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receptor deficits could account for the cognitive impairments observed in schizophrenia or, in many cases, the lack of restitution of cognitive function in response to drugs. If this hypothesis is correct for even a subgroup of patients, then the finding that D1mediated impairment can be reversed by a selective D1 agonist has clinical implications. Although our study supports an important role of the D1 receptor in optimizing cognitive function in the nonhuman primate, its role in schizophrenia or other conditions of dopamine deficiency, such as aging and Parkinson's disease, deserves further investigation.

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References

1. Y. Okubo et al., Nature 385, 634 (1997).

CORRECTIONS AND CLARIFICATIONS

Letters: "Many modes of movement," letter by A. Carl Leopold (23 June, p. 2131). The second author of this letter, Mordecai J. Jaffe, was omitted as a result of an editorial error. He is at the Boyce Thompson Institute of Plant Research, Ithaca, NY 14853, USA.

News of the Week: "Penn report, agency heads home in on clinical research" by Eliot Marshall (2 June, p. 1558). It was incorrectly reported that a company in which the University of Pennsylvania and researcher James Wilson hold an interest (Genovo Inc. of Sharon Hill, Pennsylvania) supplied reagents for a gene therapy trial at the university. The university, not the company, provided the reagents.

Research Article: "Timing the ancestor of the HIV-1 pandemic strains" by B. Korber *et al.* (9 June, p. 1789). The Web address in reference 23 is missing a "~" character. The correct address is http://www.santafe.edu/ ~btk/science-paper/bette.html

Perspectives: "Absorbing phenomena" by S. E. and P. R. Buseck (12 May, p. 989). The source of the image on page 990 was reference 15, not reference 14 as stated in the credit.

ScienceScope: "Unconventional committee" (12 May, p. 941). Harvey Bialy's last name was misspelled.



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