Erratum

A. G. Renehan et al. Acromegaly and Colorectal Cancer: A Comprehensive Review of Epidemiology, Biological Mechanisms, and Clinical Implications. Horm Metab Res 2003; 35: 712 – 725 and Erratum Horm Metab Res 2004; 36: 70 – 71

Figures **3 c** and **4 a, b** were printed in black and white but should have been printed in colours as shown below:

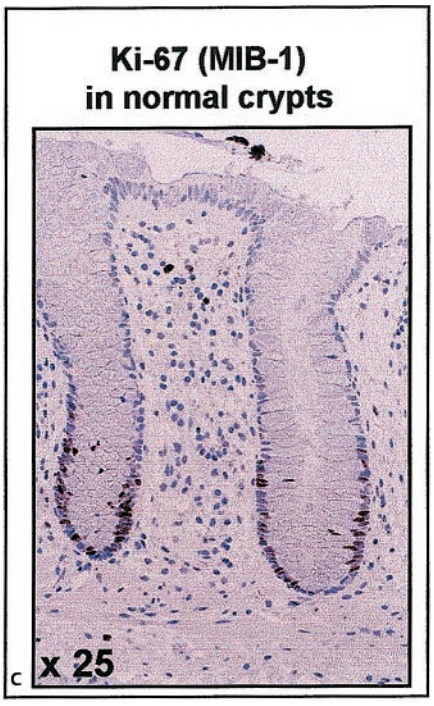
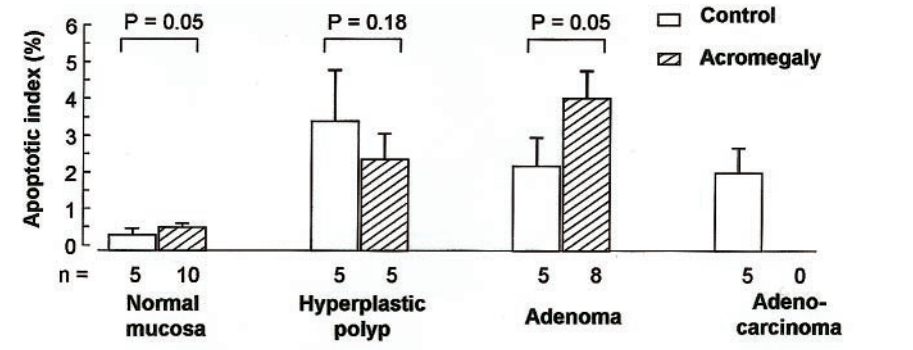
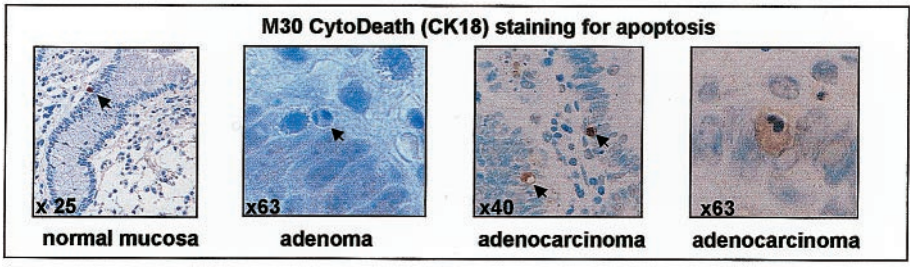
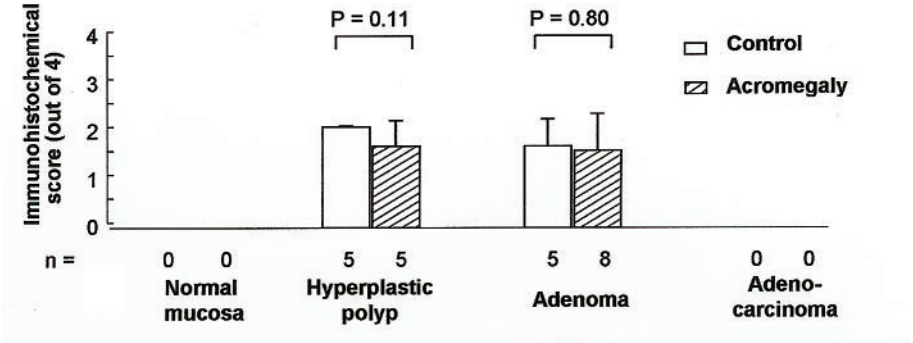


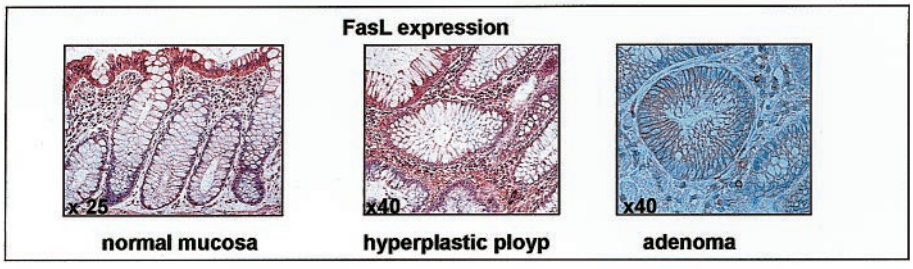
Fig. **3** Crypt cell positional analysis – acromegaly versus control. "Pinch" biopsies of macroscopically normal mucosal were taken from the left colon at 40 cm during screening colonoscopy in 12 acromegalic patients: controls were biopsies, taken from the same site in 15 non-acromegalic individuals, and without a family history of colorectal cancer, undergoing screening colonoscopy. To maintain crypt orientation, specimens were laid on microfilter paper discs, prior to fixation in 4% neutral buffered formalin for 24 h and paraffin embedded taking care to maintain crypt orientation. Several 3 µm sections (every eighth section to avoid duplicate counting of labelled cells in serial sections) were cut, stained with H & E, and orientation checked. Only crypts that were sectioned longitudinally were selected and divided into two equal halves and scoring performed by crypt cell positional analysis for labelled cells (Ki-67-MIB-1, Immunotech, Marseille, France) (**c**). In total, 274 hemi-crypts were analysed for acromegalics and 274 hemi-crypts for controls. The data were analysed using the in-house WinCrypts software with results represented as frequency plots of labelled cells at each cell position in the crypt (starting at position 1 in the crypt base) (**a, b**). Frequency plots were then compared through sequential estimates of the probability of differences occurring at each cell position while simultaneously taking account of the ten adjacent cell positions (**d**). As there is considerable variability from person to person, only P values < 0.001 relative to at least three adjacent and consecutive cell positions were considered biologically significant. Original magnifications as indicated.





a





b

Fig. **4** Apoptotic activity and FasL expression – acromegaly versus control. (**a**) Apoptosis within tissues was assessed using the CK18 monoclonal antibody (M30 CytoDeath: Boehringer, Mannheim, Germany) which recognises cleaved products of caspase 3 activity, a marker of early apoptosis. The immunohistochemical method was performed in accordance with the manufacturer's instructions. CK18 staining was compared with the morphological assessment in H & E sections cut in series. Cells were only scored as apoptotic if they were CK18 positive and/or contained both pyknotic nuclei showing evidence of nuclear condensation and eosinophilic, condensed cytoplasm with or without surrounding apoptotic bodies (arrows). As baseline apoptotic activity is very infrequent and not ordered in normal human intestinal mucosa, crypt cell positional analysis was not appropriate. Thus, in both normal and tumour tissues, the apoptotic index (AI) was determined as the number of positive cells expressed as a percentage, counting at least 2000 epithelial cells per specimen. Detection of apoptosis and confirmation of morphological criteria required the use of at least the ×40 magnification oil lens. Detection of apoptosis by CK18 correlated well with detection by morphological assessment (for indices, n = 15: $r_s = 0.66$: $p = 0.01$). (**b**) FasL expression in normal and tumour sections was determined using a standard peroxidase ABC approach with a rabbit polyclonal anti-human FasL-specific IgG (Santa Cruz Biotechnology, Santa Cruz, CA, USA) at 0.1 µg/ml. In tumour specimens, FasL expression was semi-quantified using an immunohistochemical score 0 = negative; 1 = 0 – 25 % staining; 2 = 25 – 50 % staining; 3 = 50 – 75 % staining; 4 = 75 – 100 % staining, and averaged together. Negative sections were re-stained to re-assess immunoreactivity. Original magnifications as indicated.