

## Neutralizing tumor growth in embryonic stem cell therapy

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Researchers at the Hebrew University of Jerusalem have discovered a method to potentially eliminate the tumor-risk factor in utilizing human embryonic stem cells.

Their work paves the way for further progress in the promising field of stem cell therapy.

Human embryonic stem cells are theoretically capable of differentiation to all cells of the mature <a href="human body">human body</a> (and are hence defined as "pluripotent"). This ability, along with the ability to remain undifferentiated indefinitely in culture, make regenerative medicine using human embryonic stem cells a potentially unprecedented tool for the treatment of various diseases, including diabetes, Parkinson's disease and heart failure.

A major drawback to the use of stem cells, however, remains the demonstrated tendency of such cells to grow into a specific kind of tumor, called teratoma, when they are implanted in laboratory experiments into mice. It is assumed that this tumorigenic feature will be manifested upon transplantation to human patients as well. The development of tumors from embryonic stem cells is especially puzzling given that these cells start out as completely normal cells.

A team of researchers at the Stem Cell Unit in the Department of Genetics at the Silberman Institute of Life Sciences at the Hebrew University has been working on various approaches to deal with this



problem.

In their latest project, the researchers analyzed the <u>genetic basis</u> of tumor formation from human embryonic stem cells and identified a key gene that is involved in this unique tumorigenicity. This gene, called survivin, is expressed in most cancers and in early stage embryos, but it is almost completely absent from mature normal tissues.

The survivin gene is especially highly expressed in undifferentiated human embryonic stem cells and in their derived tumors. By neutralizing the activity of survivin in the undifferentiated cells as well as in the tumors, the researchers were able to initiate programmed cell death (apoptosis) in those cells.

This inhibition of this gene just before or after transplantation of the cells could minimize the chances of tumor formation, but the researchers caution that a combination of strategies may be needed to address the major safety concerns regarding tumor formation by human embryonic stem cells.

Source: Hebrew University of Jerusalem (<u>news</u>: <u>web</u>)

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