Effect of growth hormone on human alveolar macrophage oxidative metabolism

M P Keane, R Coakley, R Costello, S J O'Neill

Abstract

Background – Growth hormone (GH) has diverse immunological actions and has been shown to augment oxidative metabolism in rat peritoneal and porcine alveolar macrophages and both human and animal neutrophils. A study was performed to determine the effects of GH on human alveolar macrophages in vitro.

Methods – Macrophages were harvested from 10 patients undergoing bronchoalveolar lavage and incubated with 0, 10 and 100 nmol/ml GH for four hours. Oxidative metabolism was assessed by means of a fluorescent assay using FMLP and E. coli as stimulants. Fluorescence was measured using flow cytometry.

Results – No difference in basal or stimulated oxidative metabolism was found between the GH and control groups.

Conclusions – GH does not have a direct stimulatory action on human alveolar macrophages in vitro. However, this does not exclude an indirect effect in vivo. The results contrast with previous studies on animal alveolar macrophages.

Keywords: growth hormone, human alveolar macrophages, oxidation.

Results

There was no significant difference between the control and the growth hormone groups at either concentration (fig 1).

![Figure 1: Effect of growth hormone (GH) on oxidative metabolism. Values are mean (SE).](http://thorax.bmj.com/)
Discussion
Our results contrast with previously described results in animal macrophages. It is possible that the effects of GH on human macrophages are mediated in vivo by IGF-1 or somatomedin C, the synthesis of both of which are stimulated by GH. However, GH has been shown to prime human neutrophils directly and IGF-1 antibodies do not antagonise this effect. GH augments human granulopoiesis in vitro by inducing synthesis of somatomedin C. The fact that we demonstrated no increase in oxidative metabolism with GH stimulation rules out an effect mediated via macrophage-derived somatomedin C or IGF-1. It does not, of course, exclude a more distant paracrine mode of action in vivo.

While our method of measuring oxidative metabolism differs from the other studies on GH, it has been previously validated. It measures intracellular reactive oxygen metabolite release, but it is unlikely there would be a rise in extracellular free radicals without a rise in intracellular levels.

Functional heterogeneity of diverse macrophage and T cell properties has been demonstrated both for intra-species populations of macrophages and inter-species populations of T cells. We propose that our failure to demonstrate an augmenting effect of GH on human alveolar macrophage oxidative metabolism represents a further example of interspecies functional heterogeneity. In conclusion, we have shown that growth hormone fails to stimulate oxidative metabolism directly in human alveolar macrophages. This does not rule out an indirect effect in vivo.