TABLE 2 The v-*myc* N-terminal domain can complement the C-terminal domain of E1a-289

Oncogenes	Foci per plate (experiments 1, 2)	Growth in soft agar	Tumours in rats
Ej-*ras* +MLV-v-*myc*	52, 62	+	3/3
Ej-*ras* +MLV-E1a289	74, 78	+	3/3
Ej-*ras* +MLV-EMCC	38, 70	+	3/3
EJ-*ras* +MLV-ME289	12, 29	+	3/3
EJ-*ras* +MLV-E1a243	165	+	3/3
EJ-*ras* +MLV-ME243	0, 0-1*	−	NT
EJ-*ras* alone	0, 0-1*	−	NT

Transformation assays were done as described in Table 1. NT, Not tested.
* Foci did not give rise to established cells.

absence of *ras* (data not shown). Expression of the chimaeric EMCC and ME-289 proteins in transformed cells was demonstrated using antibodies raised against defined regions of E1a and v-*myc* (Fig. 2). The ability of N-terminal or C-terminal transforming domains of v-*myc* and Ad5 E1a-289 to complement each other in *cis* suggests that these oncoproteins may interact with common substrates. The molecular basis for common interactions is obscure, however, because mapping studies[4,8] have not supported a relationship between structurally similar regions[9-11] and transforming activity.

The N-terminal domain of the c-*myc* protein interacts with Rb the retinoblastoma gene product and HPV-16 E7 can compete with c-*myc* for Rb binding[12]. The requirement of the HPV-16 E7 homology region of CR1 for transformation by E1a/*myc* chimaeras complements these observations and lends support to the notion that Rb binding is involved in transformation by *myc* oncogenes. But clear biochemical[3,13] and biological[14] differences between *myc* and E1a are apparent from several studies, and even in the case of Rb, *myc* seems to bind with reduced efficiency relative to E1a (ref. 12). These differences may be reflected in the greater transformation activity of E1a/*myc* chimaeras compared with *myc*/E1a chimaeras.

Functional similarities between *myc* and E1a also may not be apparent under all conditions[15,16]. For example, c-*myc* function in normal cells (transcriptional control?) may be regulated by Rb, as suggested for E2F (refs 17, 18); overexpression of c-*myc* protein in cancer cells may allow *myc* to titrate Rb, thus providing a novel E1a-like function. Despite these caveats, the apparent interchangeability of transforming domains of E1a and *myc* suggests that there may be common targets in addition to Rb (ref. 19). The E1a/*myc* chimaeras described here may provide useful reagents for their identification. □

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CORRECTIONS

Plasma fireballs formed by microwave interference in air

Y. H. Ohtsuki & H. Ofuruton

Nature **350,** 139-141 (1991).

THE fifth line in the right-hand column on page 139 states: "A standing wave should be set up in the cavity by interference between a propagating wave and a reflected wave, and in this case there should be theoretically six antinodes at which the electric field is strongest." However, because we could not identify the mode which existed in the cavity and the guide wavelength must be used in the calculation, there should be theoretically plural (at most five) antinodes in the cavity. This correction does not affect any of the conclusions in the paper.

A protein-tyrosine phosphatase with sequence similarity to the SH2 domain of the protein-tyrosine kinases

Shi-Hsiang Shen, Lison Bastien, Barry I. Posner & Pierre Chrétien

Nature **352,** 736-739 (1991).

WE have discovered errors made in the assembly of our published cDNA sequence affecting the end of the coding region in Fig. 1 of the above paper. One -g- should be inserted between nucleotides 2,028 and 2,029. Thus, the open reading frame of the PTP1C gene terminates at nucleotide 2,046 instead of nucleotide 2,082. The corresponding amino acid sequence of the last 6 residues at the C-terminus of PTP1C from nucleotides 2,029 to 2,046 is GSLKRK. The correct sequence is available on the EMBL database under accession number X62055. These errors do not affect the conclusions of the paper in any way.

ERRATUM

Preparation and structure of the alkali-metal fulleride, A_4C_{60}

R. M. Fleming, M. J. Rosseinsky, A. P. Ramirez, D. W. Murphy, J. C. Tully, R. C. Haddon, T. Siegrist, R. Tycko, S. H. Glarum, P. Marsh, G. Dabbagh, S. M. Zahurak, A. V. Makhija & C. Hampton

Nature **352,** 701-703 (1991).

THE positions of the alkali atoms, originally reported as $(0, \frac{1}{2}, z)$ with $z = 0.28$, should read $(x, \frac{1}{2}, 0)$ with $x = 0.22$. These are the positions used in the calculations for Table 1 and the positions illustrated in Fig. 2.