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Abstract.

The paper entitled "Regulation of DARPP-32 dephosphorylation at PKA and Cdk5-sites by NMDA and AMPA receptors", authored by Nishi, A., Bibb, J.A., Matsuyama, S., Hamada, M., Higushi, H., Nairn, A.C. and Greengard. P., and published in the Journal of Neurochemistry. [J. Neurochem. (2002) Vol. 5, pp832-841] describes the study the effects of ionotropic glutamate NMDA and AMPA receptors on DARPP-32 phosphorylation in neostriatal slices and purported to show that activation of NMDA and AMPA receptors caused the decrease of phosphorylations of threonone 34 and threonine 75 DARPP-32 mediated by Ca^{2+}-dependent activation of calcineurin and protein phosphatase-2A respectively. However, no supporting scientific results were provided. Figures 1A, 1B, 1C and 1D are duplicates of each other. With exactly the same figures depicting exactly the same immunublots, the authors somehow came up with different bar charts that supposedly quantify the extent of DARPP-32 phosphorylation levels under different conditions. Figures 3A and 3C are also duplicates of each other. It is not clear how the reviewer(s) and the Editor of the Journal of Neurochemistry could have missed the glaring evidence of Data Fabrication and and Data Falsification.

This is an Investigative Critique of the paper entitled "Regulation of DARPP-32 dephosphorylation at PKA and Cdk5-sites by NMDA and AMPA receptors", authored by Nishi, A., Bibb, J.A., Matsuyama, S., Hamada, M., Higushi, H., Nairn, A.C. and Greengard. P., and published in the Journal of Neurochemistry. [J. Neurochem. (2002) Vol. 5, pp832-841].

According to the authors of the paper, they investigated the effects of ionotropic glutamate NMDA and AMPA receptors on DARPP-32 phosphorylation in neostriatal

slices and claimed to have shown that activation of NMDA and AMPA receptors caused the decrease of phosphorylations of threonone 34 and threonine 75 DARPP-32 mediated by Ca^{2+}-dependent activation of calcineurin and protein phosphatase-2A respectively. Various data were presented including Figure 1 which showed (i) Immunoblots of Phospho-Thr34 in neostriatal slices following no treatment, treatment with AMPA, treatment with NMDA plus MK801, and treatment with MK801 only (Figure 1a), (ii) Immunoblots of Phospho-Thr34 in neostriatal slices following no treatment, treatment with AMPA, treatment with NMDA plus CNQX, and treatment with CNQX alone (Figure 1b), (iii) Immunoblots of Phospho-Thr75 in neostriatal slices following no treatment, treatment with AMPA, treatment with NMDA plus MK801, and treatment with MK801 only (Figure 1c), (ii) Immunoblots of Phospho-Thr34 in neostriatal slices following no treatment, treatment with AMPA, treatment with NMDA plus CNQX, and treatment with CNQX alone (Figure 1d).

Careful analysis of Figure 1(a), 1(b), 1(c) and 1(d) revealed that they are all the same exact duplicates of each other except that Figure (1b), 1(c) and (1(d) have been manipulated unscientifically to look differentially lighter than Figure 1(a). Figure 1(a), 1(b), 1(c) and 1(d) are clones of each other. Although, Figure 1(a), 1(b), 1(c) and 1(d) were generated from the same results and Immunoblots, somehow, the authors of the paper entitled "Regulation of DARPP-32 dephosphorylation at PKA and Cdk5-sites by NMDA and AMPA receptors" and published in the Journal of Neurochemistry. [J. Neurochem. (2002) Vol. 5, pp832-841] were able to magically come up with different statistical analyses showing that the four different figures were distinct experiments giving different results. The authors state in the results section the following: (i) "treatment of slices with NMDA (100 μM) for 5 min decreased the level of phospho-Thr34 DARPP-32 to 26.4 ± 7.9% of control" (Fig. 1a), (ii) "Pretreatment of slices with an NMDA receptor antagonist, MK801 (100 μM), for 10 min did not affect the basal level of phospho-Thr34 DARPP-32. How-ever, the effect of NMDA on DARPP-32 Thr34 phosphory-lation was abolished by MK801", (iii) Treatment of slices with AMPA (50 μM) for 5 min decreased the level of phospho-Thr34 DARPP-32 to 66.0 ± 7.6% of control (Fig. 1b), (iv) Pretreatment of slices with an AMPA receptor antagonist, CNQX

(20 μM), for 10 min increased the basal level ofphospho-Thr34 DARPP-32, possibly by antagonizing the effects of endogenously released glutamate. The effect of AMPA on DARPP-32 Thr34 phosphorylation was antagon-ized by CNQX. How with the same Immunoblot (Figure 1(a) and Figure 1(b), the authors were able to come up with different statistical analysis of 26.4 ± 7.9% of control for Figure 1(a) and 66.0 ± 7.6% of control for Fig. 1b is beyond normal scientific comprehension and in the realm of science fiction. Figure 1(c) and Figure 1(d) are also exact replicas of Figure 1(a) except that they have been unscientifically manipulated to appear lighter. Yet somehow, the authors of the paper were able to come up with statistical analysis of 22.1 ± 2.2% of control for Figure 1(c) and 47 ± 5.6% of control for Figure 1(d).

Figure 2(a), Lanes for control and NMDA and Figure 2(c), Lanes for control and KCl looked suspiciously similar. Figure 3(a) and 3(c) are exact duplicates except that Figure 3(c) has unscientifically manipulated to look lighter. Despite the fact that Figure 3(a) and Figure 3(c) are exact duplicates, they are stated to depict different treatment with NMDA (Figure 1a) and KCl (Figure 1c),

In conclusion, the paper entitled "Regulation of DARPP-32 dephosphorylation at PKA and Cdk5-sites by NMDA and AMPA receptors", authored by Nishi, A., Bibb, J.A., Matsuyama, S., Hamada, M., Higushi, H., Nairn, A.C. and Greengard. P., and published in the Journal of Neurochemistry. [J. Neurochem. (2002) Vol. 5, pp832-841] must be retracted because it contains data that was fabricated and falsified. How such blatant cheating managed to pass through the "peer review" system of the Journal of Neurochemistry is unfathomable. Is it because the paper came from a so called "Star Laboratory" and "Star Principal Scientific Researcher". Pursuant to the Office of Research Integrity of the United States Department of Health and Social Services, Scientific Misconduct is defined as Pursuant to the Office of Research Integrity (ORI) of the United States Department of Health and Human Services, Research Misconduct is defined as Data Fabrication, Data Falsification and Plagiarism in proposing, performing or reviewing research, or in reporting research results [1,2]. While Scientific Misconduct is not a criminal act per se in the United States of America, the use of the fruits of

Scientific Misconduct to defraud a person or entity, like obtaining a research grant from the NIH or DOD with the Fabricated Data and Falsified Data as Preliminary or Published Results in support of the grant application is a criminal act that can be prosecuted under the Mail and Wire Fraud Statutes, 18 U.S.C § 1341 and 18 U.S.C § 1343, and False Claim Act, 31 U.S.C. § 3729. Many Scientific Researchers have been prosecuted, found guilty, imprisoned and fined [3].

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