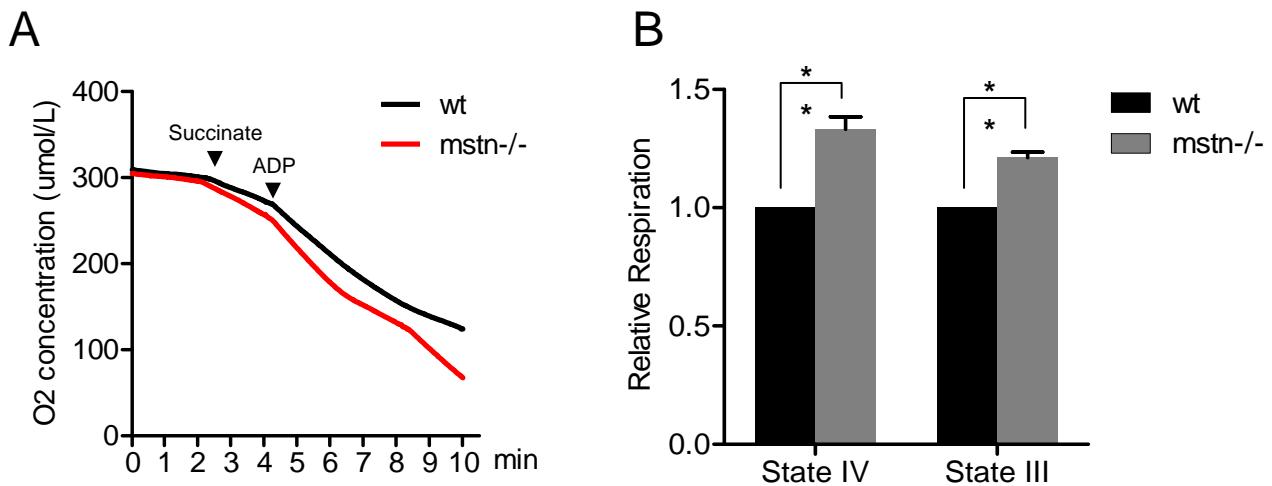
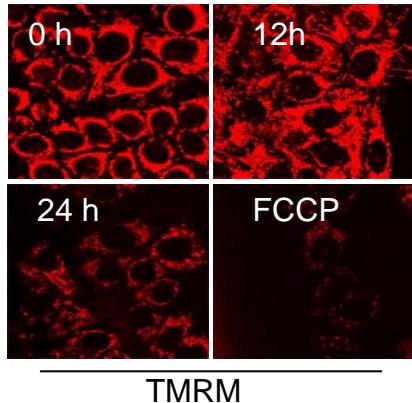


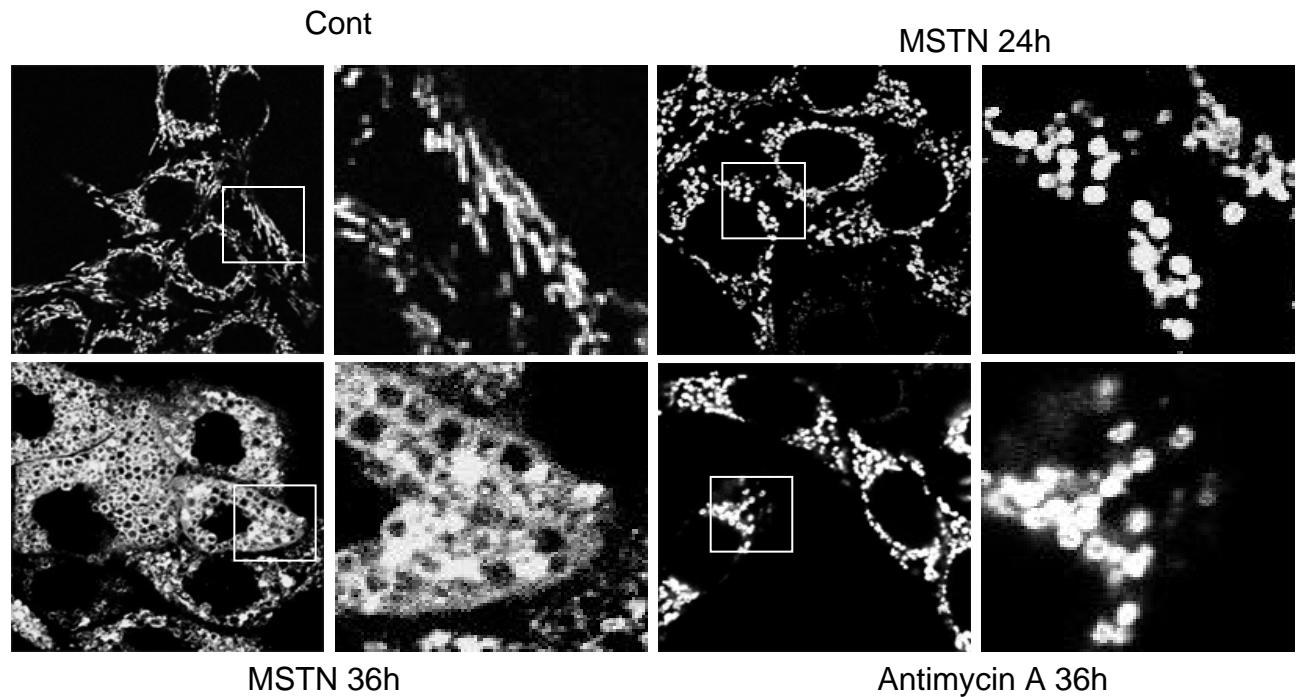
**Figure S1. Oxygen consumption is promoted in *mstn*-/- mice.**



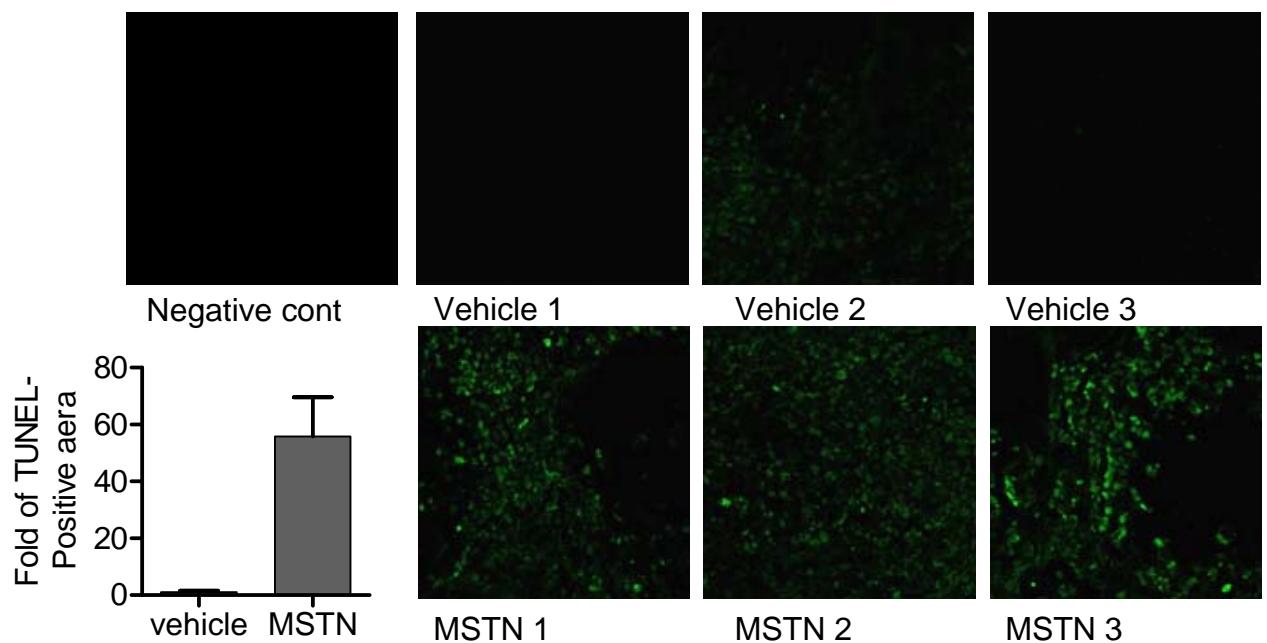
**Figure S2 Myostatin induces the decrease of mitochondrial membrane potential in cancer cells**



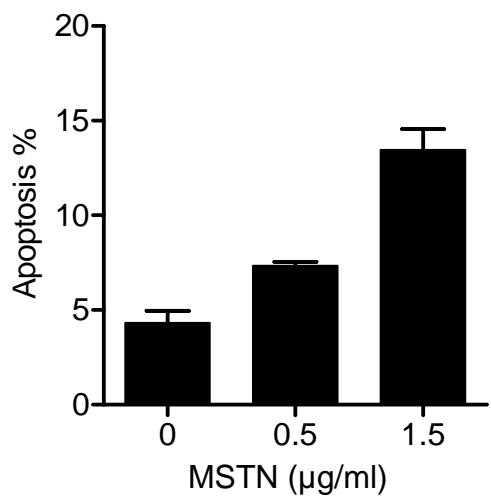
**Figure S3 Myostatin alters mitochondrial morphology of the cancer cells**



**Figure S4 TUNEL staining of tumor sections from different mice.**

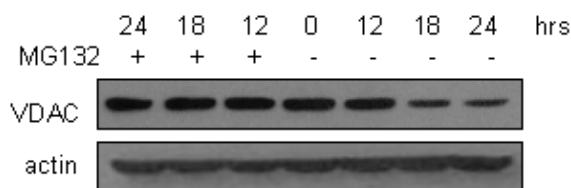


**Figure S5 Myostatin inhibit the development of B16F10-melanoma.**

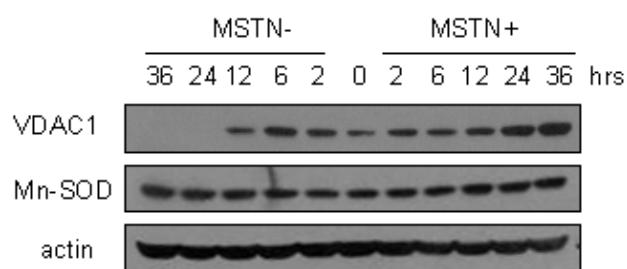


**Figure S6 Myostatin induced expression of VDAC1 was not due to either transcriptional regulation or increased de novo protein synthesis, but probably by enhancing VDAC1 stability.**

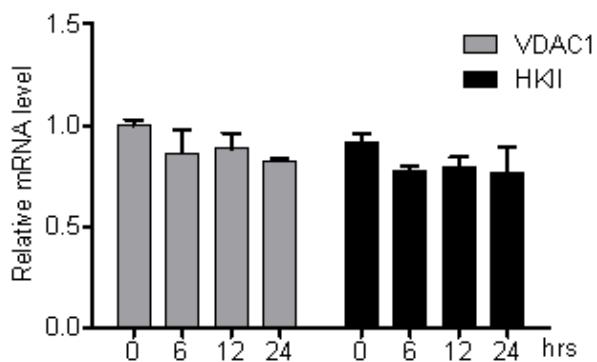
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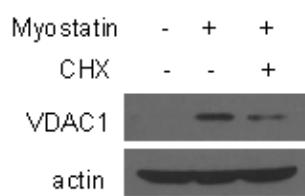
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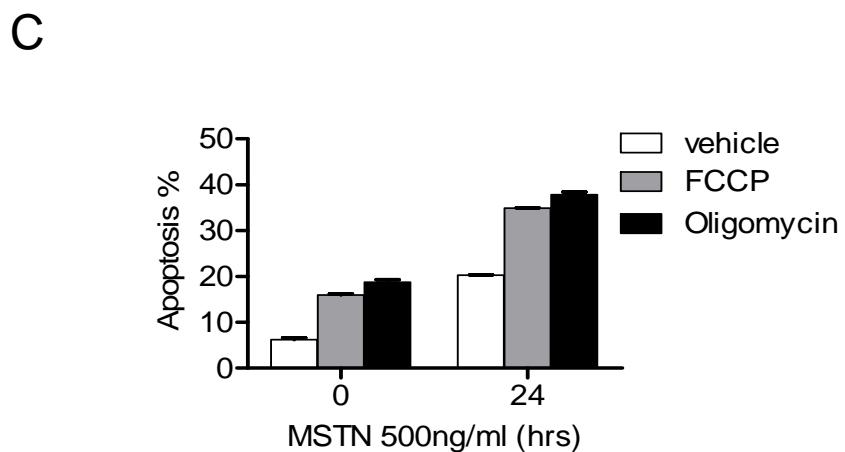
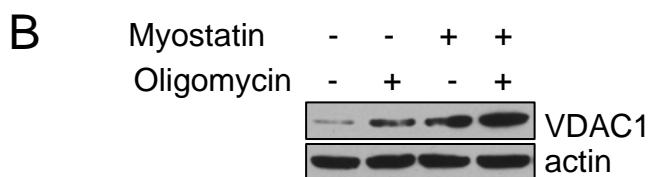
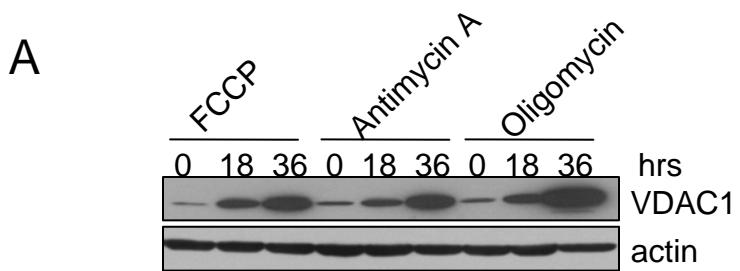
C



D

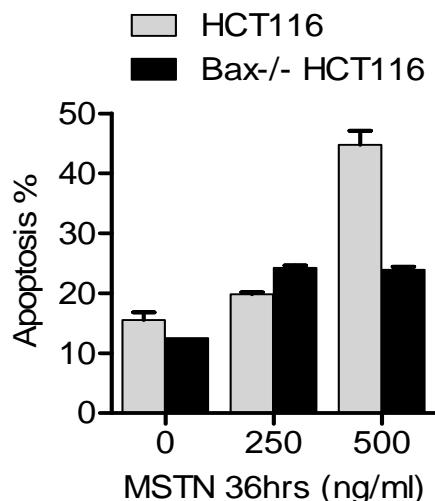


**Figure S7 Perturbation of oxidative phosphorylation results in VDAC1 upregulation**

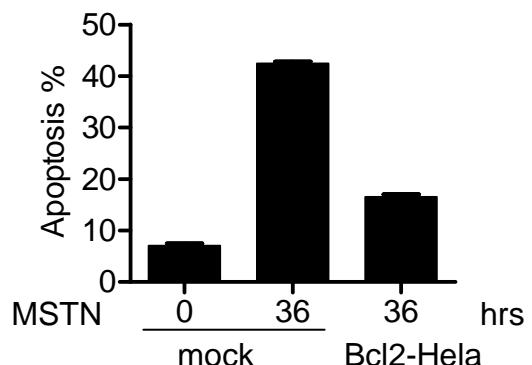


**Figure S8** The cells with Bax-deficiency or overexpression of Bcl-2 were significantly resistant to myostatin-induced apoptosis.

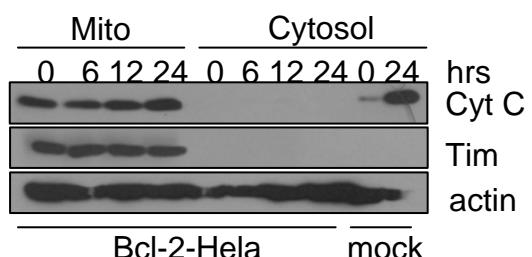
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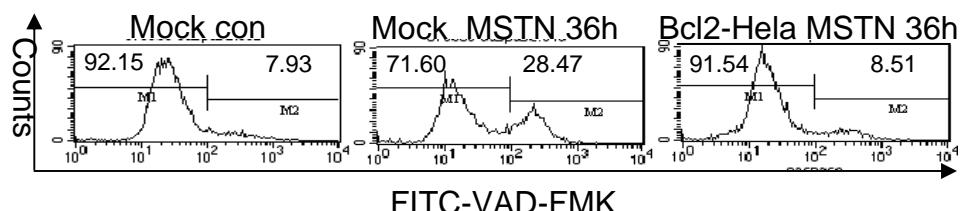
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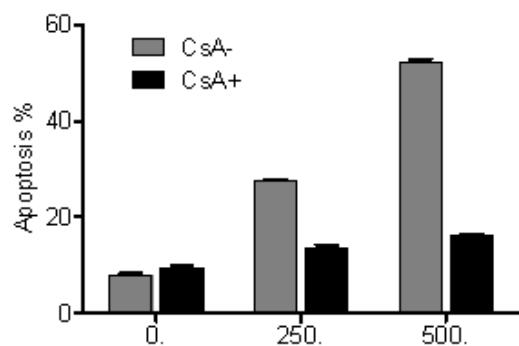
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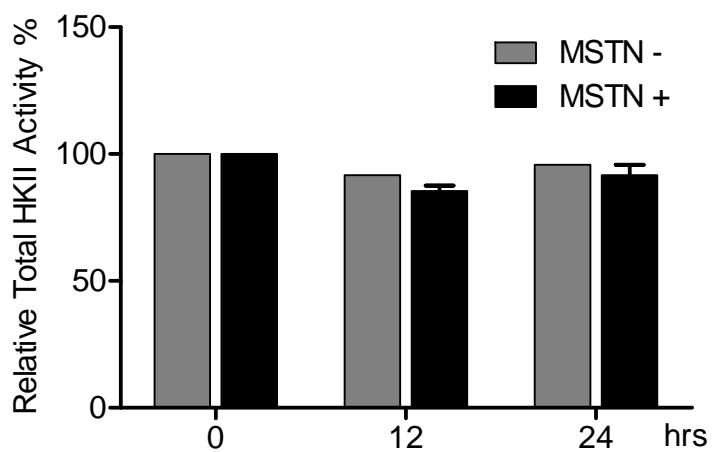
D



**Figure S9. CsA inhibits myostatin-induced apoptosis.**



**Figure S10. Total HKII activity maintained in the same level even after 24 hours myostatin treatment, when the protein level of HKII has been decreased.**



**Fig-S11 Sensitivities of different cancer cell lines to myostatin are highly correlated with their glycolytic activity.**

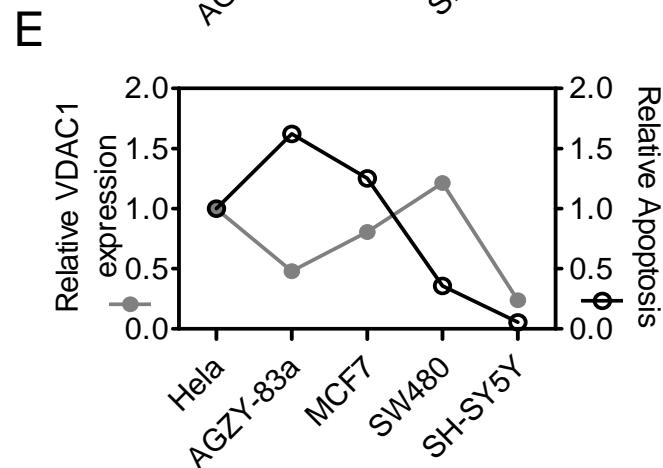
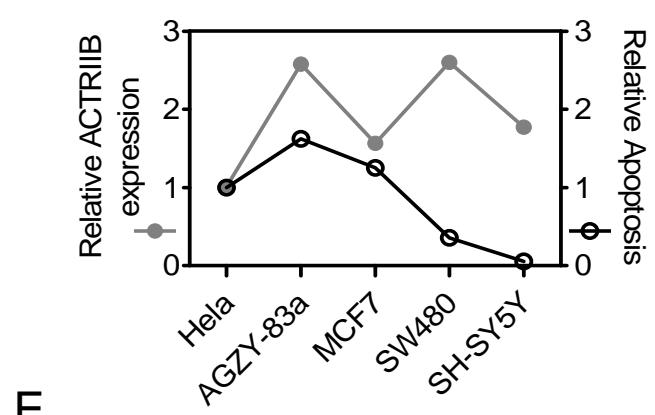
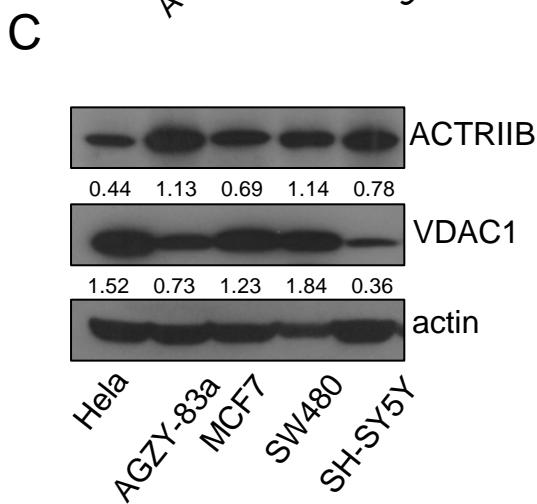
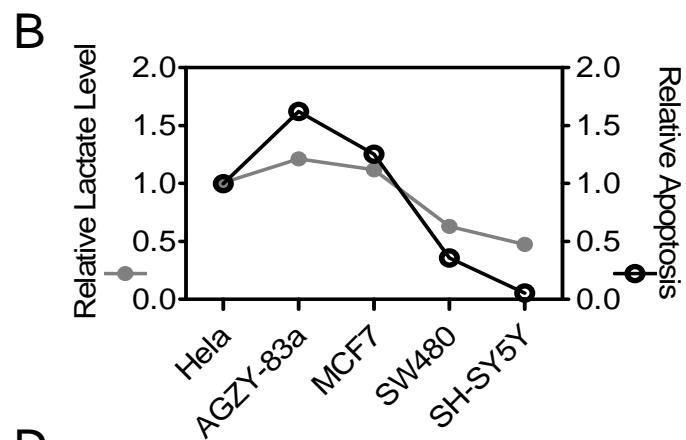
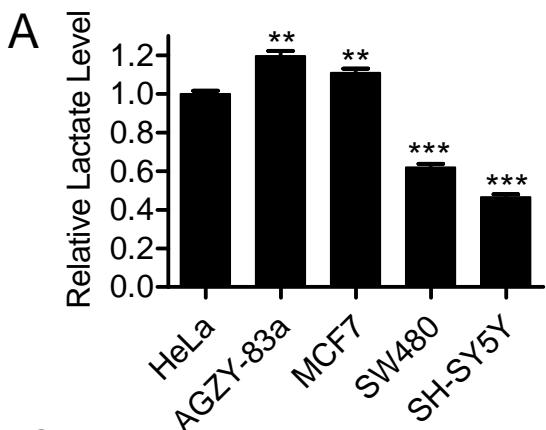
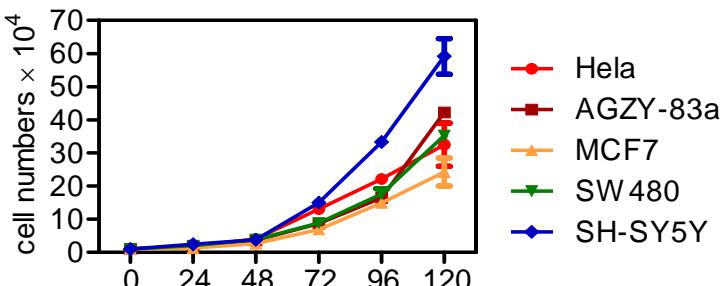
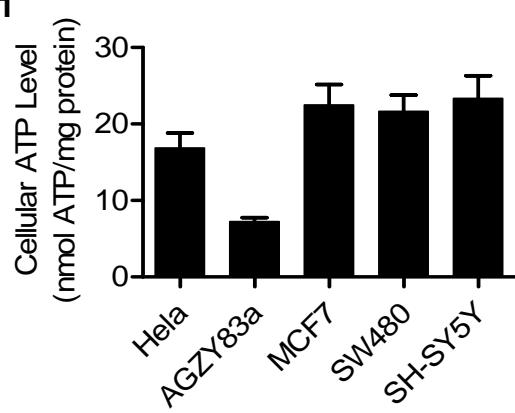


Fig-S11 Sensitivities of different cancer cell lines to myostatin are highly correlated with their glycolytic activity. (continued...)

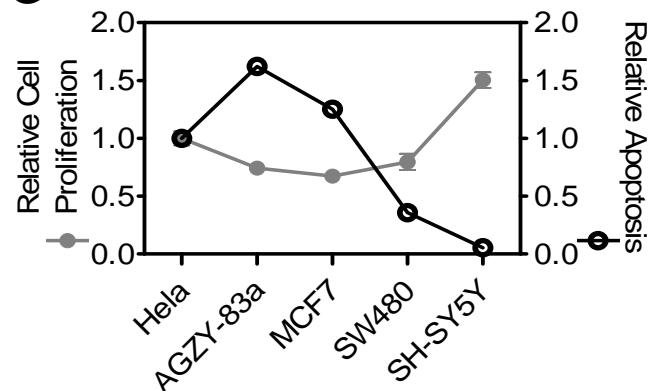
F



H



G



I

