

**Human Embryonic Stem (HESC) – Be Aware!!
HESC can potentiate tumor formation and proliferation!!**

From: Dr Mal Hooper [mailto:general@spinalrehab.com.au]
Sent: Thursday, 28 August 2008 1:22 PM
To: 'Perri'
Cc: 'jarrad anderson'
Subject: FW: Embryonic Stem Cells - be aware!

Perri – bottom line – what this means is that if your dad had latent cancer cells within his body i.e. prostate then the 'undifferentiated' embryonic stem cells could cause proliferation and activation of dormant cancer cells i.e. *fuel on a fire situation*.

Umbilical and adult derived stem cells are differentiated NOT undifferentiated – hence the recommendation to consider the Beike Corporation program which uses Umbilical derived Mesenchymal Stem Cells with Neurotrophic factors to enhance cell proliferation.

Mal

From: Dr Mal Hooper [mailto:general@spinalrehab.com.au]
Sent: Thursday, 28 August 2008 1:09 PM
To: 'Perri'
Subject: Embryonic Stem Cells - be aware!

Perri –

Internet search will bring up lots of info available about embryonic stem cells (ESC) which are from an aborted fetus – the ESC stem cells are immature and 'undifferentiated' this means they also have the ability to *act as a 'trigger' to other bad stem cells within the patient's body to potentially causes cancers and or cancer accelerating effects* – hence the term undifferentiated cancers.

Several questions you would want to ask before committing to India - what exact cells are used – human, animal or mix????!! What additional factors are added to the mix – human growth hormone (they often use to muscle up the product – the patient feels great but it does not restore function), neurotrophic factors – which ones??? Other drugs mixed??? What evidence do they have that their approach does NOT result in undifferentiated cancers??

The article attached is a simply but good straight forward read

<http://www.stemcellresearchformichigan.com/media/news/WebMD%204-20-07.pdf>

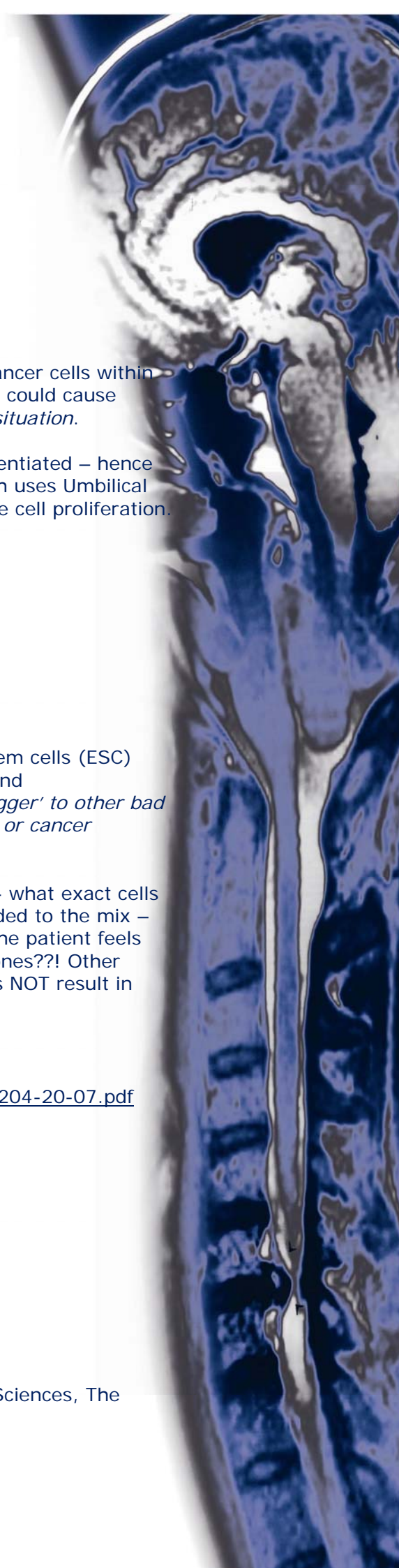
Mal

1: [Adv Cancer Res. 2008;100:133-58.](#)  [Links](#)

The tumorigenicity of human embryonic stem cells.

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Human embryonic stem cells (HESCs) are the in vitro descendants of the pluripotent inner cell mass (ICM) of human blastocyst stage embryos. HESCs can be kept undifferentiated in culture or be differentiated to tissues representing all three germ layers, both in vivo and in vitro. These properties make HESC-based therapy remarkably appealing for the treatment of various disorders.

Upon transplantation in vivo, undifferentiated HESCs rapidly generate the formation of large tumors called teratomas. These are benign masses of haphazardly differentiated tissues. Teratomas also appear spontaneously in humans and in mice. When they also encompass a core of malignant undifferentiated cells, these tumors are defined as teratocarcinomas. These malignant undifferentiated cells are termed embryonic carcinoma (EC), and are the malignant counterparts of embryonic stem cells.

Here we review the history of experimental teratomas and teratocarcinomas, from spontaneous teratocarcinomas in mice to induced teratomas by HESC transplantation. We then discuss cellular and molecular aspects of the tumorigenicity of HESCs. We also describe the utilization of HESC-induced teratomas for the modeling of early human embryogenesis and for modeling developmental diseases. **The problem of HESC-induced teratomas may also impede or prevent future HESC-based therapies.** We thus conclude with a survey of approaches to evade HESC-induced tumor formation.

PMID: 18620095 [PubMed - indexed for MEDLINE]



1: [Genes Chromosomes Cancer](#). 2008 Aug; 47(8):665-79. [Links](#)

Tumor progression of culture-adapted human embryonic stem cells during long-term culture.

Yang S, Lin G, Tan YQ, Zhou D, Deng LY, Cheng DH, Luo SW, Liu TC, Zhou XY, Sun Z, Xiang Y, Chen TJ, Wen JF, Lu GX.

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Human embryonic stem cells (hESCs) during long-term culture acquire chromosomal changes similar to those occurring in tumorigenesis. This was raised concerns about the progression from hESCs to malignant cells.

This study aimed to investigate the changes in chromosomes, cell phenotype, and genes in culture-adapted hESCs to ascertain whether tumorigenic transformation occurred. By cytogenetic analysis we found progressive karyotypic changes from simple to complex in chHES-3, one of the hESC lines established in our laboratory, during a long-term suboptimal culture. We further compared chHES-3 cells at different karyotypic stages in cell surface markers, in vivo differentiation, cell cycle, apoptosis, and gene expression profiles. We found that the karyotypically aberrant chHES-3 had higher S-phase fraction in cell cycle distributions and antiapoptosis ability. In vivo differentiation of karyotypically normal chHES-3 resulted in relatively mature teratoma, whereas karyotypically aberrant chHES-3 formed immature teratoma (grade III), in which more primary neural epithelium was revealed by pathological analysis. The microarray analysis and real-time PCR results showed that some oncogenes were upregulated in karyotypically aberrant chHES-3 cells, whereas the genes related to differentiation were downregulated, and that Wnt signal pathway was activated. In conclusion, chHES-3 cells underwent deregulation

1: [Nat Genet.](#) 2008 May; 40(5): 499-507.



[Links](#)

An embryonic stem cell-like gene expression signature in poorly differentiated aggressive human tumors.

[Ben-Porath I](#), [Thomson MW](#), [Carey VJ](#), [Ge R](#), [Bell GW](#), [Regev A](#), [Weinberg RA](#).

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Cancer cells possess traits reminiscent of those ascribed to normal stem cells. It is unclear, however, whether these phenotypic similarities reflect the activity of common molecular pathways. Here, we analyze the enrichment patterns of gene sets associated with embryonic stem (ES) cell identity in the expression profiles of various human tumor types. We find that histologically poorly differentiated tumors show preferential overexpression of genes normally enriched in ES cells, combined with preferential repression of Polycomb-regulated genes. Moreover, activation targets of Nanog, Oct4, Sox2 and c-Myc are more frequently overexpressed in poorly differentiated tumors than in well-differentiated tumors. In breast cancers, this ES-like signature is associated with high-grade estrogen receptor (ER)-negative tumors, often of the basal-like subtype, and with poor clinical outcome. The ES signature is also present in poorly differentiated glioblastomas and bladder carcinomas. We identify a subset of ES cell-associated transcription regulators that are highly expressed in poorly differentiated tumors. Our results reveal a previously unknown link between genes associated with ES cell identity and the histopathological traits of tumors and support the possibility that these genes contribute to stem cell-like phenotypes shown by many tumors.

1: [Cell Stem Cell.](#) 2008 Apr 10; 2(4): 297-9.



[Links](#)

Comment on:

[Cell Stem Cell.](#) 2008 Apr 10; 2(4): 333-44.

Cancer: inappropriate expression of stem cell programs?

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Cancer stem cells (CSCs) are a subpopulation of cancer cells that possess characteristics, including self-renewal, associated with normal stem cells. In this issue of Cell Stem Cell, Wong et al. (2008) define a core embryonic stem cell (ESC)-like gene expression program that may be important for CSC function in multiple epithelial cancers.