Rare structural variants found in attention-deficit hyperactivity disorder are preferentially associated with neurodevelopmental genes

J Elia, X Gai, HM Xie, JC Perin, E Geiger, JT Glessner, M D'arcy, R deBerardinis, E Frackelton, C Kim, F Lantieri, BM Muganga, L Wang, T Takeda, EF Rappaport, SFA Grant, W Berrettini, M Devoto, TH Shaikh, H Hakonarson and PS White

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Correction to: *Molecular Psychiatry* (2010) **15**: 637–646; published online 23 June 2009; doi:10.1038/mp.2009.57

After publication of the article online, the authors wished to add the following statement to the Acknowledgement section:

We credit artist Colleen Gaynor for the artwork associated with this manuscript that is displayed on the cover of this issue.

The full Acknowledgement appears below:

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Targeted disruption of serine racemase affects glutamatergic neurotransmission and behavior

AC Basu, GE Tsai, C-L Ma, JT Ehmsen, AK Mustafa, L Han, ZI Jiang, MA Benneyworth, MP Froimowitz, N Lange, SH Snyder, R Bergeron and JT Coyle

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After the above article was published, the authors noted that the terminology used in the above article may create confusion for some readers. The substance of the clarification is as follows:

Throughout the article the term 'exon 1' was used to refer to the first coding exon of the serine racemase gene. It should be noted that the murine serine racemase gene includes two non-coding exons upstream of that which we have deleted.