Nuclear Hormone Receptors, Targets for Therapeutic Intervention, July 28–29, 1997, The Ritz-Carlton, Philadelphia, PA. Contact: IBC USA Conferences, Inc., 225 Turnpike Road, Southborough, MA 01772-1749. Phone: (508) 481-6400; Fax: (508) 481-7911; E-mail: reg@ibcusa.com.

Cell Culture and Hybridomas: Quality Control and Cryopreservation Techniques, September 8–10, 1997, Rockville, MD. Contact: ATCC, Workshop Coordinator, 12301 Parklawn Drive, Rockville, MD 20852. Phone: (800) 359-7370; Fax: (301) 816-4364; E-mail: workshops@atcc.org.

Fifteenth Annual ATCC Biotech Patent Forum, September 15–16, 1997, Rockville, MD. Contact: ATCC, Workshop Coordinator, 12301 Parklawn Drive, Rockville, MD 20852. Phone: (800) 359-7370; Fax: (301) 816-4364; E-mail: workshops@atcc.org.

Cytogenetic and Fluorescence *in Situ* Hybridization, September 17–19, 1997, Rockville, MD. Contact: ATCC, Workshop Coordinator, 12301 Parklawn Drive, Rockville, MD 20852. Phone: (800) 359-7370; Fax: (301) 816-4364; E-mail: workshops@atcc.org.

Gene Therapy in Cancer, September 27–30, 1997, Athens, Greece. Deadline for abstract submission: June 30, 1997. Contact: Dr. John G. Delinassios, International Institute of Anticancer Research, 1st km Kapandritiou-Kalamou Road, P.O. Box 22, Kapandriti, Attiki 19014, Greece. Phone and Fax: (0030) 295-53389.

Apoptosis, October 2–5, 1997, Athens, Greece. Deadline for abstract submission: June 30, 1997. Contact: Dr. John G. Delinassios, International Institute of Anticancer Research, 1st km Kapandritiou-Kalamou Road, P.O. Box 22, Kapandriti, Attiki 19014, Greece. Phone and Fax: (0030) 295-53389.

Hormones, Blood Cells, and Immunity, October 3–8, 1997, Obernai (Strasbourg), France. Deadline for applications: June 15, 1997. Contact: Dr. Josip Hendekovic, European Science Foundation, 1 quai Lezay-Marnesia, 67080 Strasbourg Cedex, France. Phone: (33) 3-88-76-71-35; Fax: (33) 3-88-36-69-87; E-mail: euresco@esf.org.

Hybridoma Technology and Monoclone Antibody Product Development, October 6–9, 1997, Rockville, MD. Contact: ATCC, Workshop Coordinator, 12301 Parklawn Drive, Rockville, MD 20852. Phone: (800) 359-7370; Fax: (301) 816-4364; E-mail: workshops@atcc.org.

Invasion and Metastasis, October 7–10, 1997, Athens, Greece. Deadline for abstract submission: June 30, 1997. Contact: Dr. John G. Delinassios, International Institute of Anticancer Research, 1st km Kapandritiou-Kalamou Road, P.O. Box 22, Kapandriti, Attiki 19014, Greece. Phone and Fax: (0030) 295-53389.

New Anticancer Agents, October 12–15, 1997, Athens, Greece. Deadline for abstract submission: June 30, 1997. Contact: Dr. John G. Delinassios, International Institute of Anticancer Research, 1st km Kapandritiou-Kalamou Road, P.O. Box 22, Kapandriti, Attiki 19014, Greece. Phone and Fax: (0030) 295-53389.

Eighth Conference on DNA Topoisomerases in Therapy, October 15–17, 1997, Amsterdam, the Netherlands. Contact: The European Cancer Centre, P.O. Box 7057, NL-1007 MB Amsterdam, the Netherlands. Phone: 31 (0)20 644-4500; Fax: 31 (0)20 644-4551; E-mail: ecc@euronet.nl.

Correction

Recently, it was brought to our attention that in our paper entitled "Transforming Growth Factor β1 Suppresses Genomic Instability Independent of a G_1 Arrest, *p53*, and *Rb*" (Glick *et al.*, Cancer Res., *56:* 3645–3650, 1996), we incorrectly stated that Garrigue-Antar *et al.* (Cancer Res., *55:* 3982–3987, 1995) reported a mutation in the TGF-β type II receptor in the TGF-β-resistant FaDu human carcinoma cell line, when it is actually wild type. This misstatement does not alter the results of our study showing that FaDu cells do not respond to growth inhibition or suppression of PALA resistance by TGF-β1. To determine the basis for nonresponsiveness to TGF-β, FaDu cells were transfected with the TGF-β response plasmid p800luc containing the PAI promoter (Abe *et al.*, Anal. Biochem., *216:* 276–284, 1994). TGF-β1 caused a 3-fold induction of luciferase activity in the control H4 mouse keratinocyte cell line, whereas there was no induction in either the FaDu cell line or the HCT116 colon carcinoma cell line, which contains a mutated TGF-β type II receptor (Fig. 1). This indicates that the lack of responsiveness to TGF-β1 in the FaDu cells is due to a defect in the TGF-β1 signaling pathway rather than other alterations indirectly affecting growth regulation by TGF-β1. Additionally, this suggests that the suppression of PALA resistance by TGF-β1 is linked to the signaling pathway regulating gene expression. We thank Dr. Michael Reiss for drawing our attention to the status of the TGF-β type II receptor in FaDu cells.

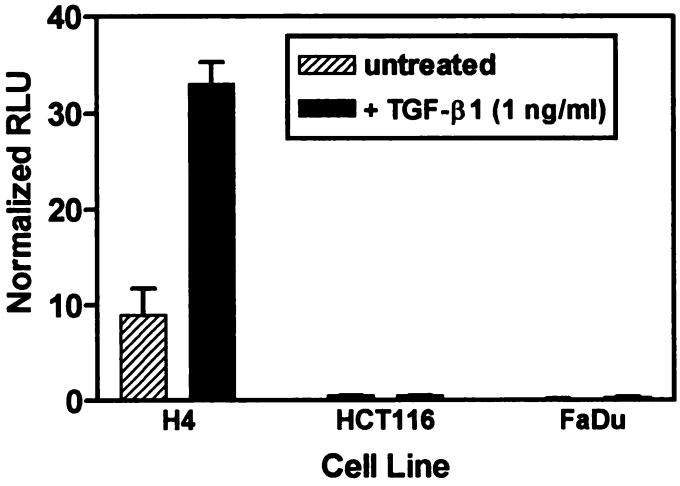

Fig. 1. FaDu cells are defective in the induction of gene expression by TGF-β1. Triplicate wells were cotransfected with the plasmid p800Luc containing the TGF-β response element from the PAI promoter (Abe, *et al., Anal. Biochem., 216:* 276–284, 1994) and the internal control pRL-TK (Promega) and treated with 1 ng/ml TGF-β1 for 48 h. Luciferase activity was measured using the dual luciferase system (Promega), with luciferase activity from p800luc normalized to that obtained from pRL-TK. Similar results were obtained in two additional experiments. *RLU*, relative luciferase units.

Adam B. Glick
Laboratory of Cellular Carcinogenesis and Tumor Promotion
National Cancer Institute
Bethesda, MD 20829