Erratum

X. Tu et al. Subcellular Localization of IRS-1 in Cell Proliferation and Differentiation. Horm Metab Res 2003; 35: 734–739 and Erratum Horm Metab Res 2004; 36: 72

Figures **1 a, b**, **2** and **4** were printed in black and white but should have been printed in colours as shown below:

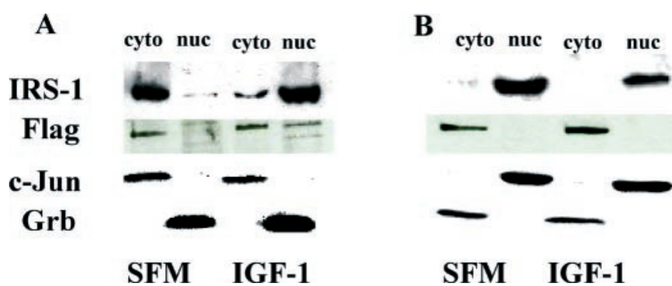

Fig. **1** Subcellular localization of endogenous IRS-1 and exogenous δ-Pleckstrin (PH) IRS-1 in R^- derived cells. The cells used were R^+ cells (panel A) and R^-/T cells (panel B). R^+ cells are R^- cells expressing the human IGF-I receptor, R^-/T cells have no IGF- receptors and express the SV40 T-antigen. Both cell lines were stably transfected with a retrovirus expressing a mutant IRS-1, with a deletion of the PH domain and tagged with a FLAG epitope at the 3' end (34). The cells were left in serum-free medium or stimulated for 8 h with IGF-I (50 ng/ml). Lysates were made, subcellular fractions separated and western blots carried out as described in Methods and Materials. The blots were developed with antibodies to IRS-1, FLAG, c-jun and Grb2.

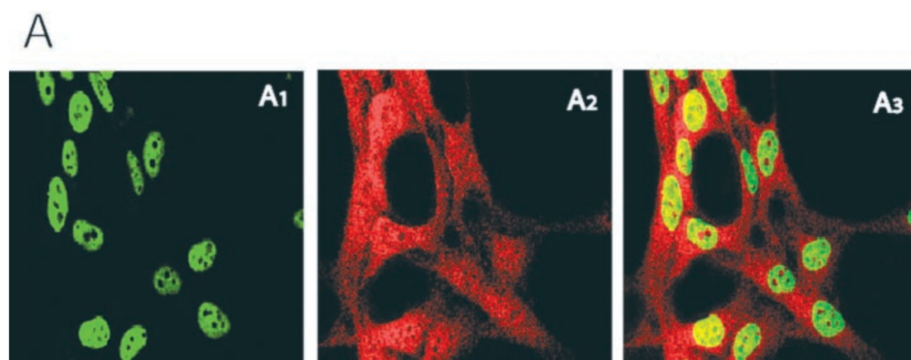

Fig. **2** Nuclear and nucleolar localization of IRS-1 in AR5 cells. AR5 cells are immortalized human diploid fibroblasts that express the SV40 T-antigen (see Methods and Materials). Exponentially growing cells were stained with antibodies to T-antigen (A1, green) and to IRS-1 (A2, red). These pictures and the merged picture (A3) show that T-antigen is exclusively localized to the nuclei (but not the nucleoli), while IRS-1 is found all over the cell, including the nucleus and the nucleolus.

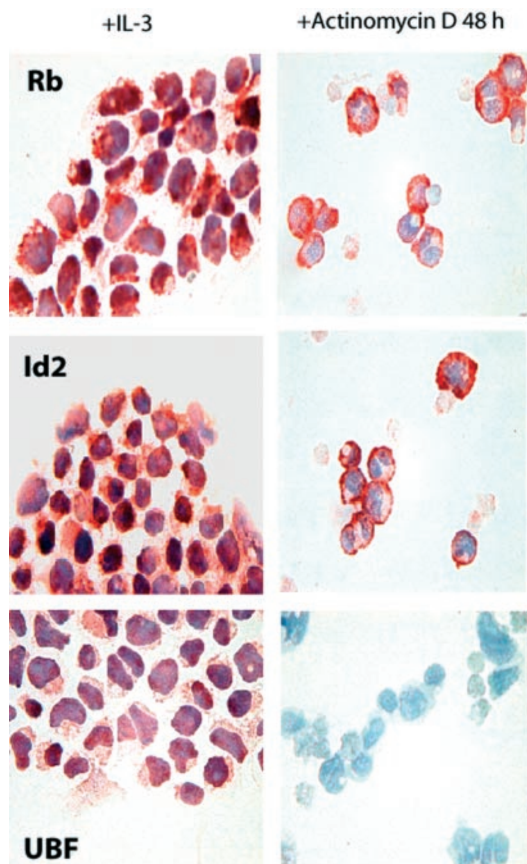

Fig. **4** Subcellular localization of nuclear proteins in cells treated with Actinomycin D. 32D IGF-IR cells were grown in serum supplemented with Interleukin-3 (IL-3). Aliquots were treated with Actinomycin D (0.5 μg/ml) for 48 h (right panels). Untreated cells (left panels) served as controls. Cells in each group were fixed and stained with antibodies to pRb (upper panels), ID2 (middle panels) and UBF (lower panels), and counterstained with hematoxylin. All 3 proteins are predominantly nuclear while the cells are growing exponentially, but pRb and ID2 translocate to the cytoplasm when ribosomal RNA synthesis is inhibited by Actinomycin D. UBF disappears with the disappearance of the nucleolus. Controls unstained with antibodies have the same appearance as the cells treated with Actinomycin D and stained for UBF (see also [40]).

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