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Research on Transfer Factors in Disease Treatment and Prevention

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Transfer factor (TF) ... has been used successfully over the past quarter of a century for treating viral, parasitic, and fungal infections, as well as immunodeficiencies, neoplasias, allergies and autoimmune diseases. Moreover, several observations suggest that it can be utilized for prevention, transferring immunity prior to infection ... Thus, a specific TF to a new influenza virus can be made swiftly and used for prevention as well as for the treatment of infected patients.

- Giancarlo Pizza et al, 2006

The last century witnessed several important advances in disease treatment and prevention. In 1949, at a time when the benefits of vaccines were becoming apparent and penicillin, along with the sulfa antibiotics developed in Germany, were gaining reputations as wonder drugs, a New York University tuberculosis researcher named Dr. H Sherwood Lawrence made another important discovery. He extracted intracellular fluid from circulating white blood cells in patients who had been exposed to tuberculosis (TB). He then injected the contents of these cells into non-exposed patients. Using a test for an immune response known as delayed type hypersensitivity, he demonstrated that the non-exposed patient's immune system now recognized TB and responded to it as though it had already fought it. In other words, immunity to TB was somehow *transferred* from one person to the next via the white blood cell extract.

In the years that followed, Dr. Lawrence began to refer to the mystery components of the white blood cell extract as "transfer factor," as they somehow transferred immunity from one patient to the next. Research during the decades since suggests that the information contained in the molecules Dr. Lawrence called transfer factor can instruct the immune system to do several different things, thereby influencing immunity via different routes, and might actually come in several – three to be exact – different sizes. Thus, rather than one factor, there seem to be multiple factors involved in transferring immunity.

Where Do Transfer Factors Come From, Why Are They Made, and How Do They Work?

Transfer factors appear to be short strands of amino acids and perhaps small bits of ribonucleic acid (RNA). It is thought that transfer factors are manufactured within Helper T-cells; cells that coordinate attacks launched by the immune system. Once released by Helper T-cells, transfer factors influence immune system activity in several ways. Their presence is read by other immune cells as an indication that a Th1-mediated immune battle is under way (see Glossary for a definition of Th1 immunity). This results in the birth of new Helper T-cells, Natural Killer cells and macrophages, the conversion of young lymphocytes into Th1-related immune cells, decreased levels of Th2-related cytokines, increased levels of Th1-related cytokines and a general strengthening of the Th1 response.

In addition, like antibodies, transfer factors bind to specific antigens. In the case of transfer factors, the antigens are located on the surface of infected body cells. New Helper T-cells use the presence of antigen-specific transfer factors to focus the immune response against particular threats. By sticking to antigens on infected cells, transfer factors presumably flag the infected cells for destruction by Cytotoxic T-cells.

In essence, transfer factors are the smaller siblings of antibodies, but operate to facilitate the destruction of infected body cells via cell-mediated immunity rather than the labeling of free-floating antigens via antibody-mediated immunity.

Transfer Factors In Disease Treatment and Prevention

In the last half-century, hundreds of published studies have examined the ability of transfer factors to help the body deal with diseases. A search of MedLine for work on transfer factors yields roughly 1000 relevant publications spanning more than 50 years. Of those studies, 600 examined the therapeutic value of transfer factors in disease treatment and prevention. By all accounts, casting a wider net reveals thousands more relevant publications.

Cancer

Nearly 100 studies have been conducted on the effects of transfer factors on cancer, either in patients suffering from cancer, or on cancerous cells in *in vitro* preparations – such as cancer cells grown in laboratories.

Based on *in vitro* (laboratory) studies, there can be little doubt that transfer factors facilitate the ability of lymphocytes to destroy cancerous tissue. In 2006, researchers in Mexico examined the ability of extracts taken from white blood cells from cows to prevent breast cancer cells from further division and to facilitate their destruction (Franco-Molina et al, 2006). The experiment was successful, and the researchers demonstrated a dose-dependent effect of the extract on cancer cells, meaning that more extract led to greater destruction of cancer cells. The extract had no impact on normal, healthy cells, only cancerous cells, indicating that the constituents of the extract, including transfer factors, specifically help the immune system deal with pathogens and do not damage healthy tissue.

In 2005, Pineda and colleagues (Pineda et al, 2005) reported on a fascinating study in which they examined the impact of transfer factors on glioma – brain cancer involving glial cells – in rats. Here is what they found:

“TF reduced significantly the tumour size, and increased CD2+, CD4+, CD8+ and NK cell counts, it also increased the percentage of apoptotic [dying] tumour cells”

Dozens of studies have examined the ability of transfer factors to help humans suffering from cancers of various types. In 1996, the Italian researcher Giancarlo Pizza and his colleagues (Pizza et al, 1996) reported on efforts to treat a form of prostate cancer typically unresponsive to conventional therapies. The authors generated transfer factors that bind to antigens on prostate cancer cells and injected them into sick patients once each month. In the words of the authors,

“Fifty patients entered this study and received one intramuscular injection of 2-5 units of specific TF monthly. Follow-up, ranging from 1 to 9 years, showed that complete remission was achieved in 2 patients, partial remission in 6, and no progression of metastatic disease in 14. The median survival was 126 weeks, higher than the survival rates reported in the literature for patients of the same stage.”

Thus, compared to the expected length of survival for men with this form of prostate cancer, it appears that transfer factor treatment significantly prolonged the life of patients in the study. Though such findings are promising, the lack of a true control group – a group receiving everything else but the transfer factors – makes it difficult to determine the role that transfer factors actually played in the patients’ disease progression.

During that same year, Pizza and colleagues (Pilotti et al, 1996) reported on a study in which a control group was used. Ninety-nine patients with small cell lung cancer were given transfer factors and their survival times were compared to 257 patients with lung cancer who were not given transfer factors. Those given transfer factors survived significantly longer than those not given the treatment.

Herpes viruses

There are eight known herpes viruses, including the herpes simplex viruses (HSV-1 and HSV-2), which cause herpes outbreaks on the face and genitals, the varicella-zoster virus (VZV) that causes chicken pox and shingles, cytomegalovirus (CMV), the Epstein-Barr virus (EBV) that causes mononucleosis, and the HHV-6 A&B viruses now associated with conditions like ME/CFS and MS. Research evaluating the ability of transfer factors to help the immune system overcome these viruses has yielded overwhelmingly positive results.

Khan and colleagues (1981) examined the ability of transfer factors to prevent relapses in 16 subjects with recurrent HSV-1 (cold sores) and HSV-2 (genital) outbreaks. Patients were injected with broad spectrum transfer factors on a weekly or monthly basis. Following treatment, 8 patients stopped having outbreaks altogether while the remaining 8 exhibited a significant reduction in the frequency of outbreaks. Roughly half of all subjects exhibited low T-cell counts at the study onset, and all patients exhibited an increase in T-cell numbers after treatment with transfer factors.

Pizza et al (1996) reported that 44 patients suffering from recurrent HSV outbreaks – 22 with genital outbreaks and 22 with labial (face) outbreaks – responded positively to treatment with transfer factors specific to HSV-1 and HSV-2. Research from the same lab, also published in 1996 (Meduri et al, 1996), demonstrated the ability of HSV-specific transfer factors to reduce the frequency of outbreaks in those whose outbreaks occurred in the eyes.

Currently, there are several prescription drugs available that are capable of reducing the frequency and duration of HSV-1 and HSV-2 outbreaks. The active component of most such drugs is acyclovir. The drugs differ in the amount of acyclovir that they actually deliver to the body in useable form. When taken daily, it reduces the frequency of outbreaks, and the outbreaks that occur are of shorter duration and lesser intensity. When taken only for acute outbreaks, they shorten their duration but have no impact on the frequency of outbreaks throughout the year.

As discussed above, like acyclovir, transfer factors also reduce the frequency and severity of herpes outbreaks. A few studies have directly compared the efficacy of these two approaches against the viruses. Estrada-Parra and colleagues (1995) administered transfer factors to 20 patients suffering from recurrent outbreaks of HSV-1. Most patients had already been treated with acyclovir prior to their inclusion in the study. Broad spectrum transfer factors reduced both the frequency and duration of outbreaks in patients. Their observations led the authors to conclude:

“These results suggest that, at present, TF may be considered the therapeutic agent of choice in the treatment of herpes simplex type 1 disease.”

The findings of the above study are limited by the fact that no direct comparison to acyclovir was actually made. The superiority of transfer factors over acyclovir was inferred from the comparison of current treatment outcomes to the patients' prior experiences with acyclovir and other drugs.

In 1998, this same group of researchers reported findings from a study in which they directly compared the effectiveness of transfer factors and acyclovir against outbreaks of VZV, the herpes virus that causes chicken pox in kids and shingles in adults. In this study, 28 patients with acute outbreaks of shingles were given either transfer factors or acyclovir for seven days and then monitored for another 14 days. Transfer factors were superior to acyclovir at shortening the duration of the outbreaks. Further, transfer factors, but not acyclovir, increased the number of Helper T-cells and improved other markers of immune function.

Research with lab animals, like that with humans, demonstrates the powerful effects of specific transfer factors against herpes viruses. Viza et al (1985) assessed the efficacy of transfer factors in preventing the deleterious effects of HSV-1 and -2 by administering HSV-1 and -2 specific transfer factors to mice before exposing them to a normally deadly dose of the viruses. The HSV-specific transfer factor preparation protected the animals from death. Interestingly a transfer factor preparation specific to CMV rather than HSV-1 and -2 did not protect the animals from the lethal dose of HSV.

Human Immunodeficiency Virus (HIV)

At its core, the pathogenesis of HIV and its progression to AIDS involves a debilitating attack on cells that comprise the Th1-mediated immune response, particularly Helper T-cells. It seems logical to expect that transfer factors could be powerful adjuvants in the treatment of HIV given their ability to trigger increased levels of Helper T-cells, as well as Cytotoxic T-cells, Natural Killer cells and macrophages. Currently, only a few studies have examined the utility of transfer factors in the treatment of HIV/AIDS, but the available data are promising.

Carey et al (1987) assessed the impact of transfer factors from healthy controls on immune system function in nine patients with HIV infections that had progressed to full-blown AIDS. They concluded that: "... administration of transfer factor to patients with AIDS resulted in partial immune reconstitution. Further studies are indicated to examine the clinical efficacy of this immune response modifier in the treatment of AIDS."

In 1996, Giancarlo Pizza and colleagues reported on a study in which they administered HIV specific transfer factors, in an oral preparation, to 25 patients infected with HIV. They noted clinical improvements or a stabilization of clinical markers in 20/25 patients.

That same year, Pizza and colleagues (Raisz et al, 1996) reported the results of a study in which they directly compared groups receiving standard antiviral therapy to those receiving combined antiviral therapy and HIV-specific transfer factors. Patients received either Zidovudine alone or in combination with transfer factors. Those receiving the Zidovudine-transfer factor combination exhibited a larger increase in Cytotoxic T-cell counts and in the levels of interleukin-2, a critical immune messenger that promotes the genesis of T-cells and promotes the Th1 immune response in general. Interestingly, however, there were no differences between the groups regarding actual levels of Helper T-cells.

A 2002 report from Russian scientists also suggests that transfer factors could play an important role in the management of HIV (Granitov et al, 2002). The researchers examined immune cell counts in HIV patients administered a commercially available oral transfer factor supplement. Fifteen patients were treated with transfer factors alone. Ten patients in a control group were treated with common antivirals for HIV but not transfer factors. Treatment lasted for only seven days and immune measurements were taken one week later.

The majority of subjects in the transfer factor group exhibited increased levels of Helper T-cells and Cytotoxic T-cells. The pattern of cytokine release was altered in transfer factor-treated subjects in a way that facilitates Th1 immune activity. Levels of circulating immune complexes, which reflect the combination of an antibody and an antigen, were reduced to normal levels in 10 of 15 subjects. In the control group, positive changes were as likely to occur as negative changes. In the words of the authors (Granitov et al, 2002):

"We conclude that transfer factors therapy considerably improves the immune status of HIV-infected patients and can be recommended in combating the pathogenesis of the disease. Further studies are needed to determine optimal therapy, the necessity to repeat courses of the treatment and the frequency of therapy needed."

Two recent studies from researchers at the Center for Biological Research in Havana, Cuba, suggest that transfer factors are capable of directly interfering with HIV replication (Ojeda et al, 2000; Fernandez-Ortega et al, 2004).

Using Transfer Factors to Transfer Immunity

It appears that supplemental transfer factors allow a person not previously exposed to a pathogen to skip the immune response normally required to trigger the creation of transfer factors for that particular pathogen. Transfer factors transfer "memory" for immune battles of the past, no matter in which host those battles originally took place. As far as the transfer factor recipient's immune system is concerned, it has already been exposed to the pathogen, and thus reacts promptly if the pathogen ever enters the body. By preparing the immune system for a pathogen before it enters the body, transfer factors remove the element of surprise critical to infestation by infectious agents and prevent cellular invasion by viruses, mycobacteria and cell wall deficient bacteria.

Let us look at an interesting example in which the ability of transfer factors to transfer immunity was used to protect patients from contracting an illness. In 1980, Steele and colleagues used transfer factors in an effort to immunize young leukemia patients against chicken pox, caused by VZV. In the words of the authors:

“Sixty-one patients with leukemia and no immunity to chickenpox were given dialyzable transfer factor or placebo and followed for 12 to 30 months in a double-blind trial designed to examine the clinical efficacy of transfer factor. Sixteen patients in the transfer-factor group and 15 in the placebo group were exposed to varicella zoster, and most of them had a rise in antibody titer. Chickenpox developed in 13 of 15 exposed patients in the placebo group but in only one of 16 in the transfer-factor group.”

Thus, when patients with leukemia and no resistance to chicken pox are treated with transfer factors for the chicken pox virus, almost everyone develops immunity to the virus.

Availability of Transfer Factors

Until quite recently, transfer factors were only used in hospital settings, most outside of the U.S. They were custom-made for patients from human blood cells in a laboratory and were not available to the public or to most MDs. In the last decade, several companies began using patented processes to extract transfer factors from cow colostrum and chicken eggs. Such extracts are now widely available. The ability to obtain transfer factors from colostrum and eggs is a remarkable development with the potential to significantly alter the future of disease treatment and prevention.

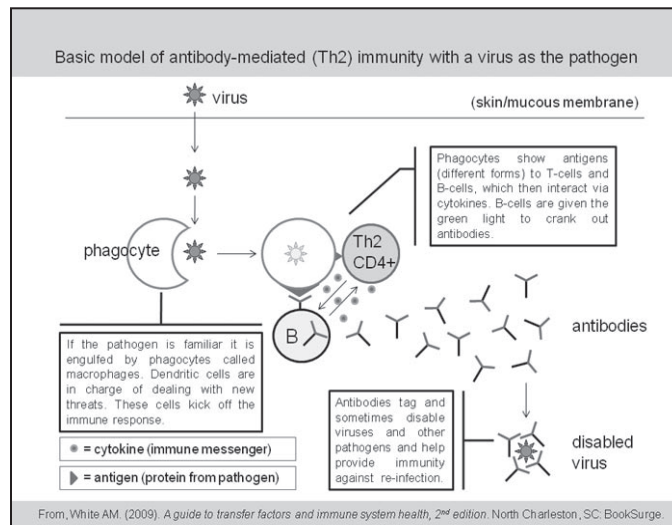
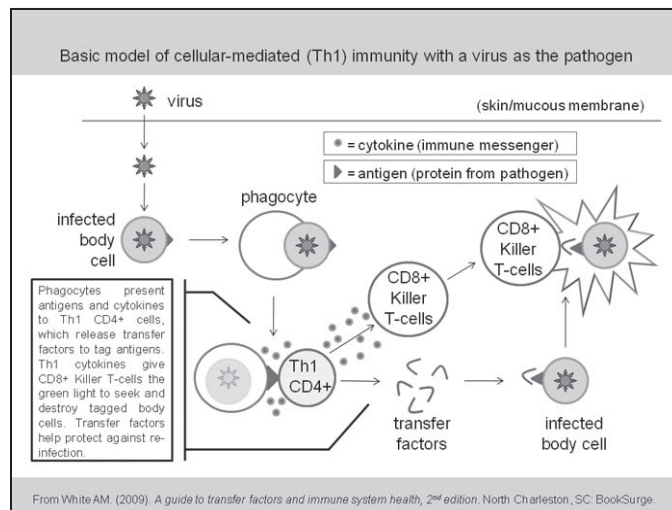
Summary

Research strongly suggests that transfer factors are effective for helping the body beat an array of disease states that involve faulty or overloaded immune function and can prevent infections before they occur. Transfer factors seem to prime the body for battle against intracellular pathogens, like viruses, mycobacteria and cell wall deficient bacteria, and help quash infections before they can take root. Transfer factors turn non-immune-related white blood cells into immune-related white blood cells and stimulate the birth of new Helper and Cytotoxic T-cells, Natural Killer cells and macrophages. After stimulating an increase in T-cell counts, transfer factors orient these new T-cells toward a target, presumably by influencing the nature of the antigen receptors expressed by the cells. Further, by binding to antigens on infected body cells, transfer factors paint infected cells for destruction by Cytotoxic T-cells.

Given in supplement form, they can be used to help the body beat infections. Given before infections occur, they act in a manner similar to vaccines and protect the body from becoming infected. While antibodies are at the heart of antibody-mediated (Th2) immunity, transfer factors hold the key to cell-mediated (Th1) immunity. 🌸

This article is comprised of excerpts from the book, A Guide to Transfer Factors and Immune System Health, 2nd Edition (North Charleston, SC: BookSurge Publishing, 2009) by Aaron White, PhD.

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Glossary

Th1 response: Immune response triggered by Th1 Helper T-cells. Natural Killer cells and Cytotoxic T-cells are used to identify and destroy cells infected with viruses and bacteria. Also important for fighting cancer cells, fungi and parasites. The pathway to cell-mediated immunity. Suppressed in people with many immune-related conditions and infections, like Lyme and HIV.

Th2 response: Immune response triggered by Th2 Helper T-cells. B-cells are used to produce antibodies which label extracellular pathogens for destruction. Pathway to humoral or antibody-mediated immunity. Overactivity here involved in allergies and autoimmune conditions. The response primarily evoked by standard vaccines.

Transfer factors: Strands of amino acids and RNA released by Th1 Helper T-cells. Signal other Helper T-cells to direct resources toward Th1 response. Like antibodies, they bind to antigens, but on the surface of infected body cells rather than on free-floating pathogens. Thought to label infected cells for destruction by Cytotoxic T-cells. Small enough to be absorbed orally. Can transfer immune information from one person or other animal to another. Their importance for disease treatment and prevention could be on par with antibiotics and vaccines.