CORRIGENDA

A genome-wide survey and functional brain imaging study identify *CTNNB1* as a memory-related gene

A Papassotiropoulos, E Stefanova, C Vogler, L Gschwind, S Ackermann, K Spalek, B Rasch, A Heck, A Aerni, E Hanser, P Demougin, K-D Huynh, R Luechinger, M Klarhöfer, I Novakovic, V Kostic, P Boesiger, K Scheffler and DJ-F de Quervain

Molecular Psychiatry (2013) **18**, 264; doi:10.1038/mp.2012.36

Correction to: *Molecular Psychiatry* advance online publication 22 November 2011; doi: 10.1038/mp.2011.148

Following the online publication of this article, the authors noted that the work by Jabbour L, Welter JF, Kollar J and Hering TM

(Sequence, gene structure, and expression pattern of CTNNB1, a minor-class intron-containing gene—evidence for a role in apoptosis. *Genomics*. 2003;81(3):292–303), which reports on the initial discovery, description and characterization of CTNNB1, was not cited in the article. The authors apologize for this omission.

Disorder-specific functional abnormalities during sustained attention in youth with Attention Deficit Hyperactivity Disorder (ADHD) and with Autism

A Christakou, CM Murphy, K Chantiluke, AI Cubillo, AB Smith, V Giampietro, E Daly, C Ecker, D Robertson, MRC AIMS consortium, DG Murphy and K Rubia

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Following the online publication of this article, the authors identified an error: only 60% of patients with ADHD ($N = 12$) were

medication-naïve, while the other 40% ($N = 8$) were free of psychostimulant medication (Methylphenidate: 5 Concerta; 3 Equasym) for 36 h, more than 9 times the half-life of the drug.

Genome-wide association study meta-analysis of European and Asian-ancestry samples identifies three novel loci associated with bipolar disorder

DT Chen, X Jiang, N Akula, YY Shugart, JR Wendland, CJM Steele, L Kassem, J-H Park, N Chatterjee, S Jamain, A Cheng, M Leboyer, P Muglia, TG Schulze, S Cichon, MM Nöthen, M Rietschel, BiGS and FJ McMahon

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Following the online publication of this article, the authors noted the following co-authors were not included:

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This study makes use of data generated by the Wellcome Trust Case-Control Consortium. A full list of the investigators who contributed to the generation of the data is available from www.wtccc.org.uk. Funding for the project was provided by the Wellcome Trust under award 076113 and 085475. We thank the Wellcome Trust Case Control Consortium for making data and results available for further analysis.

Data and biomaterials in the NIMH bipolar disorder sample were collected in four projects that participated in the NIMH Bipolar Disorder Genetics Initiative. From 1991 to 1998, the Principal Investigators and Co-Investigators were: Indiana University, Indianapolis, IN, U01 MH46282, John Nurnberger, MD, PhD, Marvin Miller, MD, Howard J Edenberg, PhD and Elizabeth Bowman, MD; Washington University, St Louis, MO, U01 MH46280, Theodore Reich, MD, Allison Goate, PhD, and John Rice, PhD; Johns Hopkins University, Baltimore, MD U01 MH46274, J Raymond DePaulo, Jr, MD, Sylvia Simpson, MD, MPH, and O Colin Stine, PhD; NIMH Intramural Research Program, Clinical Neurogenetics Branch, Bethesda, MD, Elliot Gershon, MD, Diane Kazuba, BA, and Elizabeth Maxwell, MSW. Data and biomaterials were also collected as part of ten projects that participated in the NIMH Bipolar.

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Access Committee. Informed consent was obtained from all subjects.

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CYP2D6 ultrarapid metabolism and early dropout from fluoxetine or amitriptyline monotherapy treatment in major depressive patients

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Following the online publication of this article, the authors noted an error in the table: below 'yes' (with the number of individuals

who discontinued from ADP treatment between parentheses), the word 'no' should appear instead of 'number'.

De novo induction of amyloid- β deposition *in vivo*

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The authors would like to correct a labeling mistake in Figure 4 of the article. The top two panels in the left (developed with anti-A β antibody) correspond to an animal killed at 450 days post inoculation and not 585 days as stated in the labeling. Indeed, the panel shown at $\times 200$ magnification corresponds to a new picture taken with higher resolution of the same section shown in

Figure 3a (center panel). The other two top panels in the right (stained with thioflavin S and anti-GFAP antibody, respectively) are correctly labeled as coming from an animal killed at 585 days post inoculation. This mistake does not affect in any way the results or conclusions of the experiment, as the purpose of Figure 4 was to show a morphological and staining comparison of representative deposits observed in HuAPPwt transgenic mice after inoculation with Alzheimer's brain homogenate and those developed spontaneously in a mice expressing human mutant amyloid precursor protein (Tg2576).