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Corrigendum: Valenced action/inhibition learning in humans is modulated by a genetic variant linked to dopamine D2 receptor expression

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A Corrigendum on

Valenced action/inhibition learning in humans is modulated by a genetic variant linked to dopamine D2 receptor expression

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We observed some errors that occurred during the genotyping of DARPP-32 rs907094. Naming of CC and TT homozygotes was swapped, and, furthermore, six genotypes were wrongly identified (three people changed from CT to CC, two people changed from CT to TT, and one person changed from TT to CT). All statistics that included DARPP-32 rs907094 genotype were recomputed. We have corrected the text in the corresponding text passages of the manuscript accordingly (last paragraph of the Results Section and **Table 2**). Importantly, these corrections did not affect our main findings, the effects attributable to the DRD2 TaqIA polymorphism.

Find below the last paragraph of the Results Section and **Table 2** with the corrected statistics including DARPP-32 rs907094 genotype.

Corrected version of the last paragraph of the Results Section

Because the TaqIA polymorphism is located downstream of the DRD2 gene, the observed genotype effects might putatively result from linkage disequilibrium with other DRD2 polymorphisms, including the C957T. We indeed observed an imbalanced distribution of the C957T polymorphism (rs6277) among TaqIA A1 carriers vs. A2 homozygotes numerically in the first cohort ($\chi^2 = 4.04$, $p = 0.132$) and significantly in the second cohort ($\chi^2 = 25.49$, $p < 0.001$). Moreover, the DARPP-32 polymorphism (rs907094) was unequally distributed in the second cohort only ($\chi^2 = 7.62$, $p = 0.022$). In order to rule out confounding effects, we included the polymorphisms as covariates in an additional ANCOVA. The same was done for COMT Val108/158Met (rs4680), because the cohorts

Corrected version of Table 2.

TABLE 2 | Demographic data.

	A1+	A1−	
COHORT 1			
Women/Men ($n = 87$)	17/20	26/24	$\chi^2 = 0.31$, $p = 0.577$
Mean age ($n = 87$)	24.9 ± 3.6	24.3 ± 2.6	$t_{(85)} = 0.83$, $p = 0.410$
Smokers/Nonsmokers ($n = 87$)	15/22	14/36	$\chi^2 = 1.51$, $p = 0.220$
COMT mm/vm/vv ($n = 87$)	13/14/10	18/15/17	$\chi^2 = 0.73$, $p = 0.694$
DAT1-VNTR 9+/9− ($n = 85$)	11/25	15/34	$\chi^2 < 0.01$, $p = 0.996$
C957T CC/CT/TT ($n = 87$)	11/19/7	8/24/18	$\chi^2 = 4.04$, $p = 0.132$
DARPP-32 CC/CT/TT ($n = 87$)	4/13/20	3/18/29	$\chi^2 = 0.68$, $p = 0.714$
COHORT 2			
Women/Men ($n = 95$)	13/21	35/26	$\chi^2 = 3.20$, $p = 0.074$
Mean age ($n = 95$)	25.2 ± 3.3	24.2 ± 2.4	$t_{(93)} = 1.58$, $p = 0.121$
Smokers/Nonsmokers ($n = 95$)	5/29	14/47	$\chi^2 = 0.93$, $p = 0.335$
COMT mm/vm/vv ($n = 95$)	11/14/9	19/27/15	$\chi^2 = 0.09$, $p = 0.957$
DAT1-VNTR 9+/9− ($n = 93$)	17/17	32/27	$\chi^2 = 0.16$, $p = 0.693$
C957T CC/CT/TT ($n = 95$)	15/17/2	3/37/21	$\chi^2 = 25.49$, $p < 0.001$
DARPP-32 CC/CT/TT ($n = 95$)	3/15/16	0/20/41	$\chi^2 = 7.62$, $p = 0.022$

Gender distribution, age (means ± standard deviations), number of smokers and nonsmokers. Allelic distributions for following polymorphisms: COMT Val108/158Met (mm, met homozygotes; vm, val/met heterozygotes; mm, met homozygotes), DAT1-VNTR (9+: carriers of the 9-repeat allele 9/9 and 9/10; 9−: 10-repeat homozygous subjects 10/10), C957T (CC/CT/TT carriers), and DARPP-32 (CC/CT/TT carriers). A1+, carriers of the A1 allele; A1−, A2 homozygotes.

were stratified with respect to that polymorphism. Importantly, the fourfold action by valence by time by genotype interaction for the TaqIA polymorphism remained significant [cohort 1: $F_{(1, 82)} = 4.67$, $p = 0.034$, cohort 2: $F_{(1, 90)} = 4.65$, $p = 0.034$], while there was no effect for C957T (cohort 1: $p = 0.484$, cohort 2: $p = 0.832$), DARPP-32 (cohort 1: $p = 0.610$, cohort 2: $p = 0.235$), or COMT Val108/158Met polymorphism (cohort 1: $p = 0.149$, cohort 2: $p = 0.842$).

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