

Corrigenda

Epigenetic Mechanisms for the Early Environmental Regulation of Hippocampal Glucocorticoid Receptor Gene Expression in Rodents and Humans

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Correction to: *Neuropsychopharmacology Reviews* (2012) 38, 111–123; doi:10.1038/npp.2012.149; published online 12 September 2012.

Figure 1 Maternal licking/grooming (LG) induces glucocorticoid receptor (GR) expression in the pup hippocampus by increasing association between NGFI-A and the GR₁₇ promoter. Increased frequency of maternal LG activates the 5-HT₇ receptor; inducing NGFI-A through a cAMP-PKA dependent pathway *in vivo*. In hippocampal cell culture 5-HT binds the 5-HT₇ receptor and increases GR expression through the same cAMP-PKA pathway. ACTH, adrenocorticotropin releasing hormone; CRF, corticotropin releasing factor; cAMP, cyclic adenosine 3,5 mono-phosphate; NGFI-A, nerve growth factor-inducible A; PKA, protein kinase A; 5-HT, serotonin.

Figure 2 Glucocorticoid receptor (GR) gene organization. Schema describing the organization of the rat and human glucocorticoid receptor gene, including the 9 exon regions. Exons 2–9 code for the glucocorticoid receptor protein. Exon 1 is comprised of multiple, tissue-specific promoter regions (rat is adapted from McCormick *et al* (2000) and human from Turner and Muller (2005)). The rat exon 1₇ shares ~70% sequence homology with the human exon 1_F, and both are highly expressed in hippocampus.

In this article, there are errors in the legends of Figures 1, 2, and 3. Below are the correct legends.

Figure 3 DNA 5-hydroxymethylcytosine (5-hmC) and 5-methylcytosine (5-mC) analyses of the GR exon 1₇ promoter in hippocampal samples from offspring of high and low LG dams. 5-hmC (left panel) and 5-mC (right panel) levels are expressed as a percentage (mean ± SEM) of input DNA (5-hmC *n* = 3–4 per group; 5-mC *n* = 5–6 per group). Controls show negligible signal (ie, 0–3%; data not shown) assayed using a commercially available kit (5-hmC, Diagenode Cat. No. AF-104-0016; 5-mC: Epigentek, Cat. No. p-1015-24).

Yohimbine Depresses Excitatory Transmission in BNST and Impairs Extinction of Cocaine Place Preference Through Orexin-Dependent, Norepinephrine-Independent Processes

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In this article, we inadvertently referred to a compound used in experiments in figure 3 as JNJ-10397047. However,

it should read JNJ-10397049 (1-(2,4-dibromo-phenyl)-3-((4S,5S)-2,2-dimethyl-4-phenyl-[1,3]dioxan-5-yl)-urea).