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Healing Cells: Use What the Almighty Created to Heal Thyself

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Abstract

The adult human body is composed of trillions and trillions of cells. These cells can be divided into three categories: functional [parenchyma and stroma] cells comprising 50%, maintenance [progenitor] cells comprising 40%, and healing [stem] cells comprising 10% of the cells of the body. Healing cells were discovered in 1975 residing within the connective tissue stroma of adult terrestrial salamanders undergoing complete limb regeneration. Healing cells were shown to form all damaged or lost tissues of an amputated limb, thereby restoring function to the limb. Since 1975, healing cells have been extensively characterized, four animal models generated, and 32 human clinical studies undertaken to test and validate healing cells for their ability to reverse signs and symptoms of traumatic injuries, chronic (terminal) diseases, and orthopedic disorders. Two methodologies were developed to activate the healing cells. Ex vivo activation occurred after healing cells were removed from the body, activated, and then returned to the body in selected locations. In vivo activation occurred by ingestion of a nutraceutical to activate the normally quiescent healing cells in situ. Clinical studies proved that both methodologies were 100% safe for human use. Fresh isolates have demonstrated 83% efficacy and the nutraceutical 100% efficacy at reversing signs and symptoms of traumatic injuries, chronic diseases, and disorders, with minimal to no side effects. Stem cells are considered the "holy grail" for regenerative medicine. We propose that adult healing cells be used in regenerative medicine.

Keywords: adult; chronic diseases; telomerase; pluripotent; severe trauma

INTRODUCTION

The human body is composed of trillions and trillions of cells. These cells can be divided into three categories: functional cells, maintenance cells, and healing cells (1).

are absent the telomerase enzyme(2), whose func-form every cell type of the body (7). tion is to renew telomeres at the ends of the chromosenesce and die when they become worn out (1-3).

the individual (1).

Healing (stem) cells comprise 10% of all cells of (14-18). Johnston suggested "know your model systhe body. They are the "true" stem cells and are tem" and "tissue never lies".

Functional (differentiated) cells comprise 50% of lomerase enzyme after birth, such as the adult teall cells of the body. Functional cells are composed lomerase positive stem cells(7). While low in numof parenchyma and stroma and are represented by ber, healing cells can be activated to proliferate pri-220+ distinct differentiated cell types (1). Examples or to forming maintenance cells. By retaining the of parenchyma are signal transmitting neurons, gas telomerase enzyme, they have an unlimited proliferexchanging lung cells, and cardiac muscle cells that ation potential, and therefore unlimited lifespan if pump blood throughout the body. Examples of stro- they remain in their respective naïve state(3-7). ma are the fibrocytes forming the connective tissue Healing cells remain constant in number throughout structural framework of the body. Functional cells the life of the individual from birth to death and will

somes after each cell division. Hence, functional Discovery of healing cells occurred in 1975 while cells have a finite lifespan [70 population doublings studying limb regeneration in the adult terrestrial in humans $(3-6)$] before they are pre-programmed to salamander, Ambystoma annulatum (7). Previous **Maintenance (progenitor) cells** comprise 40% of limb within a time frame of 30-45 days (8-12). all the cells of the body and are the immediate re-However, the significantly larger adult terrestrial placement cells for the functional cells (1). Exam-form had lost the ability to regenerate an appendage ples of maintenance cells are neuroblasts, lung alve-[limb or tail] (13). I wanted to know why there was olar-blasts, cardiac myoblasts, fibroblasts, and mes-this discrepancy between aquatic and terrestrial verenchymal progenitor (stem) cells (1). Like function- sions of the same species, Ambystoma. My first al cells, maintenance cells have lost the telomerase attempt failed miserably because the salamanders enzyme and therefore have a finite lifespan of 70 became emaciated and died before I even began my population doublings before they are pre-experiments. My graduate school mentor [Dr. P.M. programmed to senesce and die (2-6). Progenitor Johnston] asked "what might have happened to cells decrease in number with the increasing age of cause them to die". I said I was using adult terrestristudies reported that juvenile aquatic versions of this species were quite capable of regenerating a al salamanders, but following the methods reported in the scientific literature for the aquatic version. Dr.

ubiquitously located throughout all connective tis-I began my research by understanding the model sue stroma in the body. The pre-programmed func-system that I was using. The studies claiming that tion of healing cells is to repair and restore all dam-the terrestrial version would not regenerate a limb aged or missing cells of the body (7). Examples of were keeping juvenile salamanders in an aquatic healing cells are cells in the adult that retain the te- environment at 4°C, feeding them beef liver during the day, and assaying morphological changes at 5- perichondrium, epineurium, perineurium, and endoment, are nocturnal animals, sleeping during the appeared proximal to and at the wound site, debfood sources are nightcrawlers followed by cock-the amputation sites so only healthy tissue re-"apparently happy" for the limb regeneration stud-area and form a mass of undifferentiated cells. Ocof regenerating an appendage, but that they required off the wound (7,20). A ridge within the distal epicomplete success. I repeated the study in four spe-limb (21). The base of the epidermal ridge, just discies of terrestrial salamanders, e.g., Ambystoma: tal to the mass of undifferentiated healing cells, bemaculatum, texanum, tigranum, and annulatum to gan secreting glycoproteins [periodic-Schiffvalidate the results. All species demonstrated the positive acellular material] into the mass of undif-370 days post amputation (20). The original studies began to proliferate outward in a unidirectional disthe regeneration process, for a total of 6-9 time mass of cells occurred between intact tissues proxipoints, depending on the species (16,17). I used a mal to the amputation site and the ridge of epider-31-74 time points, dependent on species, for assess-tissues began. There was a continuum between in-

day time points for 30-45 days (14-18). I discovered neurium, epimysium, perimysium, endomysium, that when terrestrial adult salamanders are placed and tunica adventitia of blood vessels (7,20). When into an aquatic environment at $4^{\circ}C$ [similar condi- cellular damage (limb amputation) occurred, the tions for their breeding cycle] they would not eat, healing cells became activated, were visible as rather they have copulation/procreation on their unique cell types, and were seen migrating towards minds, and will starve themselves to death (19). the site of damage [distal end of the transected The terrestrial salamanders, in a terrestrial environ-limb] (7,20). During this same time, macrophages day and foraging for food at night. Their preferred rided, and removed all the dead and dying tissue at roaches. When their conditions were changed to mained. Once the amputation site was completely their preferred environment, they became fat, and debrided, the healing cells would migrate into the ies to commence (19). My studies demonstrated curring parallel to debridement and migration of that adult terrestrial salamanders were quite capable stem cells, the epidermis along the periphery closed an extended amount of time [up to 370 days] for dermis formed at the distal tip of the transected ability to completely regenerate a limb from 155- ferentiated cells (22). These undifferentiated cells with the aquatic juvenile salamander used five-day tal direction towards the epidermis at the distal tip intervals to assess changes in morphology during of the limb. Once elongation of the undifferentiated similar five-day period for 1+ years, amounting to mis distal to the amputation site, formation of new ment of their morphology. That is when the discov-tact functional cells [proximal to the wound site], ery was made of the adult telomerase positive heal-newly formed functional cells [at the wound site], ing cells, 1975 (20). The normally hibernating qui-to newly forming progenitor cells [just distal to escent (and invisible) healing cells were located wound site], to the undifferentiated mass of cells throughout all the structural connective tissue further distal to the wound site, and finally to the frameworks of the limb, e.g., dermis, periosteum, base of the epidermis. This progression of cell types

cells, thus restoring function to the individual (23- stem cells are derived from embryonic tissue (60 conditions in which they were kept as previously proliferation potential until they begin differentiamander itself (7,19,20,28).

Clones of healing cells, derived by repetitive single will spontaneously differentiate into a mass of canattributes, e.g., size, cell surface markers, and ex-either ESCs or iPSCs are pre-committed to a specifpressed genes; growth characteristics in cell culture, ic cell lineage will they not spontaneously differentie.g., nutrition requirements, freeze-thaw, individual ate, either in cell culture or implanted into an indilifespans; differentiation potential using functional vidual (67). cell expression markers; reactivity to inductive faction; presence throughout the life span of the indi-bone marrow, adipose tissue, umbilical cords, pla-Crane), mice, rats, rabbits, cats, dogs, sheep, goats, adults and therefore have not been readily used out-

Why is this important to you? There are several

pigs, cows, horses, and humans (29-59).

categories of cells used routinely in the field of re-My research group has been studying adult healing generative medicine to replace damaged cells and cells since their initial discovery in 1975 restore function, e.g., embryonic stem cells [ESCs], (7,20,28,29). We discovered that adult healing cells

eventually replaced all the missing cells of the limb, the induced pluripotent stem cells [iPSCs], and mesfunctional cells, maintenance cells, and healing enchymal stem cells [MSCs] (60-72). Embryonic 28). This observational study proved that adult ter-62), while induced pluripotent stem cells (iPSCs) restrial salamanders can completely regenerate a are derived from adult functional cells after insertion limb, although requiring a longer amount of time. of the Yamanaka factors [Oct-3/4, SOX-2, c-Myc, The actual cause of inability to regenerate a limb in and Klf4] (63,67). Both ESCs and iPSCs contain the terrestrial salamanders was due to the environmental enzyme telomerase, thus they have an unlimited reported, rather than something inherent to the sala-tion (64). When ESCs and iPSCs are grown in cell Characterization of adult healing cells was then will spontaneously form every cell type of the body undertaken to understand the potential of these heal-in an unregulated manner (66). When naïve ESCs or ing cells to affect a positive repair response (29). iPSCS are implanted into an animal or human, they cell clonogenic analysis, were examined for unique cerous cells, known as a teratoma (66). Only when culture in the absence of an external inhibitor, such as leukemia inhibitory factor [LIF] (62,65,66), they

tors; reactivity to tissue specific exosomes, progres-The adult mesenchymal stem cell (68) is a tripotent sion agents, inhibitory agents; physiological func-maintenance cell (69) that has been isolated from vidual; and location in multiple organs of 15 species centa, dental pulp, etc.(68-72). Clones of MSCs of animals including humans, e.g., amphibians have shown a capacity to form only three cell types, (adult terrestrial salamanders), reptile (Komodo e.g., fat, cartilage, and bone (29,69). Healing cells Dragon), avians (Gallus domesticatus, Wedel have been widely reported by others NOT to exist in side my research group for studies in the field of regenerative medicine.

form all the cells of the body (29,33,60,61). Since histoarchitecture for regain of function. ESCs, iPSCs, and healing cells have the telomerase (2,7,29,36) and naïve cells transplanted into animals rats. Two weeks after injection of 6-OHDA, the in-This contrasts with ESCs and iPSCs that spontane-1B). The animals were then stereotactically reinjectously form all body cell types when cultured and ed with either sterile saline (Fig. 1C) or the pluripoimplanted into an animal or human in their naïve area void of tyrosine hydroxylase activity due to prestate (62,64-67).

hypothesis of whether healing cells have a positive activity, an indicator of dopamine synthesis and sechronic diseases in adult rats were developed, e.g., a glial scar along the needle tract for the sterile saendoderm lineage] (4,31,54,57,58). These three along the needle tract with adjacent formation of tywould reverse the signs and symptoms in models of cells can restore missing or damaged tissues and rechronic disease. A Lac-Z genomically labeled naïve turn function to the individual with Parkinson's dishealing cell clone, Scl-40β (31), with demonstrated ease. pluripotent capabilities, was utilized to tract the implanted healing cells in vivo in all three animal models to evaluate two issues. First, whether a pluripotent healing cell could form cells from ectodermal, mesodermal, and endodermal lineages, proving beyond a shadow of a doubt that pluripotent stem cells

are similar yet different from ESCs and adult iPSCs. do exist in adult animals. And second, whether heal-ESCs, iPSCs, and healing cells share the ability to ing cells would heal damaged tissues in the correct

enzyme, they also have an unlimited lifespan when A **Parkinson's model** (55,73,74) [approved by the kept in their naïve state (3-6,33,36,60,61). However, IACUC] was created by stereotactically injecting of in the 48+ years that I have been studying adult the neurotoxin, 6-hydroxydopamine [6-OHDA], into healing cells (both outside the body in cell culture the substantia nigra of the ventral midbrain of adult and humans (31,36,54-59,73-76,78-90) adult heal-jected region was void of dopaminergic neurons and ing cells have never formed cancers of any kind. their associated neural networks (Fig. 1A and Fig. routinely form cancerous tissue (teratomas) when tent healing cell clone, Scl-40β (Fig. 1D), into an Animal models of chronic diseases. To answer the processed histochemically for tyrosine hydroxylase impact in chronic diseases, three animal models of cretion. Sterile saline injected animals demonstrated Parkinson's Disease [neural, ectoderm lineage] line injection (Fig. 1C). Animals injected with the (55,73,74), Myocardial Infarction [heart, mesoderm pluripotent healing cell clone, Scl-40β, demonstrated lineage] (4,31,53,56), and Pulmonary Fibrosis [lung, formation of tyrosine hydroxylase-positive cells models were used to test, validate, and potentially rosine hydroxylase-positive neural networks (Fig. demonstrate that implanted naïve adult healing cells 1D). This data suggests the potential that healing vious injection of neurotoxin. Animals were monitored for an additional six weeks, euthanized, and

> Figure 1. Adult rat model of Parkinson's disease created by stereotactically injecting a dopaminergic neurotoxin, 6-hydroxydopamine (6-OHDA), into substantia nigra pars compacta of the adult rat ventral midbrain.

Figure 1A, Control section two weeks after injection of saline only. Note dark brown reaction product indicating tyrosine hydroxylase activity in area of midbrain, indicating presence of dopaminergic neurons and their associated dopaminergic neural networks.

Figure 1B, Experimental section two weeks after injection 6-OHDA, note loss of tyrosine hydroxylase staining at injection site, indicating loss of both dopaminergic neurons and their associated neural networks.

Figure 1C. Adult rat model of Parkinson's Disease injected stereotactically with neurotoxin 6-OHDA to create zone devoid of tyrosine hydroxylase activity (staining) indicative of lost dopaminergic neurons and disintegration of neural networks. Section depicts experimental animal (Fig. 1C) six weeks after injection with sterile saline into area void of tyrosine hydroxylase activity. Sections stained histochemically for tyrosine hydroxylase activity and counterstained with methyl green to denote host cells (neurons and glial cells). Note line of green-stained glial cells in needle track, indicating a glial scar, along with disintegrating dopaminergic neural networks.

Figure 1D, Adult rat model of Parkinson's Disease injected stereotactically with neurotoxin 6-OHDA to create zone devoid of tyrosine hydroxylase activity (staining) indicative of lost dopaminergic neurons and disintegration of neural networks. Section depicts experimental animal (Fig. 1D) six weeks after injection with genomically-labeled naïve pluripotent aTPSC clone, Scl-40β into area void of tyrosine hydroxylase activity. Section stained histochemically for tyrosine hydroxylase activity (brown) and counterstained with methyl green to denote host cells (neurons and glial cells). Note tyrosine hydroxylase-positive cells within needle track of experimental animal and development of dopaminergic neural networks along all sides of the tyrosine hydroxylase-positive cells. Reprinted with permission from Y oung et al., A dult-denervated stem cells and their potential for tissue repair and molecular medicine. J Cell Molec Med. 2005; 9:753- 769 (55).

A myocardial infarction model (7,31,41,56) [approved by the IACUC] was created by two methods. The first method was to freeze the apex of the heart with liquid nitrogen. The second method utilized transient ligation of the left coronary descending coronary artery. A genomically labeled pluripotent healing cell clone, Scl-40β (31), was either injected directly into the damaged heart muscle [liquid nitrogen model] or delivered systemically by tail vein infusion [transient ligation model], to determine the effectiveness of healing cells to affect a positive response in heart muscle and coronary arteries after damage. Besides repairing damaged heart muscle (Fig. 2), Scl-40β was also involved in repairing damaged blood vessels (Fig. 2B, Fig. 3) and damaged connective tissue of the cardiac skeleton, that forms the structural framework of the heart (Fig. 2D) as shown by the green label residing in the cytoplasm of these respective regenerating cells.

Figure 2. Adult rat myocardial infarction model created by freezing apex of left ventricle with liquid nitrogen. A genomically labeled naïve pluripotent healing cell clone, Scl-40β, was then injected directly into the damaged heart muscle.

A, Genomically labeled naïve pluripotent healing cell clone, Scl-40b, in culture. In the undifferentiated state, the genomic label (green) is in the nucleus of the cell. With differentiation the genomic label (green) relocates to the cytoplasm of the cell (B,C,D).

B, Pluripotent healing cell clone, Scl-40β, was involved in the repair of damaged vasculature. Note the presence of the genomic label in the cytoplasm of regenerating blood vessels.

C, Pluripotent healing cell clone, Scl-40β, was involved in the repair of damaged cardiac muscle. Note the presence of the genomic label in the cytoplasm of cardiac myocytes.

D, Pluripotent healing cell clone, Scl-40β, was involved in the repair of damaged cardiac connective tissue skeleton. Note presence of the genomic label in the cytoplasm of fibrocytes of the connective tissue skeleton of the heart. Reprinted with permission from Young et al. Clonogenic analysis reveals reserve stem cells in postnatal mammals. II. Pluripotent epiblastic-like stem cells. Anat Rec. 2004; 277A:178-203 (31).

Figure 3. Systemic infusion of genomically labeled pluripotent healing cell clone, Scl-40β, into the tail vein of an adult rat after transient ligation of the left anterior descending coronary artery. Note repair of damaged coronary artery. Phycoerythrin (red) labeled healing cells located in all three connective tissue layers of the coronary blood vessel, tunica intima (TI), tunica media (TM), and tunica adventitia (TA), although they were most prominent in the tunica intima. Reprinted with permission from Y oung et al, Cardiovascular disease and adult healing cells: From bench top to bedside. J Stem Cell Res. 2017; 1(3) 002:1-8 (56).

A pulmonary fibrosis model (57,58) [approved by the IACUC] was created by ingestion of the chemotherapeutic drug busulfan. One of the serious adverse side effects of busulfan for treating ovarian cancer is the formation of pulmonary fibrosis in the lungs. Adult rats were challenged with the ovarian chemotherapeutic drug, busulfan. At six weeks post ingestion, animals were sacrificed and processed for immunocytochemistry using cell surface marker antibodies, e.g., stage specific embryonic antigen-4 [SSEA-4] to identify pluripotent healing cells and carcinoembryonic antigen-cell adhesion molecule-1 [CEA-CAM-1] to identify totipotent healing cells within the tissue (29,36). SSEA-4 and CEA-CAM-1 antibodies were visualized in damaged tissues undergoing repair (Fig. 4), as well as in peripheral areas of the lungs regenerating new

structures of the broncho-pulmonary tree, e.g., bronchi, bronchioles, alveolar ducts, and alveolar sacs (Fig. 5).

Figure 4. Regenerating lung tissue six weeks post ingestion of busulfan.

A, Cells labeled with SSEA-4 [PSCs] are located at the periphery of a damaged bronchopulmonary segment.

B, Cells labeled with CEA-CAM-1 [TSCs] are in the interstitial tissues between forming blood vessels, alveolar ducts, and alveolar sacs.

C, Smooth muscle cells in the walls of forming blood vessels are labeled with antibody, IA4 for smooth muscle alpha-actin. Also used as positive procedural control for immunocytochemistry.

D, Negative procedural control for immunocytochemistry tested whether there was any non-specific binding of reagents to the tissue. There was no non-specific binding as shown by the absence of staining of the tissue. Reprinted with permission from Y oung et al. Pulmonary diseases and adult healing cells: from bench top to bedside. J Stem Cell Res. 2017; 1(2) 003:1-9 (58).

Figure 5. Newly regenerated healing cells in post-busulfan-treated rat lung fibrosis .

- A, Regeneration of new bronchi.
- B, Regeneration of new alveolar ducts.
- C, Regeneration of bronchi.
- D, Regenerating alveolar sacs.

Reprinted with permission from Young et al. Pulmonary diseases and adult healing cells: from bench top to bedside. J Stem Cell Res. 2017; 1(2) 003:1-9

functional cells or maintenance cells (29,33,36). WBCs, platelet), and segregated into their respectissue stroma throughout the body (35-39,41-53). gradient centrifugation with serum, saline, and ster-(containing blood elements and healing cells) was a single 24-hour period (77).

Harvesting of human healing cells. When site- removed from the median cubital vein using venispecific locations were compared, the number of puncture (75). The healing cells were then isolated adult healing cells per unit volume was far less than from all cellular blood elements (e.g., RBCs, And their numbers are further reduced at any given tive categories. This was performed using time, site due to their ubiquitous location in all connective temperature, zeta potential, and differential density Instead of harvesting a specific site for healing ile water (76). Various methods were devised using cells, a proliferation agent was ingested to stimulate minimally manipulative procedures to transplant proliferation of the healing cells in situ. After a two- selected healing cells into specific sites throughout month interval, a mobilization agent was ingested to the body. This healing cell placement was based on move the newly formed daughter healing cells into the disorder being treated, the inherent sizes of the the blood stream. Four hundred-ml of blood cells, and differentiation organ involved, all within

Human Clinical Studies. The initial studies includ- Throughout the clinical studies, we noted recurring ed harvesting healing cells from patients using their themes that would greatly affect the efficacy of the own autologous healing cells coupled with diseases results, e.g., local anesthetics utilized (91), failure to affecting the nervous system [Parkinson's Disease] follow informed consent guidelines (92), separating (55,73,74), the heart [Myocardial Infarction] small healing cells from exosomes during isolation (4,31,54,55,57), and the lungs [Pulmonary Fibrosis] (93), and donor selection criteria (94). (4,31,54,57,58). Results showed 100% safety for Pulmonary Fibrosis (86) and Chronic Obstructive damage. 5. Activate cell surface receptors on healamined were Systemic Lupus Erythematosus (88), (tissue-specific exosomes). 6. Support a strong in-Nephritis leading to Chronic Kidney Disease (90). wound healing). And 7. Prevent tissue overgrowth. Orthopedic problems were addressed with respect to osteoarthritis of hip, knee, and ankle joints (51) and We currently have 20 people in the IRB-approved rheumatoid arthritis. Cumulative results demonstrat-CNSP open enrollment open access clinical study, ed a 100% safety record for transplantation of some for as long as 3-4 years, with most starting freshly isolated healing cells, from both autologous their study during the initial Covid lockdowns. The and matched allogeneic donors, and an 83% efficacy results so far show a 100% safety record and 100% for reversing signs and symptoms in 97 individuals. efficacy at reversing signs and symptoms of their

transplant methods and 100% efficacy for reversing **Combinatorial Nutraceutical Supplement Pill** signs and symptoms. Subsequent studies were ex- (CNSP) Some individuals attempting to gain access panded to include other diseases and trauma. We to the fresh isolate open access open enrollment used both autologous (self) healing cells and alloge-clinical studies were too fragile to undergo the fresh neic healing cells (as donor cells from adults isolate harvest procedures and therefore excluded matched for gender and ABO blood group). Chronic from the fresh isolate studies. For these individuals, diseases of the nervous system studied were Alzhei-I developed CNSP to mimic the in vivo activity of mer's disease (75), Age-related Dry Macular De-ex vivo-activated transplanted healing cells for those generation (76), Traumatic Blindness (78), Traumat-individuals too fragile to undergo the fresh isolate ic Spinal Cord Injury (79), Traumatic Brain Injury harvesting procedures. CNSP was designed to 1. (80), Stroke (81), Multiple Sclerosis (82), Chronic Stimulate proliferation of healing cells within their Inflammatory Demyelinating Polyneuropathy (83), connective tissue niches in situ. 2. Mobilize healing Sciatica, Neuropathies, and Amyotrophic Lateral cells from their connective tissue niches into the Sclerosis (84). Cardiovascualr Diseases included blood stream, 3. Increase circulation throughout all congestive heart failure and myocardial infarction organs of the body. 4. Activate cell surface receptors (85). The lung diseases examined were Idiopathic on healing cells to migrate towards sites of tissue Pulmonary Disease (87). Autoimmune diseases ex-ing cells to respond to local environmental cues Allergies, Celiac Disease (89), and Inflammatory nate immune system (key critical for successful

respective aliments. In addition, these individuals

pression, decreased brain fog, increased energy, in- [http://www.youtube.com/watch? creased cognition, and a better outlook on life, lead- v=tLpkIBCWlAY&feature=channel_page]. 3. ALL ing to a better quality of life.

popularly held beliefs, healing cells are present in https://www.researchgate.net/profile/Henry Young/ healing cells to be used for regenerative medicine. positive repair and restorative response, and/or ap-Healing cells and CNSP have been tested and vali-ply for acceptance into our IRB-approved open acdated for their ability to reverse signs and symptoms cess open enrollment Phase-0 (everyone gets treatstrate an efficacy of 83% at reversing signs and email state your interest. symptoms of certain diseases. Healing cells activat-

FDA. We are continuing, under IRB-oversight, with minimal to no adverse side effects. open enrollment and open access clinical studies.

nologies.

Due Diligence. 1. Google Henry E. Young PhD. 2.

independently and unanimously reported unforeseen I have posted lectures to You Tube discussing the side effects of CNSP. These side effects included ethics of using various types of stem cells for regenincreased color acuity (colors were brighter and erative medicine [http://www.youtube.com/ watch? sharper), a decrease in systemic pain, decreased de- $v=$ wj5zXVRfU2c &feature=channel page] and

What Does This Mean for You? Contrary to You can download my materials free of charge at adult animals, including humans. Methods have publications/?page=1. 4. If you would like to learn been developed to isolate and purify populations of more about healing cells and their ability to affect a of chronic (terminal) diseases, severe trauma, and ed) clinical studies for either fresh isolates and/or orthopedic disorders. Fresh isolate healing cells ac-CNSP, you can email Dr. Henry E. Young PhD at tivated outside the body are 100% safe and demon- young.hey1@yahoo.com. In the Subject line of the my research with adult healing cells has been posted to an open access website called ResearchGate.net.

ed inside the body with CNSP are 100% safe and **Conclusion.** Adult autologous healing cells and/ 100% effective at reversing signs and symptoms of or gender-matched and ABO blood group-matched certain diseases leading to a better outlook on life allogeneic healing cells offer HOPE for the reversal and a better quality of life. Currently, the fresh iso-of signs and symptoms that indicate repair of damlated healing cells and CNSP technologies are treat-aged tissues, reduction in pain, and restoration of ments, and not cures for these aliments. Healing function in individuals with severe trauma and/or cells, their isolation and transplantation technolo-chronic diseases having no known cure. Healing gies, and CNSP have not yet been approved by the cells and CNSP have done this with demonstrated

We are enrolling more patients to boost numbers of Conflict of Interest. There is a conflict of interest participants before application for FDA R-MAT with respect to enrolling participants in our IRBapproval, and subsequent general use of these tech-approved open access open enrollment Phase-0 (everyone gets treated) clinical studies for either fresh isolates and/or CNSP.

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