Role of GSK3 β in behavioral abnormalities induced by serotonin deficiency

We were interested to read the article by Beaulieu *et al.* (1), suggesting that GSK3 β haploinsufficiency counteracts depressive-like behavior in mice expressing a functional TPH2 mutant with a marked decrease in serotonin production. In the figures of the current article, it is clear that GSK3 β haploinsufficiency had no antidepressant effect in the tail suspension test in animals without the TPH2 mutant enzyme. However, Beaulieu *et al.* fail to mention the paper by O'Brien *et al.* (2), who reported that GSK3 β haploinsufficiency alone gives dramatic antidepressant-like effects in the Porsolt swim test, a closely related test for antidepressant behavior. We

also have not been able to replicate the results of Beaulieu *et al.* (see ref. 3). However, it would have seemed essential for the discussion of the current Beaulieu *et al.* article that they mention the previous report, which found that GSK3 β haploinsufficiency is quite sufficient to cause an antidepressant phenotype, in contradiction to their own findings.

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1. Beaulieu JM, *et al.* (2008) Role of GSK3 β in behavioral abnormalities induced by serotonin deficiency. *Proc Natl Acad Sci USA* 105:1333–1338.
2. O'Brien WT, *et al.* (2004) Glycogen synthase kinase-3 β haploinsufficiency mimics the behavioral and molecular effects of lithium. *J Neurosci* 24:6791–6798.
3. Bersudsky Y, *et al.* (2008) Glycogen synthase kinase-3 β heterozygote knockout mice as a model of findings in postmortem schizophrenia brain or as a model of behaviors mimicking lithium action: Negative results. *Behav Pharmacol*, in press.

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The authors declare no conflict of interest.

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