tions of methane; gas hydrates are ubiquitous under the floor of the lake, as shown by seismic profiling⁵. The small dependence of the near-vent community on photosynthesis suggests that vents of this kind could have been important in the nascence of the unique faunistic complex of Lake Baikal consisting of 1,500 endemic species: vents could have many times served as refuges under unfavourable climates, and sources of species radiation under more favourable ones during the 20-million-year-long history of the lake. Communities of organisms built of ancient carbon are not uncommon in a marine ecosystem⁶, but this is the first time they have been found in a freshwater ecosystem.

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- Crane, K., Hecker, B. & Golubev, V. *Nature* **350**, 281 (1991).
- Gebruk, A.V., Kuznetsov, A.P., Namsaraev, B. B. & Miller, Yu. M. *Izv. RAN, Ser. biol. Iss.* **6**, 903-908 (1993).
- Lake Baikal Paleoclimate Project Members *EOS Trans. AGU* **73**, 461 (1993).
- Shanks, W. C. & Calendar, E. *Geology* **20**, 495-497 (1992).
- Scholz, C. A. et al. *EOS Trans. AGU* **74**, 465 (1993).
- Paull, C. K., Jull, A. J. T., Toolin, L. J. & Linick, T. *Nature* **317**, 709-711 (1985).

Another obese gene function

SIR — The cloning of the *obese* (*ob*) gene¹ is indeed a breakthrough in obesity research. As Rink highlighted in News and Views², the evidence strongly suggests that the normal *ob* protein is a previously undescribed hormone which regulates satiety. I would like to comment on another potential function of the *ob* gene.

When allowed free access to food, *ob/ob* mice eat more than normal *ob/ob* mice and develop obesity. However, the lean body mass of *ob/ob* mice is lower than *ob/ob* mice, and shows characteristics of stunted animals³. Furthermore, food restriction to the level of the normal *ob/ob* mouse does not reduce adiposity of the *ob/ob* mouse,

but it only causes a further decrease in lean body mass with little change in fat/lean body ratio compared with the *ob/ob* mouse fed without food restriction⁴. The same results are observed in the *fafa* rat, another genetic model of obesity caused by a single recessive gene⁵. The *fafa* rat is thought to have a homologous defect to the *db/db* mouse⁶, which may have a defective receptor for the *ob* protein².

These effects of the *ob* gene cannot be explained by suppression of appetite and subsequent reduction of food intake. Rather, the observations indicate that the *ob* protein regulates the energy partition between fat deposits and the lean body by some mechanism(s) not secondary to the effect on food intake, and that a defect in *ob* gene function causes an increased energy flow toward fat accumulation even at the expense of lean body growth.

In humans, uncommon obesity caused by a single gene has been reported. On the other hand, humans generally show increased lean body mass when the degree of obesity increases; and the treatment of obesity by energy restriction effectively reduces body fat without reducing lean body mass. Thus, it will be particularly interesting to determine the role of the human homologue of the mouse *ob* gene in human obesity. The discovery of the *ob* gene sequence will soon lead to the answer to this question. Even if this gene has little relevance to most obesity in humans, the potential use of the *ob* protein for the treatment of obesity will remain.

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Recombination in HIV-1

SIR — Globally circulating strains of human immunodeficiency virus type 1 (HIV-1) exhibit extreme genetic diversity¹⁻⁵. Phylogenetic analyses (a in the figure) have revealed two distinct 'groups' (M and O), and numerous 'sequence subtypes' within the major group M. Even though retroviruses (such as HIV-1) are highly recombinogenic⁶, recombination among viruses from different subtypes has not been considered to be a significant source of new variation in HIV-1 because evidence for coinfection with multiple divergent HIV-1 strains has remained rare⁷. Here we report an extensive analysis of published HIV-1 sequences which reveals a surprisingly large number of apparently recombinant viruses. This find-

ing has immediate consequences for our understanding of HIV-1 pathogenesis and for vaccine development⁸, and of course implies that coinfection with divergent HIV-1 strains is not as rare as previously thought.

Recombination can be detected when different genes, or different regions within the same gene, are placed by phylogenetic analysis into different sequence subtypes. A single HIV-1 isolate (MAL) has long been suspected to be recombinant, and we have recently described⁹ a detailed analysis of this viral genome in which the crossover points were localized. Here we have applied the same techniques to examine all HIV-1 isolates for which near-full-length *gag* or *env* sequences have been

Evolutionary relationships of HIV-1 sequences. a, Phylogenetic tree for representatives of each of the subtypes of HIV-1, derived as in ref. 2. For illustration, the alternative phylogenetic positions of ZR-VI191 are shown. b, Segments of recombinant viral *gag* and *env* sequences belonging to different subtypes; colour coding is as in a. Some segments (in white) cannot be designated as belonging to any currently known subtype. Localization of the breakpoints between regions of differing phylogenetic affinity is described in the table.

- Zhang, Y. et al. *Nature* **372**, 425-432 (1994).
- Rink, T. J. *Nature* **372**, 406-407 (1994).
- Johnson, P. R., Greenwood, M. R. C., Horwitz, B. A. & Stern, J. S. A. *Rev. Nutr.* **11**, 325-353 (1991).
- Dubuc, P. U. *Am. J. Physiol.* **230**, 1474-1479 (1976).
- Pullar, J. D. & Webster, A. J. F. *Br. J. Nutr.* **31**, 377-392 (1974).
- Weigle, D. S. *FASEB J.* **8**, 302-310 (1994).