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Cover legend: A schematic illustration of Reelin expression in glutamatergic pyramidal neurons in layer II of the entorhinal cortex and their projections to the hippocampus, illustrated here by seahorses (*Hippocampus sp.*). Reelin is a neuromodulator that regulates a number of neuronal functions, including synaptic strength and plasticity, suggesting that Reelin produced in the entorhinal cortex may influence hippocampal plasticity. In humans with Alzheimer's disease as well as in transgenic mouse models of the disease, these neurons are lost and/or decrease expression of Reelin, indicating that decreased Reelin from the entorhinal cortex may contribute to hippocampal dysfunction in Alzheimer's disease. For more information, see the article by Chin et al. in this issue (pages 2727–2733). Artwork by Katherine Nagel.

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Correction: In the article “Novel Isoforms of Dlg Are Fundamental for Neuronal Development in *Drosophila*” by Carolina Mendoza, Patricio Olguín, Gabriela Lafferte, Ulrich Thomas, Susanne Ebitsch, Eckart D. Gundelfinger, Manuel Kukuljan, and Jimena Sierralta, which appeared on pages 2093–2101 of the March 15, 2003 issue, the authors described a novel dlg isoform containing an extended N-terminal domain (dlgS97). Although the authors stand by their results on the description of the gene, its localization, and

biochemical analysis, they have now found errors in their RNA interference (RNAi)-based approach to understand *dlgS97* function. It was reported that injection of a double-stranded RNA (dsRNA) directed to the novel isoform *dlgS97* correlated with strong defects in the development of the nervous system. However, the recent isolation of a *dlgS97* null mutant that lacks defects in embryonic nervous system development, as well as their developmental expression showing that *dlgS97* is not present at early embryonic stages, leads the authors to reexamine the RNAi experiments. They have found that the defects reported were attributed to unintended target effects caused by a low-complexity fragment of <50 bp at the end of the 1.2 kb RNAi construct. This conclusion is supported by new injection experiments in which the reported defects are not observed after injection of a dsRNA lacking the 50 bp fragment. In addition, injection of 21 mers contained within this 50 bp region replicated the nervous system described in the paper. In conclusion, although the authors stand by the experiments describing the gene and its expression pattern, the conclusion from the RNAi studies are incorrect.

Additional data can be found at <http://www.ceni.cl/sierralta-RNAi>

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