(153.4 ± 14.17 in arm A, 111.9 ± 15.0 in arm B, P = not significant). It follows that, before treatment, groups A and B of the study in question were equivalent in terms of CFU-GM count.

A comparison between the two 0 days in terms of absolute values of CFU-GM is, therefore, inappropriate because at that time the comparison would be between an MPA-treated group (arm B) and a non-MPA-treated group (arm A).

(b) Our hypothesis of a "protective" bone marrow effect of MPA is based on the analysis of the variance for paired data [1] which shows a significantly different behaviour between the values of CFU-GM observed at time 0 and 14 in MPA-treated and non-treated patients within the two groups.

This analysis does not compare the absolute average value of CFU-GM at time 14, as this data is not representative of the phenomenon studied, but the behaviour in time of bone marrow activity in MPA-treated and non-treated cases. This was significantly different in the two groups and this seems to support the hypothesis of a myeloprotective effect of the drug at the nadir of CT.

Long-term bone marrow rescue, on the other hand, evaluated on the 30th day from the beginning of the last of the three CT cycles, was not affected by treatment with MPA (see analysis).

With regard to the explanation for the increase on day 14 of the number of progenitors in 3 cases, we think that the reasons for this could be problems relating to the absorption or metabolic pathway of the drug in the stem cells.

We agree with the criticism regarding the value of our clinical data, supporting a lower haematological toxicity based on a few cases in whom lower degrees of toxicity were observed.

The data concerning low haematological toxicity observed by us in MPA-treated cases requires further study on larger populations, as we suggested.

As far as the role of MPA as a myeloprotective agent is concerned, in the era of haemopoietic growth factors, we believe that the drug is not an alternative to growth factors, but a complement to them. If our data are confirmed, MPA could be used before and during chemotherapy, while haemopoietic growth factors such as granulocyte colony-stimulating factor or GM-colony-stimulating factor could be employed shortly before the nadir of chemotherapy. In this setting, we would expect that, working on a bone marrow reserve "protected" from chemotherapy by MPA, a better effect of growth factors could be observed and thus perhaps lower doses could be used.

1. Amadori D, Frassineti GL, Flamini E, *et al.* Clinical and laboratory evaluation of the myeloprotective effect of medroxyprogesterone acetate in head and neck cancer. *Eur J Cancer* 1992, **28A**, 1331–1334.

Correction

Role of Interleukin-2 in Regulating Lymphocyte Activation and Recirculation by C. Fortis *et al.* In this article published in Vol. 29A, No. 3, pp. 474–475, Fig. 1 was incorrect. The correct version of Fig. 1 is reproduced below:

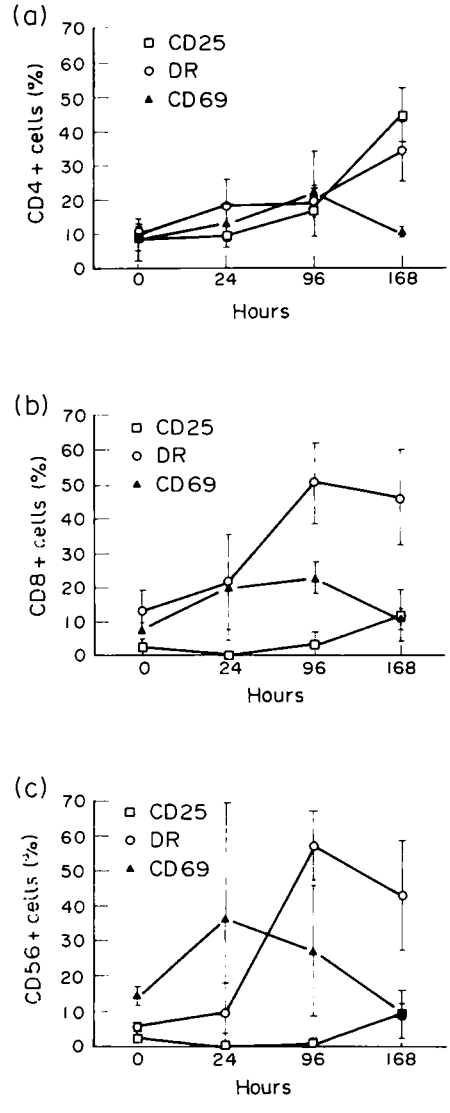

Fig. 1. Percentage of circulating (a) CD4+, (b) CD8+ and (c) CD56+ cells bearing surface activation markers during a cycle of rIL-2 administration.