our study indicate that it is unlikely that inhibitors of calcineurin activation can reduce cardiac hypertrophy induced by hemodynamic overload. It remains possible that calcineurin functions as a modulator of cardiac hypertrophy arising from less-common etiologies, such as endocrine or genetic disorders. However, ventricular hypertrophy induced by pressure overload, commonly associated with hypertension or valvular disease in humans, may involve the activation of other molecular pathways.

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## No evidence for MUC 1-induced apoptosis

*To the editor*—We note the paper of Agrawal *et al*.[1] which, in contradiction to our earlier report (Gimmi *et al*., ref. 2), could not find evidence of MUC 1-induced apoptosis of T cells.

Prior to the Agrawal *et al*. publication, one of the authors of Gimmi *et al*. reported that he was unable to reproduce the apoptosis finding and indicated that a colleague in another institution had also failed to reproduce that finding. In view of the significance of this disagreement, Dana-Farber Cancer Institute appointed an internal committee of experienced immunologists to examine our paper and define an appropriate action. The committee recommended that the Nadler laboratory repeat the apoptosis experiments that it had previously performed. These new experiments were carefully supervised and reviewed, and they showed that neither purified MUC 1 nor stable transfectants expressing MUC 1 cDNA caused detectable apoptosis. Hence we agree with Agrawal *et al*. that apoptosis is not involved in MUC 1 induced T cell dysfunction.

We do not know why our previously reported results were wrong and regret the confusion that we have caused in the scientific literature. The role of MUC 1 in cancer cell biology remains obscure.

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