2 (G)	261	219	2.56	0.011	2.24	0.025
8	2 (A)	279	232	2.81	0.005	2.46	0.014

Table 2 SNP-based sliding-window analysis of Block 1 showing windows which demonstrated significant ($P < 0.05$) global transmission disequilibrium in the entire resource when analysed using the PDT

SNP							*Frequency in parents (%)*	*Transmitted*	*Not transmitted*	*Sum PDT*		*Global*		*AVE PDT*		*Global*	
2	*3*	*4*	*5*	*6*	*7*	*8*				$Z_{(1d.f.)}$	*P-value*	$\chi^2_{(d.f.)}$	*P-value*	$Z_{(1d.f.)}$	*P-value*	$\chi^2_{(d.f.)}$	*P-value*
2	2						31.5	202	157	2.34	0.020	$9.49_{(3)}$	0.024	2.69	0.007	$9.39_{(3)}$	0.025
2	2	1					30.2	180	140	2.16	0.031	$16.51_{(7)}$	0.021	2.50	0.013	$14.59_{(7)}$	0.042
	2	1					44.1	255	214	2.51	0.012	$9.39_{(3)}$	0.025	2.27	0.023	$7.13_{(3)}$	0.068
	2	1	1				40.0	236	192	2.68	0.007	$12.66_{(6)}$	0.049	2.46	0.014	$9.74_{(6)}$	0.136
		1	1				48.4	270	228	2.53	0.011	$9.93_{(3)}$	0.019	2.22	0.026	$7.24_{(3)}$	0.065
		1	1	1			42.2	215	170	2.86	0.004	$14.24_{(6)}$	0.027	2.78	0.006	$13.58_{(6)}$	0.035
			1	1	2		36.8	203	155	3.07	0.002	$20.95_{(7)}$	0.004	3.02	0.003	$20.79_{(7)}$	0.004
			1	1	2	2	36.3	199	148	3.29	0.001	$27.69_{(12)}$	0.006	3.33	0.001	$27.62_{(12)}$	0.006
				1	2		40.2	218	171	3.02	0.003	$15.07_{(3)}$	0.002	2.95	0.003	$14.86_{(3)}$	0.002
				1	2	2	39.4	215	159	3.74	0.000	$27.77_{(6)}$	0.000	3.49	0.001	$25.24_{(6)}$	0.000
					2	2	45.8	270	218	3.06	0.002	$16.16_{(3)}$	0.001	2.95	0.003	$14.88_{(3)}$	0.002

Only haplotypes showing significant ($P < 0.05$) overtransmission are shown.

for this analysis. As in the original analysis, no single complete haplotype within the LD block was sufficiently common to allow demonstration of disease association on the global level. However, using the sliding window approach, associated haplotypes were identified composed of combinations of SNPs 2–8. The individual haplotypes which are overtransmitted within each window together form a larger haplotype composed of the alleles 2211122.

While subtle differences have been found in this re-analysis, this was not found to alter the conclusions drawn previously.

Unexpected genetic heterogeneity in a large consanguineous Brazilian pedigree presenting deafness

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Since the publication of the above paper, the authors have identified three typographical errors regarding Table 1. The amended table is shown below.

Table 1 Summary description of the genotypic data

Genotypic data	*Number of individuals*	*Pedigree position*
MYO15A mutations in both alleles	20	
c.10573delA homozygotes	15	V:8, V:12, V:18, V:22, V:23, V:24, V:25, V:27,V:34, VI:2, VI:3, VI:4, VI:8, VI:9 and VI:11
c.10573delA/c.9957_9960delTGAC compound heterozygotes	5	V:1, V:2, V:3, V:4 and VII:2
Unsolved cases	6	
One *MYO15A* mutation detected	1	V:17
No *MYO15A* mutations	5	VI:17, VI:19, VII:4, VII:3 and VIII:1
Total	26	