CALIFORNIA STATE SCIENCE FAIR
2005 PROJECT SUMMARY

Name(s)
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Project Number
S0424

Project Title
Proliferation and Extracellular Matrix Remodeling in Tumor Cells HT-lo/diss and HT-hi/diss

Abstract

Objectives/Goals
There are two objectives: 1. To determine if the HT-lo/diss (45a) and the HT-hi/diss (47a) tumor cells are in fact different in extracellular matrix (ECM) remodeling and 2. if the cell lines are different in proliferation in 3-D.

Methods/Materials
HT-Proliferation in 3D-Collagen: Same numbers of cells were seeded in 7 sets of 3 wells for each cell line. The cells were suspended in collagen for a 3-D environment effect. Each set represented a day (Day 0 to Day 6). Each day the corresponding set was used and cells were isolated from the collagen, counted, and the numbers recorded.
Collagen Contraction Assay: Same numbers of cells were seeded in 2 sets of 3 wells for each cell line. Cells were suspended in collagen. 1 set per cell line had solutions of dimethylsulphoxide overlaid on the collagen; the other contained solutions of control ilomastat. After 5 days, the collagen was cut from the sides of the well and the amount of contraction was measured on the 8th day.
Materials: HT-lo/diss (45a) and HT-hi/diss (47a) from Scripps Research Institute; Control Ilomastat, Dimethylsulphoxide, Dispase (Calbiobchem); Invitrogen Dulbecco Modified Eagle Medium with 10% Fetal Bovine Serum; Sigma-Aldrich Collagen (3.0 mg/ml density); incubator (37¡aC in 5% CO2/95% air and passaged at confluence); 24-well plates

Results
Results from HT-Proliferation in 3-D Collagen, demonstrate that 45a cells have a longer doubling time than 47a cells when placed in 3-D collagen. Because the cells lines grow at the same rates in 2-D collagen, it is the 3-D collagen that slows the proliferation of 45a.
Results from Collagen Contraction show 45a contracts collagen less than 47a in 3-D collagen, meaning it is less able to modify its environment. Thus, 45a is less able in ECM remodeling when compared to 47a.

Conclusions/Discussion
My conclusion is that in vitro 3-D collagen proliferation is impaired for 45a and that the proliferation difference between the cell lines is not inherent but due to the 3-D collagen environment. Also, 45a is found to be less able in ECM remodeling than 47a. Although the result of this research is only a small part of the overall project, when pieced together, it will offer a more comprehensive understanding of the mechanics of tumor growth and metastasization. Such an insight will pave the road to discover the means to inhibit tumor growth and metastasization and the eventual treatment of cancer.

Summary Statement
I investigated the differences in the proliferation and extracellular matrix remodeling abilities of two similar tumor cell lines.

Help Received
Used the equipment of Dr. James Quigley's lab at the Scripps Research Institute under the supervision of Dr. Elena Deryugina