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Cancer, Inflammation and The Role of Nutrition

Tuomas Westermarck[1], Marika Crohns[1], and Faik Atroshi[1]
(1) Pharmacology & Toxicology, 00014 –University of Helsinki, Sanofi-aventis Oy, Helsinki, Finland

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Abstract

Cancer is a disease with multiple interwoven causes and the role of diet has become a well-recognized factor in cancer incidence. It has also been described as “wounds that fail to heal”. Cancer is due to accumulation of DNA mutations that confer a growth advantage and invasive properties on clones of cells. A variety of factors have been studied in relation to cancer including, nutritional deficiencies, chemical carcinogens, physiological conditions, habits, infections, medications and socioeconomic interacting with genetic susceptibility influence the accumulation of mutations in cells. There are many different types of cancers.

Some cancers will have tumours and others are cancers of the blood that do not have visible growths. Not all tumours are malignant; some are benign and are simply a tumour that can be surgically removed and the patient will recover. Other types of cancer are more insidious and cannot easily be treated. Fortunately, there are now treatment options for most cancers and those treatments are improving all the time. Chronic inflammation is associated with a high cancer risk. There is increasing evidence for close correlation between inflammation, the microenvironment and tumour-associated neo-angiogenesis causing the adverse outcomes of cancer. Good nutrition is the bridge to better health and may help lowering the risk of many diseases. Nutrition is important at every stage of carcinogenesis from initiation to promotion to progression and metastasis and.

Proper nutrition in the diet along with certain behavioural changes including lifestyle and environmental factors may help to maintains health. Although it is difficult to prove that eating certain foods can cure cancer, however, eating a wide variety of foods helps to ensure you get all the nutrients you need. While certain risks are un-changeable (genes for example), some lifestyle factors if modified may help reduce cancer risk. However, the relationship between the two is often very complicated and we don't yet have all the answers, but the message appears to be very simple - eating healthy and exercising for a strong body also applies to fighting cancer. It is premature to say that we can keep cancer from forming or returning through diet and exercise, however, it at least seems clear that we can better our odds against it. Not all health problems are avoidable, but one have more control over his/her health than one may think. Whether a person with a history of cancer in his family or are currently battling the disease a change in lifestyle factors can make a difference in helping to fight off cancer. It is also important to point out that some foods (food contaminants) actually increase the risk of cancer, while others support the body and strengthen the immune system. The challenge, however, is finding a consensus about which dietary changes you should make, or which types of diets can trigger certain cancers. For example, it's suspected that breast cancer might be caused by a high-fat diet, being overweight or obese which leads to the release of certain hormones in the body. However, no clear evidence exists to make that a proven fact right now.

Some risk factors are well established, such as smoking's link with lung cancer. But others are less recognised. And for oesophageal or gullet cancer, half of the risk comes from eating too little fruit and veg, while only a fifth of the risk is from alcohol. For stomach cancer, a fifth of the risk comes from having too much salt in the diet, some cancers, like mouth and throat cancer, are caused almost entirely by lifestyle choices, the studies shows. By making a good food choice, one may protect his/her health.

Introduction

Cancer is considered to be a complex multistep disorder, the result of a combination of factors including exposure to radiation and/or carcinogens (damage to DNA), infection, genetics, aging, immune function disorders, and lifestyle factors such as smoking (Nelson et al. 2003; Mahan & Escott-Stump, 2004; Kamangar et al.2009). A variety of external factors including nutrients in the environment interacting with genetic susceptibility influence the accumulation of mutations in cells. Nutrition is important at every stage of carcinogenesis from initiation to promotion to progression and metastasis. Therefore, Cancer may be regarded as a complex metabolic deficiency disease. Clinical trials have evaluated the effect of dietary nutrients on tumour development (Tallberg and Atroshi, 2011; Tallberg et al.2011). These dietary agents may help to suppress the transformative, hyper proliferative and inflammatory processes that initiate carcinogenesis. In traditional Chinese medicine feeding patients with exotic herbs could cure them. At that time it was impossible to analyse the precise functional factors ingested, but we seem now to have reached an academic form of traditional Chinese medicine since we can include specific pure alimentary components to construct a supportive curative diet. Spontaneous regression of cancer is rare, and has been called "The metabolic triumph of the host". It implies that these patients by chance have ingested a complicated combination of bio-modulating natural components to regain observations signify that the complex metabolic deficiency triggering cancer, and also genetic weaknesses, can be compensated by feeding patients specific functional alimentary components. Therefore, a biological, economical and non-invasive treatment modality is needed.

Cancer develops when cells multiply in the presence of oxidation and other damage. According to micro-evolutionary models, cells become damaged and change their behaviour, growing uncontrollably, and act like the single-celled organisms from which they originally evolved. The cancer cells' individualism overwhelms the cooperative control processes that are essential to a complex multicellular organism. Importantly, antioxidants limit oxidative damage and thus inhibit early benign cancer growth, preventing cancer from developing. Cancerous tumours results from a series of genetic changes having to do with cell division and growth control and genetic instability, mortality, the suicide mechanism in cells; the ability of the cells to migrate; the ability of the cells to attract to them a blood supply. Cancers represent a revolt within our bodies in which some cells have decided selfishly to go their own way, propagating their individual genes at the expense of the body as a whole. There are many forces that can prompt this internal uprising. Often it is mutation, damaged DNA, switching on genes. That is, cancer develops from changes that cause normal cells to acquire abnormal functions. These changes are often the result of inherited mutations or are induced by environmental factors such as UV light, X-rays, chemicals, tobacco products, and viruses. Several environmental factors affect one's probability of acquiring cancer. These factors are considered carcinogenic agents when there is a consistent correlation between exposure to an agent and the occurrence of a specific type of cancer. The effect of environmental factors is not independent of cancer genes. Sunlight alters tumour suppressor genes in skin cells; cigarette smoke causes changes in lung cells, making them more sensitive to carcinogenic compounds in smoke. These factors probably act directly or indirectly on the genes that are already known to be involved in cancer. Individual genetic differences also affect the susceptibility of an individual to the carcinogenic effects of environmental agents. About ten per cent of the population has an alteration in a gene, causing them to produce excessive amounts of an enzyme that breaks down hydrocarbons present in smoke and various air pollutants. Cancer appears to result from a combination of genetic changes and environmental factors. A change in lifestyle that minimizes exposure to environmental carcinogens is one effective means of preventing cancer. Individuals who restrict their exposure to tobacco products, sunlight, and pollution can greatly decrease their risk of developing cancer. Many foods contain antioxidants and other nutrients, such as colourful fruits and vegetables that may help to prevent cancer. These foods supply ample amounts of vitamin A, C, and E, as well as phytochemicals and other antioxidants that may help to prevent cancer. Eating the right kinds of foods during and after treatment can help you feel better

and stay stronger. Therefore, Cancer prevention is easier than you think. With a few simple lifestyle changes, you can drastically reduce your risk of many types of cancer. Many factors play a role in cancer development, but the good news is that most can be avoided.

The number of cancer cases around the world is increasing. The incidence has been associated with ageing, environmental factors and changes in lifestyle. Based on some research in animals and people, certain dietary measures have been suggested to prevent the progression of cancer. However, there is no solid evidence a healthy diet can prevent people developing cancer. The reasons that patients with cancer are using the dietary supplements are to enhance their health. However, consuming such dietary elements may also be at risk for drug interactions. By 2020, the world population is expected to have increased to 7.5 billion, of this number, approximately 15 million new cancer cases will be diagnosed, and 12 million cancer patients will die (Ferlay et al. 2010, Jemal et al. 2011). It is estimated that 7.6 million cancer deaths occurred worldwide in 2008. Lung (1.4 million, 18.2% of the total for men and women), stomach (0.7 million, 9.7% of the total for men and women), liver (0.7 million, 9.2% of the total for men and women), colorectal (0.61 million, 8.1% of the total men and women) and female breast cancers (0.5 million, 6.1% of the total for women) were the most common causes, accounting for more than half of all cancer deaths (Ferlay et al. 2010, Jemal et al. 2011, Soerjomataram et al. 2012).

Antioxidants are substances that prevent damage to cells caused by free radicals. Free radicals are molecules that have lost an electron, thus are unstable (Atroshi & Westermarck, 2005). These free radicals basically steal electrons from other molecules in effort to heal themselves, ultimately creating new free radicals in the process. By stealing electrons, it can cause damage to DNA, leading to the possible development of cancer.Antioxidants search for these free radicals and lend them an electron. This stabilizes the molecule, thus preventing damage to other cells. Antioxidants also turn free radicals into waste by products, and they eventually get eliminated from the body. They also have the ability to repair previous damage to cells. Free radicals are formed from a number of causes. Cigarette smoke, pollution, exposure to sunlight all causes the formation of free radicals. Other factors include normal daily processes like food digestion and breathing. Numerous animal studies have been published demonstrating decreased tumour size and/or increased longevity with a combination of chemotherapy and antioxidants. Our knowledge of antioxidants in a cancer setting is still at its infancy stage. The interactions between antioxidant and chemotherapeutics cannot be predicted solely on the basis of presumed mechanism of action when used concurrently. Fortunately, a large body of evidence is available to show a positive effect of high dose repeated use of antioxidants in the period before, during and after conventional cancer therapy.

Inflammation and Cancer

There is emerging evidence for a role of inflammation in the pathogenesis of cancer. Inflammation is known to cause DNA alterations. Chronic inflammation due to infection or injury is estimated to contribute to 25% of all cancers in the world. In recent years the relationship between cancer and oxidative stress has been extensively studied. Oxidative stress has been suggested to play a key role in carcinogenesis. Free radicals have been shown to mediate the anti-cancer actions of many chemotherapeutic regimens. Nonetheless the exact role of free radicals especially during cancer treatments is still largely unknown. Despite active investigation, knowledge is lacking concerning the local and systemic effects of free radical-generating treatments in cancer. Also radiotherapy exerts its cytotoxic effects through free radicals, either by direct action on DNA with damage as a consequence or indirectly by producing reactive oxygen species (ROS) (Figure 1). Many cancers are associated with increased production of ROS. The pathogenesis of cancer is a multistage process which involves mutations in critical genes required for maintenance of the cellular homeostasis. Oxidative mechanisms have a role in the initiation, promotion and progression of

carcinogenesis. This review will discuss free radicals involvement and the elevation of certain markers of oxidative stress in cancer with particular focus on the role of different antioxidants.

Most human disease is due to chronic inflammation resulting in loss of function of a joint, a blood vessel or an entire organ. In some organs, such as the heart and brain, acute inflammation can be fatal. Interleukin-1 (IL-l) is a master cytokine of local and systemic inflammation, and the availability of specific IL-l-targeting agents has revealed a pathological role of IL-l-mediated inflammation in a growing list of diseases (Dinarello et al. 2012). Oxidative stress is a major by-product of cellular metabolism and its regulation is critical for preventing disease and aging. Levels of reactive oxygen species (ROS) are generally higher in proliferating tumour cells than in normal cells and this may explain why ROS is a key component in the efficacy of chemotherapeutic drugs. There is evidence that carcinogens originated from food in some instance and the antioxidants can inhibit the activities of certain mutagens (Watson and Leonard, 1986).

Cancer as age - related diseases, Link between Faster 'Biological' Aging and Risk of Developing Age-Related Diseases

Although heart disease and cancers are more common as one gets older, however, not everyone gets them, and some people get them at an earlier age. It has been suspected that the occurrence of these diseases may in part be related to some people "biologically" ageing more quickly than others. Living cells have three main systems for protection and repair under oxidative stress (1) direct antioxidant enzymes (SOD, catalase, peroxidises), (2) proteases and phospholipases activated buy oxidative modification of membranes, (3) lipid and water soluble antioxidants. There is large number of physiological and pathological sources of oxygen radical and related compounds. A number of known exogenous agents are known to generate radical's species and thus increase the oxidative stress. Such agents include for example metal ions, pesticides, photochemical smog, ozone, ionizing radiation, tobacco smoke, numerous toxic chemicals and drugs. Importantly, it has also been well documented that a variety of endogenous processes are significant generators' reactions, mitochondrial electron transport, cytochrome P-450 detoxification reactions, phagocytic oxidative bursts, xanthine oxidase and lipid peroxidation etc. Lipid peroxidation has been suggested to be responsible for numerous deleterious effects observed in biological systems since it continuously proceeds by free radical reaction mechanisms after initiation. If this reaction is not terminated, a continuous self-feeding chain reaction is proceeding, making lipid peroxidation a good candidate to cause a variety of human pathology and possibly to participate in the prostate cancer processes.

Free radicals and formation of cancer

In recent years the relationship between cancer and oxidative stress has been extensively studied. The pathogenesis of cancer is a multistage process which involves mutations in critical genes required for maintenance of the cellular homeostasis (Powell et al., 2005). Oxidative mechanisms have a role in the initiation, promotion and progression of carcinogenesis (Toyokuni et al., 1995, Cooke et al., 2006; Ounjaijean et al. 2011). Oxidative DNA modifications are more common in cancerous tissues than in surrounding cancer-free tissues, which reinforces the conception that ROS play a role in the development of cancer (Jaruga et al., 1994). Persistent oxidative damage to DNA or impairment of antioxidant defence systems have been linked to mutation, activated transcription factors, modification of gene expression and chromosomal aberrations, i.e. genomic instability, processes which have been described in the progressions of cancer (Toyokuni et al., 1995, Morabito et al., 2004).

Inflammation is known to cause DNA alterations (Cooke et al., 2003). Chronic inflammation due to infection or injury is estimated to contribute to 25% of all cancers in the world (Coussens and Werb, 2002). Various chemical carcinogens such as chlorinated compounds, metal ions, barbiturates, phorbol esters, aromatic hydrocarbons and some peroxisome proliferators have been shown to induce oxidative stress and damage to DNA. They may therefore partly account for the development especially of work-related cancers. Very rare hereditary diseases such as Xeroderma pigmentosum, Franconia's anaemia and Cockney's syndrome are also associated with an increased cancer risk due to deficiencies in nucleotide excision repair (NER) (Powell et al., 2005).

Many cancers are associated with increased production of ROS (Toyokuni et al., 1995, Cooke et al., 2006).
The increased oxidative stress in cancer may be attributable to a variety of factors:
(1) Increased formation of ROS when the antioxidative defence mechanism works normally
(2) Unchanged status of exposure to ROS while the antioxidant defence mechanisms are decreased
(3) Failure in repair of oxidative damage, which leads to increased presence of ROS
(4) Combination of the above (Halliwell, 2007).

Free radicals and oxidative stress

Free radicals have been suggested to be involved in a number of disease processes. Recent studies (Russell et al. 2002) suggest that free radicals are involved in the development of cancer. The free radical theory of cancer suggests that there is a progressive decline in an organism's ability to resist free radicals reactions allowing irreversible tissue damage to occur. Regardless of their origin, it has been established that oxygen radicals represent a real threat to normal cellular function. Virtually all cellular components appear to be sensitive to radical/oxidant damage. Proteins, lipid, nucleic acids and carbohydrates are all known to undergo oxidative modification. The end result is dependent on the intensity of the oxidation stress and the capacity of the defences systems as well as the rate at which damaged bio molecules are removed. Decrease in oxidative repair may contribute to higher amounts of damaged macromolecules and ultimately to disease/cancer.

Cells are often exposed to high load of oxidants and free radicals. Oxidative stress can occur as a result of increased metabolic rate, increased oxygen tension, compromise of normal cellular antioxidants and many others endogenous and exogenous factors. Under normal conditions, the antioxidant defence systems are probably capable of maintain a low steady-state level of damage and thus protecting the cells. Among the risk factors for the development of prostate cancer are aging and life style. Under situations of oxidative stress and with increasing age, however, the organism may not be able to maintain homeostasis with deleterious and potentially unfortunate consequences. While it has not been conclusively determined whether free radicals are a cause or an effect of prostate cancer, it is clear that characteristic types of free radical damage increase with cancer. Free radicals are one of the environmental factors which contribute to cancer process.

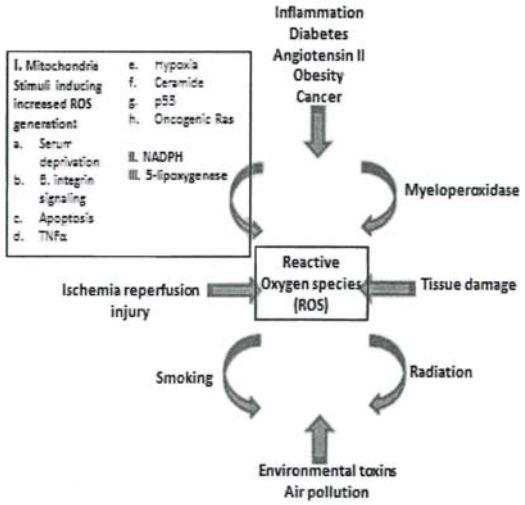

Figure 1. Cells are exposed to both endogenous and exogenous sources of reactive oxygen soecies (ROS)

Diet, Inflammation and prostate cancer

The causes of cancer have been largely attributed to genetic and environmental factors, including lifestyle, and are generally thought of as either avoidable or unavoidable. Dietary habits have been considered for years in epidemiological and case controlled studies to have an impact on cancer development and prevention. However, this association between diet and cancer has never been as clear as the correlation between smoking and cancer. Over nutrition, leading to obesity, has also been associated with increasing cancer development in many animal studies and is also considered a risk factor for many types of cancer (Johnson et al. 2007). Similarly calorie restriction appears to decrease risk for many cancers (Hursting et al. 2010). A major focus of diet and cancer research pertains to individual dietary components that may reduce or enhance cancer risk. Studying dietary components also gives more insight into the mechanisms involved in cancer development and how diet may play a role in modulating these mechanisms. There is an emerging consensus that situations of acute or chronic imbalance between the antioxidative capacity of cells and tissues, and the production of pro-oxidative species, is associated with the development of a number of human diseases. Despite enormous interest in the area of antioxidants as therapeutic tools, the development of foreign compounds as therapeutic antioxidants has provided little therapeutic benefit.

1 Many important physiological functions, such as the regulation of cell cycle (mitogenesis and apoptosis), are known to be tightly coupled to the induction of controlled episodes of oxidative stress in biological systems. This entails problems in terms of potential side effects for antioxidant therapy, which have been largely ignored in most clinical use of antioxidants. This may have serious implications for the choice of antioxidant principle to be used.

2 The actual choice of antioxidant therapy is it xenobiotic or endogenous, should be indicated based on sound molecular knowledge of the involvement of oxidative stress in the actual pathology.

Direct and indirect effects of diet and nutrition on cancer risk

The effect of diet can be direct, via the cumulative effect of exposure to nutrients and carcinogens in foods; in this case, the balance of cancer-promoting and -protective substances may contribute in defining cancer risk (Ahmad & Mukhtar, 2013). There are also indirect ways by which diet affects the cancer process. These include the effects of diet on energy balance and risk of obesity and the hormonal and metabolic responses related to energy balance. The latter are associated with

the metabolic syndrome and the inflammatory mediators linked to increased adipose tissue. In addition, diet as a determinant of growth and body composition may influence cancer risk both directly by affecting tissue growth itself and by affecting trophic hormones that mediate the growth process. Trophic hormones can influence the growth process and thus increase cancer risk. Obesity and rapid growth at critical times may increase the risk for some types of cancer; conversely, energy deficit and leanness may have a deleterious or protective effect for some cancers, depending on the timing or the tissue-specific effects of the nutritional deprivation (Parekh et al. 2012). Some infectious agents acquired through contaminated diets can affect cancer risk, such as chronic Helicobacter pylori infection, which induces inflammation of the gastric mucosa and thus affects cancer risk.

Antioxidant defence system

The human body contains a complex antioxidant defence system which depends on the dietary intake of antioxidants as well as the endogenous production of antioxidative compounds such as glutathione (Clarkson and Thompson, 2000; Majkowski et al. 2011). Antioxidants can act at different levels and by diverse mechanisms in the oxidative sequence (Halliwell & Gutteridge, 2007). Antioxidants can be classified into a number of different groups (Duarte and Lunec, 2005, Halliwell and Gutteridge, 2007):
1 Antioxidant enzymes: superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), glutathione reductase (GSR)
2 Antioxidative proteins: haemoglobin, ceruloplasrnine, transferrin, albumin, lactoferrin
3 Small-molecular-weight compounds: ascorbic acid (vitamin C), tocopherols (vitamin E), glutathione (GSH), uric acid, selenium, bilirubin, glucose
4 Ubiquinone (coenzyme Q_{10})
5 Flavonoids
6 Protein sulfhydryl (SH) groups (thiols)

The three major antioxidant enzymes are superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx or GSH-Px). SOD and CAT are proteins which act primarily in the cell cytoplasm and form the first line defence against oxidants, e.g. superoxide anion and hydrogen peroxide (Gago-Dominguez and Castelao, 2006). SOD exists in several isoforms with different active metals in the center and different amino acid constituency. In humans, three different forms of SOD are cytosolic-CuZn-SOD, mitochondrial Mn-SOD, and extracellular SOD. Fe-SOD exists in animals but not in humans. Though CuZn-SOD is located in most parts of the cell, Mn-SOD is the most important scavenger of O-, converting it to hydrogen peroxide and oxygen (Halliwell, 2007).

Prostate cancer a search for etiologic, therapeutic, predictive and prophylactic factors

Carcinoma of the prostate (CaP) is still justly considered a hormone dependent disease but lacking curative treatments and a comprehensible aetiology. Several signs for adrenal involvement coupled with metabolic factors impelled this analysis on its aetiology. CaP seems to stem from a deficient production of two neuroendocrine components produced by adrenal zona-reticularis cells (ZR). One increase FSH, the other prolactin (PRL) levels which together control CaP. A curative ZR feed-back reaction can be activated in CaP patients by dietary supplementation compensating the aetiological metabolite deficiency (Tall berg et al. 2011; Crohns et al. 2013).

Natural components prescribed are; amino-acids serine (Ser), arginine (Arg); trace- element ions, strontium (Sr), vanadium (V) and molybden (Mo) in addition to ingestion of PSA-a serine

protease- levels may become stable or regress because the ingested substrate (L-Ser) causes enzyme inhibition, PSA-levels decrease. Gleason scores may decline from 8 to 4, paired with reduced urinary distress. CaP found incidentally, or by screening should primarily be treated utilizing dietary supplementation sine PSA may decrease in a dose-response manner, whereby serious side-effects caused by invasive treatments could be avoided. A good prognosis is usually registered as increased FSH, prolactin (PRL) and SHBG levels, declining DHEAS and PSA. Androgen ablation intervals' vary from 2-24 months, based on patient response permitting time for the adrenal feed-back reaction to function (registered as FSH increase). This bio-modulating schedule has been sustained already for decades (Tallberg & Atroshi, 2011) without emergence of a hormone refractory state (HRPC). A rare form of CaP is diagnosed from soft tissue metastases, with activin levels excessively increased, while inhibition stay low, but patients respond positively to this bio-modulating treatment. Orchiectomized patients have immeasurable inhibition, although normal activin levels as in pregnant females or ladies on oestrogen substitution therapy.

During intermittent LHRH treatment analogue treatment FSH is strived to be increased, over normal levels (>10 IU), with normal or increased PSA before the next hormone treatment is indicated. Androgen ablation should not attain a PSA nadir since excessive androgen suppression decrease FSH to low levels (<1 IU) when the adrenal feed-back cycle is exhausted, instigating HRPC. Fatal adrenal exhaustion is not due to pituitary dysfunction since PRL is then markedly increased in patients. There is a new incentive for screening since CaP can be arrested by non-invasive dietary means alone. Improved diagnostic serum markers; EPCA-2, Kinases, PSA velocity, MRI and constructive dietary trails should diminish the need for 12 biopsy cores as spread of malignant cells effecting recurrent CaP, already of >35% after prostatectomy. Gleason scores decreases, and bone metastases regress, albeit BPH may be activated requiring dutasteride medication, blocking 5a reductase I & II receptors.

Constrains linked to stem cell research, as compared with the refined medical regulation of cell induction caused by organ-specific mitochondria

The excitement generated by the recent approval of stem cell research in the USA, allowing the use of embryonic stem cells in humans may although, not form a ground-breaking new medical treatment modality. Stem cell research as it is scientifically performed today is constrained by specific limitations. These procured undifferentiated cells are not genetically identical with the recipient. After harvesting they must undergo manipulation by culturing in cell media. This may in a subtle way change their basic biological functions which is so essential for a truly physiologic prospective function in the new recipients' organ. The stem cell may not be introduced in the right inductional control segment exerted by the central nervous system (CNS). After cell culturing it may also be difficult to induce stem cells to function properly in a novel epigenetic milieu, especially if they have lost their organ specific mitochondria in the culturing process or during transfer of the stem cells to the recipient.

Introducing stem cells is like transplanting an organ and the recipient's immune system might reject the intruder. The toxic drugs required to prevent the immune rejection will not make it easier for the biologist to transform the surviving stem cells into their proper function, placed in a potentially hostile new surroundings. The sum of these scientific constrains originating from these vital but un-physiological preparative exploits will naturally delay clinical applications.

The “plasticity” of stem cells may lead to the use of a stem cell bank, but it would only partially circumvent transplantation problems, since storage could eliminate the essential organ specific mitochondria required for normal cell proliferation and transcription. A further serious complication is associated with malignant induction of teratomas, or any other malformed cell structures because stem cell induction is so complex that it is prone to turn into cell-structure despite our efforts to induce specific normal cell substitutes.

The source to obtain stem cells has varied a lot from embryos, placenta, amniotic membranes, umbilical cord cells etc. It is a way of circumventing killing an embryo but the cells are still potentially foreign when used as transplants. Unfertilized eggs have been chemically stimulated to create non-viable embryo cells with only one parent and less transplantation rejection potential, but may introduce other biological problems.

Organ-specific mitochondria utilized to create viable tissue cells, a novel scientific endeavour

The world's most promising new medical technology is introduced by the function of organ specific mitochondria. The human genome project revealed the surprising nucleotide analogy of chromosomes between different species. Mitochondria don't only produce energy for the nucleus, it has its shaped its chromosomal structure over eons of its phylogenetic toil. They regulate the healthy transcription of organ specific cells and can correct faults in the nucleotide sequences. Identical mutations present in both chromosomes, in Arabiodopsis plants, could lead to 10% of the offspring to be healthy. Mitochondria detected the nucleotide aberration and corrected the fault during regeneration (Lolle & Victor, 2005). Human "male" embryonal skin transplants could cause normal skin to develop in the bottom of the skin burn lesions. The skin formed was actually the girls own skin (Hohlfeld et al., 2005), and not caused by the male embryos skin transplants used. Organ specific mitochondria must be present in the embryonal skin.

These organ-specific mitochondria can transgress into the girls' tissue cells, and since they regulate the chromosomal genomes, which they have created during evolution-and of the girl's tissue cells can transform into skin, since the skin gene is present in any cell. The learning of the use of organ-specific mitochondria in biology and medicine will form the major scientific challenge for this coming century. The clinical use of mitochondria is not hampered by the same restrictions stem cells suffer from.