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ERRATUM

Erratum to: Further evidence for the association between a polymorphism in the promoter region of SLC6A3/*DAT1* and ADHD: findings from a sample of adults

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Unfortunately, we recently identified a batch effect in PCR readings in which reverse coding for *CC* and *TT* genotypes of *DAT1* rs2652511 polymorphism was detected. The identification of the *CT* genotype remained correct. Since the frequency of heterozygotes did not change, the genotype frequencies were in Hardy–Weinberg equilibrium ($P > 0.05$) in both the original and corrected versions of the article. Importantly, we emphasize that this coding error did not affect the genotyping data of *DAT1* VNTR polymorphisms, including the association between the 6-repeat allele of the Int8 VNTR polymorphism and higher inattention scores in patients with ADHD.

We reran our statistical analyses with the corrected genotypes of *DAT1* rs2652511 polymorphism. (1) *Logistic regression for case–control analysis*: in contrast to the original paper, the authors found no evidence to support the association between the rs2652511 *C*-allele and ADHD (Table 2 in the original paper). The correct sentence in the corresponding text in *Results* section of the original paper would be "We found **no** significant association between the rs2652511 and ADHD". An updated version of Table 2 is given. (2) *Analysis of covariance (ANCOVA) for SNAP-IV scores*: once more, our findings regarding the rs2652511 polymorphism remained non-significant (Table 3 in the original paper). Thus, interpretations and conclusions related to the ADHD dimensions remain unaltered. The corrected *P* values are as follows: hyperactivity, $P = 0.643$; inattention, $P = 0.482$; total sum of ADHD symptoms, $P = 0.915$; and ODD, $P = 0.959$.

Although this error unfortunately resulted in incorrect assignment of statistical significance to the rs2652511 polymorphism in adult ADHD, our results still support one of the major interpretations and conclusions of this research, particularly regarding the association between the 6-repeat allele of the Int8 VNTR polymorphism and higher scores of inattention in adult patients with ADHD. The authors apologize for any inconvenience that our errors may have caused.

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Table 2 Genotype analyses of the *DAT1* polymorphisms 3′-UTR VNTR, Int8 VNTR, and −839 C>T (rs2652511) and haplotype analysis for VNTR polymorphisms on susceptibility to ADHD

Genotypes[a]	Frequency (%)		Wald *P* value[b]	OR (95 % CI)
DAT1 3′-UTR VNTR	ADHD ($n = 476$)	Controls ($n = 587$)		
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.	.	.	.	.
.	.	.	.	.
DAT1 Int8 VNTR	($n = 497$)	($n = 596$)		
.	.	.		.
.	.	.	.	.
.	.	.	.	.
DAT1 −839 C>T	($n = 501$)	($n = 569$)		
TT	100 (20.0)	**98 (17.2)**		1
TC	241 (48.1)	302 (53.1)	**0.240**	**0.81 (0.58–1.14)**
CC	160 (31.9)	**169 (29.7)**	**0.957**	**0.99 (0.68–1.42)**

[a] Only the most common genotypes were taken into account in the tests of association with ADHD, and for this reason, sample sizes are smaller than the original 522 adult patients and 628 controls. *DAT1* VNTR: the rate of genotypes with rare alleles did not differ between ADHD patients and controls (Fisher's exact test): 3′-UTR VNTR (2, 3, 6, 7, 8, 11, 12, 13, and 14 repeats) −0.038 and 0.040, respectively ($P = 0.878$); Int8 VNTR: (4, 7, 13, and 14 repeats) −0.010 and 0.024, respectively ($P = 0.074$). Considering the rs2652511 SNP, 4 % of patients and 9 % of controls had missing data due to genotype failure

[b] Adjusted by age and gender