dementia) provides a statistical significance ($P < 0.01$, Kendall rank test). In the group that was autopsied, there was one person with MGCs out of 18 patients with KS (5.6%), as compared with 36 people with MGCs out of 149 patients without KS (24.5%; $P = 0.06$ if a one-sided exact test is used; $P < 0.05$ if one uses the grading MGC, diffuse damage of white matter, and no signs).

Factors such as survival length in the immunodeficient stage, timing of disease events, and treatment with the neuroprotective drug Zidovudine were considered as possible confounding factors. Differences in survival and drug use were small, and these factors appear not to explain the observations. There may, however, be differences between patients with KS and those without KS (such as viral subtype and host factors) that are difficult to examine.

Thus, our results from a sample of patients from Oslo conform to the expected outcome—there is a blocking effect of KSHV-encoded chemokines where HIV binds to CCR3 on microglia that can protect a patient from the development of HIV dementia. There are, however, several open questions, and it will be a challenge to further test this interesting hypothesis with more direct methods.

Knut Liestœl
Department of Informatics, University of Oslo, N-0316 Oslo, Norway
E-mail:knut@ifi.uio.no
Anne K. Goplen
Department of Pathology, Ullevål Hospital, N-0407 Oslo, Norway
Oona Dunlop
Johan N. Bruun
Department of Infectious Diseases, Ullevål Hospital
Jan Mœhlen
Department of Pathology, Ullevål Hospital

References

1. J. Mœhlen *et al.*, *AIDS* **9**, 1165 (1995).
2. R. W. Price and B. J. Brew, *J. Infect. Dis.* **158**, 1079 (1988).

Response: Like Liestœl *et al.*, we have examined whether KS/KSHV can protect against HIV-1 dementia, as we suggested in our earlier report. We measured the probability that HIV-1 dementia and opportunistic central nervous system (CNS) disease would appear in patients who developed KS as compared with the probability for those in whom KS did not develop. These data were gathered during the follow-up of a retrospective study of a cohort of patients with AIDS (*1*).

A total of 1109 patients with an AIDS-defining illness between 1 January 1990 and 31 December 1994 who attended a specialized center of HIV medicine in London were identified from a central database. Three diagnostic groups were defined: (i) no CNS impairment ($n = 914$), (ii) HIV-1 dementia ($n = 88$), and (iii) opportunistic brain disease ($n = 107$), including progressive multifocal leukoencephalopathy (PML), cerebral toxoplasmosis, and primary CNS B-cell lymphoma. Patients with HIV-1 dementia were identified after review of medical notes to establish whether or not they met the World Health Organization criteria for HIV-1 dementia (*2*). A Cox regression analysis was applied to the survival times from AIDS diagnosis until the onset of brain disease. For patients without brain impairment (censored cases), the time until death or last observation was entered into the calculation. The following independent variables were included in the model: age, Zidovudine treatment, CD4 counts at AIDS diagnosis, and presence of confirmed KS. There were 158 cases where the CD4 count was missing at time of AIDS diagnosis (14% of the total), but there was no difference between the CNS groups (this information was missing in 13.3% of those with KS and in 14.4% of those without CNS disease). We included a variable to indicate the missing CD4 counts in the analysis. Relative risk (*RR*) was computed with reference to the group of patients without KS.

A total of 401 out of 1109 patients (36%) were diagnosed with KS, 41 of whom (10%) had suffered a CNS disease, as compared with 154 out of 708 patients (22%) without KS [$RR = 0.410$, $P < 0.00001$, with a 95% confidence interval (CI) = 0.283 to 0.593]. Unadjusted *RR* across diagnostic groups were as follows: HIV-1 dementia, 0.42 (CI = 0.25 to 0.72), PML, 0.15 (CI = 0.02 to 1.21), cerebral toxoplasmosis, 0.44 (CI = 0.25 to 0.77), and lymphoma 0.38 (CI = 0.13 to 1.16).

Although the mean CD4 count at the time of AIDS diagnosis was higher in patients with KS (129 cells per microliter, CI = 114 to 145) as compared with that for patients without KS (104 cells per microliter, CI = 94 to 115), the mean survival time (159 weeks, CI = 147 to 171) was not different in those with KS and in those without KS if one adjusts for CD4 count at the time of AIDS diagnosis (*RR* = 0.924, $P = 0.394$, CI = 0.770 to 1.11). In particular, there was no difference between the groups (those with and those without KS) in survival time from first positive HIV test, or when the CD4 count was less than 200 cells per microliter, until death or the last observation.

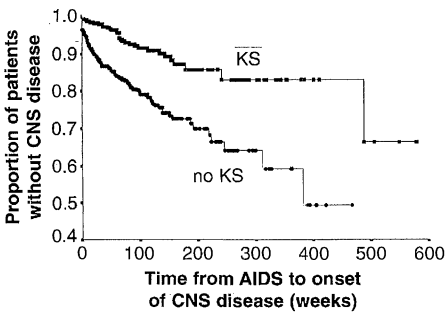

Fig. 1. Time from AIDS to onset of CNS disease in a sample of 1109 patients.

This finding suggests that KS/KSHV (or the chemokines encoded by KSHV) do not protect significantly against systemic HIV-1 progression. In contrast, the risk of developing any CNS disease (Fig. 1) was markedly lower in patients with KS as compared with those without KS (*RR* 0.385, $P < 0.0001$, CI = 0.247 to 0.601). Similar results were obtained in separate analyses for HIV-1 dementia ($P = 0.006$) and opportunistic brain disease ($P = 0.0021$). Furthermore, the immunologic status, as measured by CD4 count at onset of CNS involvement, was markedly lower in patients with KS (28 cells per microliter, CI = 16 to 40) as compared with those without KS (67 cells per microliter, $P = 0.008$, CI = 52 to 81).

Questions for further research include whether the vMIPs actually block infection of microglial cells, whether vMIPs (or cellular chemokines, or both) concentrate in large amounts in the CNS, or whether other viral or cellular cytokines in patients with KS have neuroprotective effects (*3*).

T. Baldeweg
J. Catalan
Imperial College School of Medicine, Chelsea and Westminster Hospital, London W6 8RP, United Kingdom
B. G. Gazzard
Department of HIV Medicine, Chelsea and Westminster Hospital
R. A. Weiss
C. Boshoff
Institute of Cancer Research, 237 Fulham Road, London SW3 6JB, United Kingdom
E-mail: cboshoff@icr.ac.uk

References

1. T. Baldeweg and J. Catalan, *Lancet* **349**, 1554 (1997); ———, B. G. Gazzard, *J. Neurol. Neurosurg. Psychiatr.*, in press.
2. World Health Organization, *AIDS* **4**, 935 (1990).
3. J. R. Lokensgard *et al.*, *J. Immunol.* **158**, 2449 (1997).

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Oliver Update

A Random Samples item of 1 November 1996 "'Mutant' chimp gets gene check" (p. 727) correctly described some of the fantastic rumors that have surrounded Oliver, an

African chimpanzee, since the early 1970s. Oliver's bipedal locomotion is peculiar, and many unsubstantiated rumors that qualified cytogeneticists determined Oliver's karyotype to be 2N = 47, midway between a human and a chimpanzee, led to popular suggestions that he was a hybrid.

The Random Samples item quoted a study by George Schaller (*1*), which was based on a 1976 examination of Oliver, in such a way that readers might have thought that Schaller concluded that Oliver was taxonomically unique. Schaller did indeed suggest that further study of Oliver's bipedalism would be interesting, but not because there was any doubt that Oliver was an ordinary chimpanzee, *Pan troglodytes*. Rather, he suggested that it might provide information about the physical changes necessary in an ape before it could walk easily in an upright position, including whether there had been any surgical alteration of Oliver's lower pelvis or developmental changes in his leg musculature (*1*).

Recently, the cytogenetic analysis alluded to in the Random Samples item was completed by one of us (C.M.M.), and mitochondrial DNA sequencing was also performed (J.J.E.). Our results (*2*) indicate that Oliver is a member of *Pan troglodytes* troglodytes, the Central African chimpanzee subspecies, with 48 normal chimpanzee chromosomes, and was likely trapped in Gabon.

John J. Ely
Department of Biology, Trinity University, San Antonio, TX 78212–7200, USA
Charleen M. Moore
University of Texas Health Science Center, Department of Cellular and Structural Biology, San Antonio, TX 78284–7762, USA

References

1. G. S. Schaller, unpublished report, 7 February 1976; personal communication.
2. J. J. Ely, M. Leland, M. Martino, W. Swett, C. M. Moore, *Am. J. Phys. Anthropol.* **105**, 395 (1998).

Corrections and Clarifications

■ In the Research commentary "A bridge to control" by B. Demple (*Science*'s Compass, 13 Mar., p. 1655), the accompanying figure mistakenly indicated that ferredoxin (Fd) undergoes a thiol-disulfide exchange. In fact, Fd has an iron-sulfur center that undergoes one-electron oxidation and reduction.

■ In the report "Topography of the northern hemisphere of Mars from the Mars Orbiter Laser Altimiter" by D. E. Smith *et al.* (13 Mar., p. 1686), line 18 of the second column of page 1686 should have read, in part, "topography varies by 3 km." Also, the caption for figure 7 on page 1690 should have read, "Derived atmospheric opacity (**A**) and topography (**B**) over the Ius Chasm of Valles Marineris."

■ In the report "Requirement for GD3 ganglioside in CD95- and ceramide-induced apoptosis" by R. De Maria *et al.* (12 Sept. 1997, p. 1652), the caption for figure 5B should have read, in part, "HuT78 cells were incubated for 36 hours with 20 mM PDMP (white bars) or left untreated (gray bars)...."

Letters to the Editor

Letters may be submitted by e-mail (at science_letters@aaas.org), fax (202-789-4669), or regular mail (*Science*, 1200 New York Avenue, NW, Washington, DC 20005, USA). Letters are not routinely acknowledged. Full addresses, signatures, and daytime phone numbers should be included. Letters should be brief (300 words or less) and may be edited for reasons of clarity or space. They may appear in print and/or on the World Wide Web. Letter writers are not consulted before publication.