Acknowledgements We thank E. Rizzo, S. Tognin, A. Donadini, A. M. Barbieri and L. Castelli for technical advice; for providing salt-washed purified ribosomal subunits, we thank T. Pestova. For sharing reagents, information and critical advice, we thank A. Bachi, T.-A. Sato, F. Loreni, F. Amaldi, P. Linder, J. Traugh, A. Johnson, A. Hinnebusch, A. Burlando, L. Spremulli, E. Villa, J. Verdiere, D. Ron, T. Dever, C. Groft and M. Foiani. This work was supported by AIRC, MURST (P.C.M., S.B.). This study was carried out under the framework of the Italian MUIR Center of Excellence in Physiopathology of Cell Differentiation.

Competing interests statement The authors declare that they have no competing financial interests.

Correspondence and requests for materials should be addressed to S.B. (biffo.stefano@hsr.it).

corrigenda

Colorectal carcinomas in mice lacking the catalytic subunit of PI(3)Kγ

Takehiko Sasaki, Junko Irie-Sasaki, Yasuo Horie, Kurt Bachmaier, Jimmie E. Fata, Martin Li, Akira Suzuki, Dennis Bouchard, Alexandra Ho, Mark Redston, Steven Gallinger, Rama Khokha, Tak W. Mak, Phillip T. Hawkins, Len Stephens, Stephen W. Scherer, Ming Tsao & Josef M. Penninger

Nature **406,** 897–902 (2000).

In this Letter, we reported that mice lacking the catalytic subunit of PI(3)Kγ on 129J backgrounds develop invasive colorectal tumours. We have observed this cancer phenotype in mouse lines derived from two different targeted embryonic stem-cell clones for at least two years. However, after backcrossing these mice onto a C57BL/6 background, we now find that the tumour phenotype has disappeared. Also, when we retargeted the allele in different ES cells using the same targeting construct, no tumours developed. Inactivation of PI(3)Kγ in mice therefore does not in itself cause colon cancer, but may require additional factors—for example, the impaired immunity of p110γ$^{-/-}$ mice may make them susceptible to tumours triggered by environmental and infectious agents[1]. Our finding that overexpression of p110γ in different human colon-cancer cells results in decreased cell growth still stands, as does the frequent downregulation of p110γ protein in colon-cancer patients (since independently confirmed[2]).

Some authors (Y.H., A.H., M.R., S.G. and T.W.M.) are not co-signatories to this statement. □

1. Erdman, S. E. *et al.* CD4^{+} CD25^{+} regulatory T lymphocytes inhibit microbially induced colon cancer in Rag2-deficient mice. *Am. J. Pathol.* **162,** 691–702 (2003).
2. Semba, S. *et al.* Down-regulation of PIK3CG, a catalytic subunit of phosphatidylinositol 3-OH kinase, by CpG hypermethylation in human colorectal carcinoma. *Clin. Cancer Res.* **8,** 3824–3831 (2002).

Cyanophages infecting the oceanic cyanobacterium *Prochlorococcus*

Matthew B. Sullivan, John B. Waterbury & Sallie W. Chisholm

Nature **424**, 1047–1051 (2003).

In this Letter, the data for host strains WH8012 and WH8109 in Fig. 1 were inadvertently reversed. This mistake does not affect any of the conclusions made in the paper. □

Zero thermal expansion in YbGaGe due to an electronic valence transition

James R. Salvador, Fu Guo, Tim Hogan & Mercouri G. Kanatzidis

Nature **425**, 702–705 (2003).

In this Letter, both the legend to Fig. 1 and the 'X-ray diffraction data' section of the Methods contain errors in the crystallographic data. In the Methods, for both YbGaGe and YbGaSn, $Z = 4$ (not 3). Hence, for YbGaGe, density $d_{calc} = 8.149\,g\,cm^{-3}$ and absorption coefficient $\mu = 57.752\,mm^{-1}$. For YbGaSn, $d_{calc} = 8.151\,g\,cm^{-3}$ and $\mu = 48.612\,mm^{-1}$. In the legend to Fig. 1, the atomic coordinates ($\times 10^4$) should be as follows: In both compounds, for Yb(1) (x, y, z) = (0, 0, 1/4). For Yb(2) (x, y, z) = (0, 0, 0). In YbGaGe, for Ga (x, y, z) = (1/3, 2/3, 0.1532) and for Ge (x, y, z) = (1/3, 2/3, 0.6128). In YbGaSn, for Ga (x, y, z) = (1/3, 2/3, 0.1634) and for Sn (x, y, z) = (1/3, 2/3, 0.6146). In addition, line 21 of the left column of page 704 should have read "…with one showing NTE and one PTE." □

Cdc6 cooperates with Sic1 and Hct1 to inactivate mitotic cyclin-dependent kinases

Arturo Calzada, Maria Sacristán, Elisa Sánchez & Avelino Bueno

Nature **412**, 355–358 (2001).

In this Letter it was reported that an amino-terminal CDK-inhibitory domain of the Cdc6 replication protein was required for efficient mitotic exit, and that removal of this domain lethally blocked mitotic exit in cells lacking the Sic1 CDK inhibitor, demonstrating essential CDK-inhibitory control of CDK activity. However, Archambault *et al.*[1] have shown that viable mitotic-exit-competent strains lacking both CDK-inhibitors can be constructed, a finding that has also been confirmed by the authors. Archambault *et al.*[1] suggest that CDK-inhibitors are not required for mitotic exit in the wild-type cell cycle. However, A.C. *et al.* (manuscript in preparation) have evidence that strains lacking both CDK inhibitors may play a non-essential role in the regulation of mitotic exit. This point is still under investigation. □

1. Archambault, V. *et al.* Genetic and biochemical evaluation of the importance of Cdc6 in regulating mitotic exit. *Mol. Biol. Cell* (in the press); advance online publication 5 September 2003 (doi:10.1091/mbc.E03-06-0384).

Sustained division of the attentional spotlight

M. M. Müller, P. Malinowski, T. Gruber & S. A. Hillyard

Nature **424**, 309–312 (2003).

The support of the University of Liverpool, where some of the work was carried out (by M.M.M., P.M. and T.G.), is acknowledged. □

