that our work is so exciting and urgent that we will not allow ourselves to be deterred at any cost.

NEIL SMALHEISER
Department of Pediatrics, Box 413, Joseph P. Kennedy, Jr. Mental Retardation Research Center, University of Chicago, Chicago, IL 60637

The Cellular Basis of Memory

Marcia Barinaga's Research News article (29 June, p. 1603) reporting on the recent results presented at the Cold Spring Harbor meeting on the cellular basis of memory was well written and accurate. However, we were surprised that the article contained no hint of the controversy that exists over the results of quantal analysis of hippocampal synaptic enhancement.

In a study of 33 synaptic connections obtained with a minimal stimulation technique similar to that used by Robert Malinow and Richard Tsien (*1*) as well as simultaneously recorded cell pairs, we obtained no evidence for an increase in the number of quantal components of the excitatory postsynaptic potential (EPSP). Rather, we found a significant increase in quantal size (*2*).

We see several possible sources for the apparent discrepancy between our results and those of Malinow and Tsien (*1*).

1) The increase in estimated quantal release observed by Malinow and Tsien accounts for only about half of the EPSP growth. Because their method of analysis is known to overestimate quantal release, we think it is likely that increased quantal size is actually the *predominant* effect.

2) We have shown that the method of data selection employed by Malinow and Tsien could bias the results toward increased quantal release, although this probably does not account for the entire discrepancy.

3) The conclusions of Malinow and Tsien are based on the assumption of uniformity of quantal size. For purposes of illustration, consider the extreme case in which *some* sites at which quanta are released contain *no* receptors. Addition of receptors (a postsynaptic modification) would show up in their analysis as increased quantal release.

4) Our experiments were carried out on adult tissue rather than tissue from animals in which the hippocampus is not yet mature and in which substantial synaptogenesis and structural rearrangement are known to be in progress. This difference could account for much of the discrepancy. It would hardly be surprising if the same patterns of activity that lead to an increase in the strength of responses at existing release sites in adult brains lead to both modification of existing sites and to formation of new ones in neonates.

We think a more balanced treatment of this controversial issue would have better served the scientific community.

B. L. MCNAUGHTON
T. C. FOSTER
Arizona Research Laboratories Division of Neural Systems, Memory, and Aging, University of Arizona, Tucson, AZ 85721

REFERENCES

1. R. Malinow and R. Tsien, *Nature* **346**, 177 (1990).
2. T. C. Foster and B. L. McNaughton, *Hippocampus*, in press.

Erratum: In the Perspective "Too many rodent carcinogens: Mitogenesis increases mutagenesis" by Bruce N. Ames and Lois Swirsky Gold (31 Aug., p. 970), the last paragraph on page 970 (continuing on page 971) was incorrectly printed. It should have read, "One major group of natural chemicals in the human diet are the chemicals that plants produce to defend themselves, the natural pesticides (*4*). We calculate that 99.9% (by weight) of the pesticides in our diet are natural. Few natural pesticides have been tested in at least one rodent species, and again about *half* (27/52) are rodent carcinogens. These 27 occur commonly in plant foods (*10*). The human diet contains thousands of natural pesticides, and we estimate that the average intake is about 1500 mg per person per day (*4*). This compares to a total of 0.09 mg per person per day of residues of about 100 synthetic pesticides (*4*). In addition, of the mold toxins tested at the MTD (including aflatoxin), 11 out of 16 are rodent carcinogens." Also, in paragraph 3 on page 970, "47,000 8-hydroxydeoxyguanosines per cell" should have been "90,000" per cell.