Molecular Psychiatry (2016) 21, 1153–1156

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ERRATA

Common alleles contribute to schizophrenia in CNV carriers

KE Tansey, E Rees, DE Linden, S Ripke, KD Chambert, JL Moran, SA McCarroll, P Holmans, G Kirov, J Walters, MJ Owen and MC O'Donovan

Molecular Psychiatry (2016) **21,** 1153; doi:10.1038/mp.2015.170; published online 8 December 2015

Correction to: *Molecular Psychiatry* (2015); advance online publication 22 September 2015; doi:10.1038/mp.2015.143

The first author in Reference 33 was listed incorrectly in the reference list and in the last paragraph of the Discussion section. The author's last name should have been listed as Gottesman. The correct reference appears below. The publisher regrets the error.

33 Gottesman II, Gould TD. The endophenotype concept in psychiatry: etymology and strategic intentions. *Am J Psychiatry* 2003; **160**: 636–645.

Arc expression identifies the lateral amygdala fear memory trace

LA Gouty-Colomer, B Hosseini, IM Marcelo, J Schreiber, DE Slump, S Yamaguchi, AR Houweling, D Jaarsma, Y Elgersma and SA Kushner

Molecular Psychiatry (2016) **21,** 1153; doi:10.1038/mp.2016.91; published online 24 May 2016

Correction to: *Molecular Psychiatry* (2016); **21**, 364–375; doi:10.1038/mp.2015.18

Following publication of the above article, the authors noticed that the row and column headings of Supplementary Table 1 were absent from the online version. The correct version of the table accompanies this erratum online. The publisher regrets the error.

Supplementary Information accompanies the paper on the *Molecular Psychiatry* website (http://www.nature.com/mp)

Molecular systems evaluation of oligomerogenic APP^{E693Q} and fibrillogenic $APP^{KM670/671NL}/PSEN1^{\Delta exon9}$ mouse models identifies shared features with human Alzheimer's brain molecular pathology

B Readhead, J-V Haure-Mirande, B Zhang, V Haroutunian, S Gandy, EE Schadt, JT Dudley and ME Ehrlich

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Correction to: *Molecular Psychiatry* advance online publication, 10 November 2015; doi:10.1038/mp.2015.167

The published version of Figure 3 is missing some of the graphics. The correct version of the figure appears below. The publisher regrets the error.

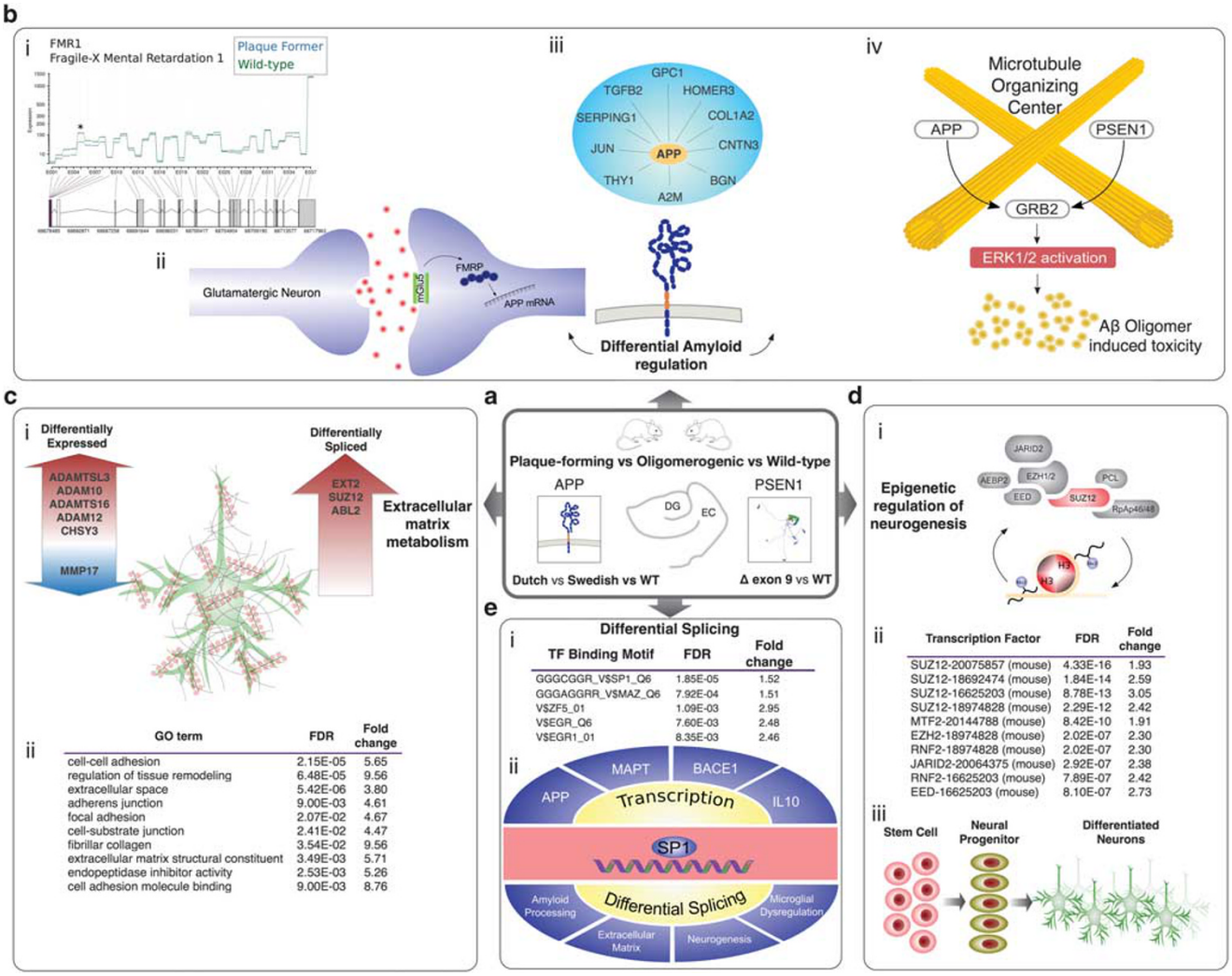

Figure 3. Multiregion transcriptome comparisons between fibrillogenic, oligomerogenic and wild-type mice implicates amyloid/Aβ processing, extracellular matrix (ECM) regulation and neurogenesis (**a, b–**i) Fragile X Mental Retardation 1 (FMR1) gene is differentially spliced in fibrillogenic $APP^{KM670/671NL}/PSEN1^{\Delta exon9}$ mice vs oligomerogenic APP^{E693Q} dentate gyrus (also vs wild type), as well as multiple brain regions in LOAD and (**b**-ii) is a known regulator of APP, binding to mRNA in the post-synaptic neuron in an mGluR5 stimulation-dependent manner. (**b**-iii) DE genes in both comparisons with wild type (see Figure 2), are enriched for known protein interactors of APP. APP interactors that are DE in the fibrillogenic $APP^{KM670/671NL}/PSEN1^{\Delta exon9}$DG vs wild type are shown. (**b**-iv) Adaptor protein GRB2 is differentially spliced in fibrillogenic $APP^{KM670/671NL}/PSEN1^{\Delta exon9}$ mice vs oligomerogenic APP^{E693Q} dentate gyrus, and interacts with APP and PSEN1, localized to the centrosomes, resulting in ERK1/2 activation, and potentiation of oligomer-induced toxicity. (**c**) ECM regulation was a recurring theme of the pathway analysis following differential gene and exon expression analysis. (**c**-i) Known ECM regulators that are differentially expressed in fibrillogenic $APP^{KM670/671NL}/PSEN1^{\Delta exon9}$ vs wild-type mice (dentate gyrus) suggest mechanisms of perturbation and compensation. (**c**-ii) Gene Ontology (GO) enrichment analysis of the 354 genes that are differentially expressed in fibrillogenic $APP^{KM670/671NL}/PSEN1^{\Delta exon9}$ vs wild-type mice (dentate gyrus) demonstrate that the trend toward ECM disruption is particularly strong in this comparison. (**d**) Pathway enrichment analysis of the differentially expressed genes in fibrillogenic $APP^{KM670/671NL}/PSEN1^{\Delta exon9}$ vs wild-type mice (dentate gyrus) indicates perturbation of stem cell, neural progenitor cell and neurogenesis pathways. (**d**-i) SUZ12 is a key member of the polycomb repressive complex 2 (PRC2), and is differentially spliced in fibrillogenic $APP^{KM670/671NL}/PSEN1^{\Delta exon9}$ mice vs oligomerogenic APP^{E693Q} dentate gyrus (and also vs wild type). (**d**-ii) A functional role for SUZ12 is strongly supported by enrichment analysis of ChipSeq-based transcription factor gene targets, with the 354 differentially expressed genes in fibrillogenic $APP^{KM670/671NL}/PSEN1^{\Delta exon9}$ vs wild-type mice (dentate gyrus). (**d**-iii) SUZ12 function within the PRC2 is associated with regulation of neurogenic differentiation of stem cells via histone H3K27 and H3K9 methylation. (**e**-i) Zinc finger gene SP1 was identified as the transcription factor most strongly enriched for DEX genes ($APP^{KM670/671NL}/PSEN1^{\Delta exon9}$ vs wild-type comparison). (**e**-ii) SP1 is a transcriptional regulator of multiple AD-associated genes, and forms a potential link between these molecular nodes and the main DEX themes we have discussed, including perturbations in neurogenesis, amyloid processing and ECM regulation.