Cancer stem cells literatures

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Abstract: The definition of stem cell is "an unspecialized cell that gives rise to a specific specialized cell, such as a blood cell". Embryonic stem cells are derived from the inner cell mass of blastocyst stage embryos. Somatic stem cells differentiate into only the cells the specific tissue wherein they reside. Stem Cell is the original of life. All cells come from stem cells.
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1. Introduction

Stem cell is the origin of an orgnism's life. Stem cells have the potential to develop into many different types of cells in life bodies, to be many different tissues and organs. Stem cells can be used in the clinical medicine to treat patients with a variety of diseases (Daar, 2003). Serving as a repair system for the living body, the stem cells can divide without limit to replenish other cells as long as the living body is still alive. When a stem cell divides, each new cell has the potential to either remain a stem cell situation or become another type of cell with a more specialized function, such as a muscle cell, a red blood cell, a bone cell, a nerve cell, or a brain cell. Stem cell research is a typical and important topic of life science.

The definition of stem cell is "an unspecialized cell that gives rise to a specific specialized cell, such as a blood cell" (Stedman's Medical Dictionary, 2002).

Stem cell is totipotent, that means it holds all the genetic information of the living body and it can develop into a mature cell. Stem cell is a single cell that can give rise to progeny that differentiate into any of the specialized cells of embryonic or adult tissue. The ultimate stem cells (fertilized egg) divide to branches of cells that form various differentiated tissues or organs. During these early decisions, each progeny cell retains totipotency. Through divisions and differentiations the embryonic stem cells lose totipotency and gain differentiated function. During normal tissue renewal in adult organs, tissue stem cells give rise to progeny that differentiate into mature functioning cells of that tissue. Stem cells losing totipotentiality are progenitor cells. Except for germinal cells, which retain totipotency, most stem cells in adult tissues have reduced potential to produce different cells.

Aristotle (384-322 BC) deduced that the embryo was derived from mother's menstrual blood, which was based on the concept that living animals arose from slime or decaying matter. This concept was accepted in western world for over 2000 years, and it controlled western philosophy for over 2000 years either. In 1855, Virchow supposed that all cells in an organism are derived from preexisting cells. Now we know that all the human cells arise from a preexisting stem cell – the fertilized egg, that come from the mating of a man and a woman naturally but now can be produced in the laboratory tube. The counter hypothesis of spontaneous generation was accepted until 1864, when the French scientist Louis Pasteur demonstrated that there would be no microorganisms' growing after sterilizing and sealing.

The animal body has an unlimited source of stem cells, almost. However, the problem is not in locating these stem cells, but in isolating them from their tissue source.

Five key stem cells have been isolated from human: (1) Blastocysts; (2) Early embryos; (3) Fetal tissue; (4) Mature tissue; (5) Mature cells that can be grown into stem cells.

Up to today, only stem cells taken from adults or children (known generically as "adult stem cells") have been used extensively and effectively in the treatment of degenerative diseases.

Embryonic stem cells hold great promise for treating degenerative diseases, including diabetes, Parkinson's, Alzheimer's, neural degeneration, and cardiomyopathies (Bavister, 2005). Embryonic stem cells are derived from the inner cell mass of blastocyst stage embryos. Embryonic stem cells can replicate indefinitely. This makes it feasible to culture the cells on a large scaled for cell transplantation therapy in clinical application. Embryonic stem cells are pluripotent and have the potential to differentiate into all three germ layers of the mammalian body including the germ cells.

Normally to say that somatic stem cells differentiate only into specific tissue cells wherein they reside. However, somatic stem cells can differentiate into cells other than those of their tissue of origin. Adult bone marrow, fat, liver, skin, brain, skeletal muscle, pancreas, lung, heart and peripheral blood possess stem or progenitor cells with the capacity to transdifferentiate. Due to this developmental plasticity, somatic stem cells may have potential in autologous regenerative medicine, circumventing problems like rejection and the ethically challenged use of embryocyte stem cells.

Cancer stem cell (CSC) is cancer cell that has two facts: they exist in cancers (or tumors) and have the characteristics of stem cells. CSCs may generate tumors. Such cells could stay in tumor or cause rise to new tumors. To treat cancer stem cell is important in the clinical cancer biology, even CSCs form only a small proportion of the tumor. Chemotherapies kill differentiated or differentiating cells that form the bulk of the tumor, but are unable to generate new cells. A population of CSCs, which gave rise to it, could remain untouched and cause a relapse of the disease. Aldehyde dehydrogenase (ALDH) is expressed in liposarcoma patient body (Stratford, Castro et al.).

In different cancer subtypes, cells in the tumor perform the functional heterogeneity, and tumors are formed from various proliferative and differentiate cells. This functional heterogeneity in cancer cells has 2 characterizations normally: the CSC and clonal evolution models. The CSCs can self-renew and are able to generate the diverse tumor cells. The tumor population is hierarchically arranged with CSCs lying at the apex of the hierarchy. Cancer cells must be capable of continuous proliferation and self-renewal in order to retain the many mutations required for carcinogenesis.

The concept of a "cancer stem cell system" that continues to supply cancer component cells has been proposed. It is time to apply stem cell studies, which is a field of expertise in regenerative medicine, to cancer treatment. Cancer treatments that effectively attack cancer stem cells acting as a manufacturing plant for producing differentiated cancer progenies will be designed by revealing the cancer stem cell system (Kobayashi, Navarro-Alvarez et al. 2008).

There are questions on the cell of origin of CSCs - whether they originate from normal stem cells that have lost the ability to regulate proliferation, or from more differentiated population of progenitor cells that have acquired abilities to self-renew.

Cancer is a disease of genes. Inherited or somatic alterations in genes are what make a normal cell ignore growth-controlling signals and form a tumor that eventually leads to the destruction of the organism. Based on accumulated knowledge on the genetic composition of cancer cells, the clonal evolution model of tumorigenesis was established, which explains multiple aspects of human disease and clinical observations. All or most tumor cells can participate in tumor evolution and restricts this property to a subset of them defined as 'cancer stem cells' due to their stem cell-like characteristics. (Shipitsin and Polyak 2008).

In 1997, Bonnet and Dick isolated a subpopulation of leukaemic cells that expressed a specific surface marker CD34, but lacked the CD38 marker. Bonnet and Dick concluded that the $CD34^{+}/CD38^{-}$ subpopulation is capable of initiating tumors in NOD/SCID mice that are histologically similar to the donor (Bonnet D and Dick JE, 1997).

In cancer biology studies, tumor cells are sometimes injected into experimental animals to induce tumors. Cancer progression is followed in time and drug candidates are tested for their ability to inhibit the cancer growth. However, efficient cancer formation requires thousands of cells to be injected. It is possible that only a small fraction of the injected cells CSCs have the potential to generate cancer. Many cancers are very heterogeneous and contain multiple cell types native to the host organ. Heterogeneity is commonly retained by cancer metastases. The cell that produced cancer have the capacity to generate multiple cell types.

The cancer stem cell (CSC) hypothesis proposes that tumor growth is maintained by a distinct subpopulation of 'CSC'. Whilst cell lines are valuable in assay development, primary cells may provide a more rewarding model for studying tumor heterogeneity in the context of CSC (Blacking, Waterfall et al.).

CSC can be generated as: mutants of developing progenitor cells, developing stem cells, adult progenitor cells, or adult stem cells. One possibility is that the cancer stem cell is generated by a mutation of stem cell niche during cell development. The logical progression claims that these developing stems are mutated and then expand and the mutation is shared by many of the descendants of the mutated stem cells. These daughter stem cells are more easy to become into tumors. There are more chance of a mutation that can cause cancer.

Although uncontrolled proliferation is a distinguishing property of a tumor as a whole, the individual cells that make up the tumor exhibit considerable variation in many properties, including morphology, proliferation kinetics, and the ability to initiate tumor growth in transplant assays. Understanding the molecular and cellular basis of this

heterogeneity has important implications in the design of therapeutic strategies. The mechanistic basis of tumor heterogeneity has been uncertain; however, there is now strong evidence that cancer is a cellular hierarchy with cancer stem cells at the apex (Dick 2008).

The cancer is a heterogeneous population of mutant cells. The tumor is made up of several types of stem cells. Some cells can become more adaption to certain environments including adaptation to cancer treatment, so that more severe for the patients.

While the great majority of cells that make up tumors are destined to differentiate, albeit aberrantly, and eventually stop dividing, only a minority population of cells, termed cancer stem cells, possess extensive self-renewal capability and can recapitulate tumor pathophysiology in an immune-compromised animal model. Tumor-initiating cells have been identified and isolated in a variety of cancers of the blood, breast, central nervous system, pancreas, skin, head and neck, colon, and prostate. (Tang, Ang et al. 2007).

CSCs are found in most human cancers. For the CSC isolation for the researches and diagnosis, fluorescence-activated cell sorting (FACS) with antibodies directed at cell-surface markers and functional approaches including SP analysis (side population assay) or Aldefluor assay can be considered. The CSC-enriched population purified by these approaches is then implanted, at various cell doses. This in vivo assay is called limiting dilution assay. The cancer cell subsets that can initiate cancer development at low cell numbers are further tested for self-renewal capacity in serial cancer capacity. CSC can be identified by efflux of incorporated Hoechst dyes via multidrug resistance (MDR) and ATP-binding cassette (ABC) Transporters. Another method used for identification of cell subset enriched with in CSCs in vitro is sphere-forming assays. Many normal stem cells such as hematopoietics or stem cells from tissues are capable, under special culture conditioned, to form three-dimensional spheres, which can differentiate into multiple cell types. Similarly as normal stem cells, the CSCs isolated from brain or prostate tumors has also ability to form anchorage-independent spheres (Nicolis, 2007).

Many cell surface markers have been used for isolation of subsets enriched CSC, such as CD133 (i.e. PROM1), CD44, CD24, EpCAM (epithelial cell adhesion molecule, i.e. epithelial specific antigen, ESA), THY1 and ATP-binding cassette B5 (ABCB5), etc.

CD133 (prominin 1) is a five-transmembrane domain glycoprotein expressed on $CD34^+$ stem and progenitor cells, in endothelial precursors and fetal neural stem cells. It has been detected using its glycosylated epitope know as AC133.

EpCAM (epithelial cell adhesion molecule, ESA, TROP1) is hemophilic CA^{2+}-independent cell adhesion molecule expressed on the basolateral surface of most epithelial cells.

CD90 (THY1) is a glycosylphosphatidylinositol glycoprotein anchored in the plasma membrane and involved in signal transduction. It may also mediate adhesion between thymocytes and thymic stroma.

CD44 (PGP1) is an adhesion molecule that has pleiotropic roles in cell signaling, migration and homing. It has multiple isoforms, including CD44H, which exhibits high affinity for hyaluronate, and CD44V which has metastatic properties.

CD24 (HSA) is a glycosylated glycosylphosphatidylinositol-anchored adhesion molecule, which has co-stimulatory role in B and T cells.

ALDH is a ubiquitous aldehyde dehydrogenase family of enzymes, which catalyzes the oxidation of aromatic aldehydes to carboxyl acids. For instance, it has role in conversion of retinol to retinoic acid, which is essential for survival.

The first isolated and identified CSC was from breast cancer. Breast CSCs have been enriched in $CD44^+CD24^{-/low}$, SP, $ALDH^+$. Breast CSCs are very phenotypically diverse. Both $CD44^+CD24^-$ and $CD44^+CD24^+$ cell populations are tumor initiating cells. CSC are most highly enriched using the $CD44^+CD49f^{hi}CD133/2^{hi}$ as marker. Stem-like cancer cells have been identified using cell surface markers including CD44, CD133, EGFR and SSEA-1 (stage-specific embryonic antigen-1).

CSCs have also been found in human colon cancer. For their identification, cell surface markers such as ABCB5, CD44, CD133 were used. Multiple CSCs have been fould in prostate, lung and many other organs, including liver, pancreas, kidney or ovary. In prostate cancer, the tumor-initiating cells have been identified in $CD44^+$ cell subset as $CD44^+\alpha2\beta1^+$, TRA-1-$60^+CD151^+CD166^+$ or $ALDH^+$ cell populations.

Drug resistance of cancer stem/initiating cells has been considered to be one of the main reasons for tumor relapse. The breast cancer cell line MDA-MB-468 was cultured with 5-fluorouracil and serially passaged. The drug resistance of cancer cells is mainly due to tumor stem/initiating cells, and that under conditions of persistent chemotherapy, the quantity or function of breast cancer stem/initiating cells increases and decreases alternately.(Lu, Deng et al.)

The existence of CSCs has several implications in terms of future cancer treatment and therapies. These include disease identification, selective drug

targets, prevention of metastasis, and development of new intervention strategies.

The epithelial-mesenchymal transition plays a crucial role in the progression of pancreatic cancer. Pancreatic cancer stem-like cells exhibit greater invasion and migration activity in vitro compared to the CD44(-)CD24(-) cells. A direct link between epithelial-mesenchymal transition and cancer stem-like cells in pancreatic cancer (Wang, Wu et al.).

Normal somatic stem cells are naturally resistant to chemotherapeutic agents. CSCs that developed from normal stem cells may also produced these proteins that could increase their resistance towards chemotherapeutic agents. The cell surface receptor interleukin-3 receptor-alpha (CD123) was found to be overexpressed on CD34+CD38- leukemic stem cells (LSCs) in acute myelogenous leukemia (AML) but not on normal CD34+CD38- bone marrow cells.

Although uncontrolled proliferation is a distinguishing property of a tumor as a whole, the individual cells that make up the tumor exhibit considerable variation in many properties, including morphology, proliferation kinetics, and the ability to initiate tumor growth in transplant assays. Understanding the molecular and cellular basis of this heterogeneity has important implications in the design of therapeutic strategies. The mechanistic basis of tumor heterogeneity has been uncertain; however, there is now strong evidence that cancer is a cellular hierarchy with cancer stem cells at the apex (Dick 2008).

The design of new drugs for the treatment of CSCs will likely require an understanding of the cellular mechanisms that regulate cell proliferation. A normal stem cell may be transformed into a cancer stem cell through disregulation of the proliferation and differentiation pathways controlling it or by inducing oncoprotein activity.

Cancer has long been viewed as a heterogeneous population of cells. While the great majority of cells that make up tumors are destined to differentiate, albeit aberrantly, and eventually stop dividing, only a minority population of cells, termed cancer stem cells, possess extensive self-renewal capability and can recapitulate tumor pathophysiology in an immune-compromised animal model. Tumor-initiating cells have been identified and isolated in a variety of cancers of the blood, breast, central nervous system, pancreas, skin, head and neck, colon, and prostate. In this review we present scientific evidence supporting the cancer stem cell model of tumor progression, and discuss the experimental and therapeutic implications. The concept of cancer stem cells may have profound implications for our understanding of tumor biology and for the design of novel treatments targeted toward these cells. Current therapeutic strategies include targeting the cancer stem cell as well as its microenvironmental niche. We present an interesting, novel strategy that takes into account the reactive oxygen species status in cancer stem cells and how it might serve as a method for eradicating these cells in tumor growth (Tang, Ang et al. 2007).

The Polycomb group transcriptional repressor Bmi-1 was discovered as a common oncogene activated in lymphoma and later shown to specifically regulate HSCs. The role of Bmi-1 has also been illustrated in neural stem cells. The pathway appears to be active in CSCs of pediatric brain tumors.

The existence of a stem cell niche, or physiological microenvironment, consisting of specialized cells that directly and indirectly participate in stem cell regulation has been verified for mammalian adult stem cells in the intestinal, neural, epidermal, and hematopoietic systems. In light of these findings, it has been proposed that a "cancer stem cell niche" also exists and that interactions with this tumor niche may specify a self-renewing population of tumor cells (Sneddon and Werb 2007).

The role of the Notch pathway in control of stem cell proliferation has been demonstrated for several cell types including hematopoietic, neural and mammary stem cells. Components of the Notch pathway have been proposed to act as oncogenes in mammary. A particular branch of the Notch signaling pathway that involves the transcription factor Hes3 has been shown to regulate a number of cultured cells with cancer stem cell characteristics obtained from glioblastoma patients.

There is increasing evidence suggesting that stem cells are susceptive to carcinogenesis and, consequently, can be the origin of many cancers. Recently, the neoplastic potential of stem cells has been supported by many groups showing the existence of subpopulations with stem cell characteristics in tumor biopsies such as brain and breast. Evidence supporting the cancer stem cell hypothesis has gained impact due to progress in stem cell biology and development of new models to validate the self-renewal potential of stem cells. Recent evidence on the possible identification of cancer stem cells may offer an opportunity to use these cells as future therapeutic targets. Therefore, model systems in this field have become very important and useful. This review will focus on the state of knowledge on cancer stem cell research, including cell line models for cancer stem cells (Serakinci and Erzik 2007).

Sonic hedgehog (SHH) and Wnt pathways are commonly hyperactivated in cancer and are required

to sustain cancer growth. The Gli transcription factors that are regulated by SHH take their name from gliomas, where they are commonly expressed at high levels. A degree of crosstalk exists between the two pathways. Metastasis is the most serious problem and lethality for the cancer patients.

Wang el at showed that a known regulator of prostate epithelial differentiation, the homeobox gene Nkx3-1, marks a stem cell population that functions during prostate regeneration. Genetic lineage-marking demonstrates that rare luminal cells that express Nkx3-1 in the absence of testicular androgens (castration-resistant Nkx3-1-expressing cells, CARNs) are bipotential and can self-renew in vivo, and single-cell transplantation assays show that CARNs can reconstitute prostate ducts in renal grafts. Functional assays of Nkx3-1 mutant mice in serial prostate regeneration suggest that Nkx3-1 is required for stem cell maintenance. These observations indicate that CARNs represent a new luminal stem cell population that is an efficient target for oncogenic transformation in prostate cancer (Wang, Kruithof-de Julio et al. 2009).

Mesenchymal stem cells (MSCs) have an inhibitory effect on tumor proliferation, but the precise mechanisms are not fully understood. Zhu and colleagues identified DKK-1 (dickkopf-1), secreted by MSCs and acting as a negative regulator of WNT signaling pathway, to be one of the molecules responsible for the inhibitory effect. When DKK-1 was neutralized by anti-DKK-1 antibodies, or when the expression of DKK-1 was downregulated by RNA interference (RNAi), the inhibitory effects of MSCs on K562 cell proliferation were attenuated. This group provides evidence that the expression of DKK-1 by MSCs is regulated by NANOG, a transcriptional factor ubiquitously expressed in some stem cells. Using the Cellmax artificial capillary modules that eliminate the immunosuppressive properties of MSCs, they further showed that MSCs were able to inhibit proliferation of K562 cells in a humoral microenvironment. MSCs probably have a general inhibitory effect on their neighboring cells, including malignant cells, en route to achieving tissue homeostasis (Zhu, Sun et al. 2009).

Enrichment of cancer stem cells for studies of carcinogenesis remains a difficult issue. The unique features of cancer stem cells (CSCs) may allow formation of their colonies in vitro with distinct morphology. Zhou et al investigated the possibility to use morphological diversity of colonies to identify and enrich CSCs from cultured malignant human glioma cells. They found that a small proportion of the cells from a human glioma cell line U251 formed tight and round-shaped colonies in culture. Most cells in such colonies were capable of self-renewal, generating tumor spheres and differentiating into lineages with markers for neurons, astrocytes and oligodendrocytes. In addition, several neural stem cell-related genes were highly expressed by tumor cells in those tight colonies. Their research results demonstrate a novel approach to the identification and enrichment of CSCs based on unique morphology of their colonies formed in vitro (Zhou, Ping et al. 2009).

Mammary stem cells are bipotential and suggested to be the origin of breast cancer development, but are elusive and vaguely characterized. Breast tumors can be divided into subgroups, each one requiring specific treatment. To determine a possible association between mammary stem cells and breast cancer, a detailed characterization of the transcriptome in mammary stem cells is essential. Williams et al used a murine mammary epithelial stem-like cell line (HC11) and made a thorough investigation of global gene-expression changes during stepwise differentiation using dual-color comparative microarray technique. Subsequently, they run a cross-species comparison to reveal conserved gene expression between stem cells and subtype-specific and prognosis gene signatures, and correlated gene expression to in vivo mammary gland development. Their analysis of mammary stem-like and stepwise cell differentiation, and an in-depth description of their findings in a breast cancer perspective provided a useful map of the transcriptomic changes and a number of novel mammary stem cell markers. The transcriptional characterization of these mammary stem-like cells and their differentiation-induced gene expression patterns is here made widely accessible and provides a basis for research on mammary stem-like cells. Some tumors are more stem-like than others, with a corresponding worse prognosis. This information would, if established, be important for treatment decisions. (Williams, Helguero et al. 2009).

Both hereditary and sporadic breast cancers may develop through dysregulation of self-renewal pathways of normal mammary stem cells. Networks of proto-oncogenes and tumor suppressors that control cancer cell proliferation also regulate stem cell self-renewal and possibly stem cell aging. Breast cancer susceptibility gene (BRCA1) is a nuclear phosphoprotein expressed in many nuclear processes, including stem cell regulator, DNA damage repair, recombination, transcription, ubiquitination, cell cycle checkpoint enforcement, and centrosome regulation. Rassi reported that BRCA1, EGFR, hedgehog, Wnt/beta-catenin, and/or Notch pathways were frequently upregulated in cancer progenitor cells during the initiation and development of breast cancer (Rassi 2009).

Cancer stem cell research is a focus for more and more cancer biologists and evidence of involvement in cancer development is becoming more abundant. Earlier studies indicated cancer stem cells to be rare as determined by the standard xenotransplantation assay using SCID mice in vivo. To estimate frequency correctly, it is necessary to considerate cancer stem cell subsets with differing capacities for tumorigenesis (Liu and Zhang 2009).
Kitamura, H., K. Okudela, et al. (2009). "Cancer stem cell: implications in cancer biology and therapy with special reference to lung cancer." Lung Cancer **66**(3): 275-81.

CSC theory is currently central to the field of cancer research, because it is not only a matter of academic interest but also crucial in cancer therapy. CSCs share a variety of biological properties with normal somatic stem cells in terms of self-renewal, the propagation of differentiated progeny, the expression of specific cell markers and stem cell genes, and the utilization of common signaling pathways and the stem cell niche. However, CSCs differ from normal stem cells in their tumorigenic activity. Thus, CSCs are also termed cancer initiating cells. CSCs of lung cancers, will lead to progress in therapy, intervention, and improvement of the prognosis of patients with lung cancer. (Kitamura, Okudela et al. 2009).

Cancer stem cells (CSCs) are thought to sustain cancer progression, metastasis and recurrence after therapy. There is in vitro and in vivo evidence supporting the idea that CSCs are highly chemoresistant. Epigenetic gene regulation is crucial for both stem cell biology and chemoresistance (Crea, Danesi et al. 2009).

The CSC compartment represents the subpopulation of tumor cells with clonogenic potential and the ability to initiate new tumors. Besides self renewal, one of their main features is their ability to differentiate into the variety of cells within the tumor. (Borovski, Vermeulen et al. 2009).

The concept of "field cancerization" describes the presence of histological abnormal tissue surrounding oral squamous cell carcinoma (OSCC). Molecular model of multistep carcinogenesis indicates that an accumulation of genetic alterations forms the basis for the OSCC progression with genetic heterogeneity (Zhou and Jiang 2008).

Tissue stem cells are responsible for replenishing and maintaining a population of cells which make up a functioning organ. They divide by asymmetric cell division where one daughter remains a stem cell while the other daughter becomes a transit cell, which divides a defined number of times and differentiates. A fully differentiated cell has a finite life-span. A tissue can be maintained by various strategies. Stem cells can divide often and differentiated cells die often (fast turnover). Alternatively, stem cells can divide infrequently, and the differentiated cells are long lived (slow turnover). Genetic alterations and mutations can interfere with tissue homoeostasis. Mutations can induce senescence and apoptosis, and this can result in a reduction of the number of functioning tissue cells which could correlate with tissue aging. Alternatively, mutations can result in the carcinogenic transformation of cells and the formation of a tumor (Wodarz 2007).

The cancer stem cell hypothesis posits that tumor growth is driven by a rare subpopulation of cells, designated CSC. Studies supporting this theory are based in large part on xenotransplantation experiments wherein human cancer cells are grown in immunocompromised mice and only CSC, often constituting less than 1% of the malignancy, generate tumors. Yoo et al showed that all colonies derived from randomly chosen single cells in mouse lung and breast cancer cell lines form tumors following allografting histocompatible mice. They suggested that the majority of malignant cells rather than CSC can sustain tumors and that the cancer stem cell theory must be reevaluated (Yoo and Hatfield 2008).

Application of Stem Cells in Clinical Medicine

There are over four thousand registered diseases specifically linked to genetic abnormalities. Although stem cells are unlikely to provide powerful treatment for these diseases, they are unique in their potential application to these diseases.

Indeed, in many research projects, scientists have demonstrated that stem cells can be used to replenish or rejuvenate damaged cells within the immune system of the human body and that damaged stem cells can repair themselves and their neighbors. For example, in what is regarded as the first documented case of successful gene-therapy "surgery", scientists at the Necker Hospital for Sick Children in Paris of French succeeded in treating two infants diagnosed with Severe Combined Immunodeficiency Disease, a life-threatening degenerative disease caused by defects on the male (X) chromosome. With the identification of stem cell plasticity several years ago, multiple reports raised hopes that tissue repair by stem cell transplantation could be within reach in the near future (Kashofer, 2005). In cardiovascular medicine, the possibility to cure heart failure with newly generated cardiomyocytes has created the interest of many researchers (Condorelli, 2005). Gene clone techniques can be widely used in the stem cell researches and applications (Ma, 2004).

Debates on Stem Cell Research

There are a lot of debates on the stem cell research. Stem cell research is a high-tech question and the people involved in this rebates should have certain scientific knowledge on the stem cell. It is OK for the politicians or religionists to show their opinions on any topic they are interested in, but not suitable for them to make decisions (or make laws) that will significantly influence the scientific research as this field the politicians or religionists are not specialized. Such as, it is not suitable for the American President George W. Bush to show the power in the stem cell research. It is scientists' job. When politics and science collide, science should do scientific way, rather political way. Major ethical and scientific debates surround the potential of stem cells to radically alter therapies in health care (Williams, 2005).

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