NK Cell-Based Immunotherapy in the Treatment of Cancer Using a New Arabinoxylan Rice Bran Compound

by Professor Serge Jurasunas

Introduction

Despite advances in cancer treatment achieved over the last decades, it still remains a failure where the outcome of standard and especially palliative treatment is often poor due to cancer cell resistance. However, in these past few decades, we have assisted in a series of articles in international publications from many countries, proclaiming that our own immune system is now a new weapon against cancer. This has become the fourth pillar of oncology after surgery, chemotherapy, and radiation.

One particular immune cell has gained more interest over the past two decades from researchers: the natural killer (NK) cell, which seems to play a crucial role in cancer.

A number of publications and published scientific papers demonstrate the association between the NK cell and cancer, particularly breast cancer.[1] Boosting the NK cells increases the killing of cancer and presents a significant advantage both in the prevention and treatment of cancer.[2]

Among many available compounds investigated, one has commonly been the subject of publication for its efficacy to activate NK cell activity and for the killing of cancer cells in vitro and in vivo. This compound is known as rice bran arabinoxylan compound (RBAC). It has been widely investigated especially over the past 25 years by scientific researcher M. Ghoneum, PhD, from Drew University in Los Angeles, California. This researcher has spent most of his professional life studying NK cell activity and substances that are able to modify immune function.

According to Dr. Ghoneum, RBAC is the most powerful compound we have to activate immune cells, particularly NK cells. But we are going to see that RBAC not only increases NK cell activity but has other qualities as an anticancer agent. In some studies, patients that were treated with RBAC in addition to conventional therapy (CT) compared with those treated with CT alone showed less recurrence of cancer, higher survival rates, and improved quality of life (QOL).

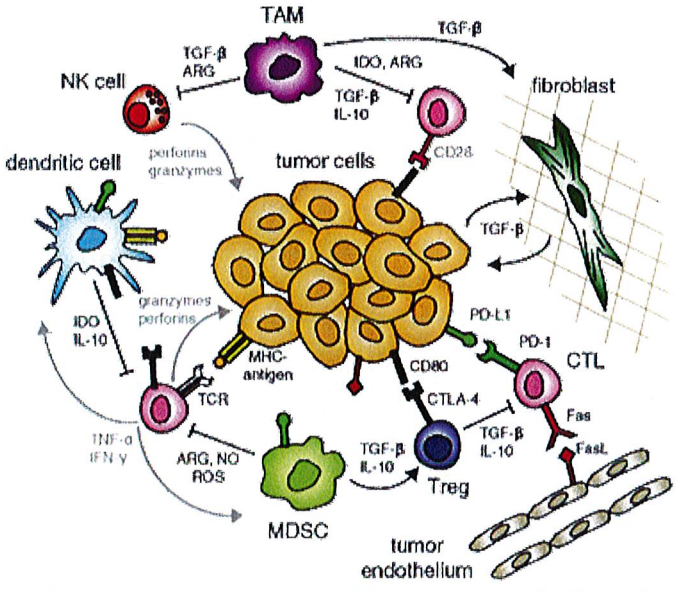
Various animal studies and human clinical trials, including different types of malignancies, have demonstrated that RBAC is a potent biological response modifier (BRM), being a safe compound with no toxicity that does not exhibit hypo-responsiveness.

It was only around 1990 when I discovered the newly developed rice bran arabinoxylan compound from my Japanese contact in Tokyo and learned about natural killer cells that I started studying NK cells and began to include this compound in my cancer protocol. The more you read about NK cells, the more you understand about the necessity to activate these unique immune cells to increase the death of cancer cells. Twenty-eight years is a long time, and it has permitted me to experiment with various protocols and compounds with cancer patients. You can imagine what I have accomplished during this period and how many cancer patients I have treated in my clinic. Of course, we always expect that what we use offers some efficacy, but this article will show what RBAC is able to do. You can read reports explaining how RBAC increases NK cell activity, and how a tumor can decrease in size, or that tumor markers decrease; but if you directly witness the results with your patient, it may better convince you.

What Are NK Cells and Their Role in Cancer?

Natural killer cells were first discovered in humans and mice in 1975 and were initially identified as unique lymphocytes ➤

Figure 1. Landscape of the Immune Tumor Microenvironment



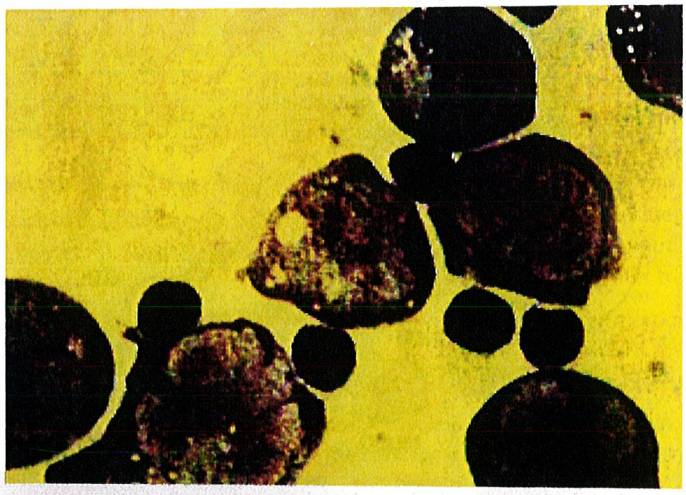
distinct from B and T cells, being critical to the innate immune system but also bridging the innate and adaptive immune systems. NK cells are known to differentiate and mature in the bone marrow, lymph nodes, spleen, tonsils, and thymus, where they then enter into the circulation. They are a large granular population of leukocytes that can directly kill virus-infected cells or tumor cells.[3] Today NK cells are viewed as our first line of defense against intruders and cancer. As we age, NK cells lose their functionality thus leaving us with more vulnerability to viral diseases and especially cancer, which could be one cause of increased cancer rates in aged individuals.[4] In the aging, NK cells may be fewer, secrete perforin with more difficulty, and die faster compared to NK cells in younger people. Many new animal and human studies show that NK cells play a central role in the immune surveillance of transformed cells,[5] while evidence suggests that our low NK cell activity may be a risk factor for malignancy or metastases, as well as a negative prognostic indicator.[6] In fact, our lifestyles, as well as our environment and food habits, influence human natural killer cells, increasing or decreasing their activity to protect against infection or cancer[7]; today, we probably have decreasing NK cell activity, which increases the risk of cancer. Cancer patients have very low NK cell activity, about 50-70% decreased activity compared to healthy individuals.

Function of NK Cells

NK cells constantly patrol in our blood and lymph in their resting phase; but if infected or cancer cells are detected through the ligand place on the surface of target cells, they become active with cytotoxic properties.

Unlike other immune cells, NK cells require no special instruction to recognize a specific antigen. They have the ability to directly recognize cellular targets without prior sensitization, mediated by a network of inhibitory or activating receptors expressed on the cell surface that control NK cell activation. It is the integration of the activating and inhibitory signals that determine if the NK cell becomes cytotoxic. The use of immunotherapeutic agents to increase activation and decrease inhibitory signaling has the potential to generate NK cells with enhanced tumor-lytic capacity.

Figure 2. RBAC activating NK cells and attacking cancer cells. We observe a single NK cell attaching itself to 3 cancer cells for destruction.



NK cells become active in response to immuno-regulatory proteins called cytokines that interact with NK cell receptors. NK cells are regulated by various cytokines such as IL-1, IL-12, IL-15, IL-18, and IL-21 that interact with inhibitory or activating receptors on NK cells and communicate with other cells. For instance, the combination of IL-12 and IL-18 is especially potent to trigger interferon (IFN)-γ. The NK cells are converted into lymphokine-activated killer (LAK) cells. These LAK cells propagate, produce cytokines and adhesion molecules to target cells, and increase tumor lysis activity.

NK cells also generate an adaptive immune response by secreting pro-inflammatory cytokines and interferon such as TNFa and IFN-γ that activate other immune cells such as macrophage, B-cells, and dendritic cells.[8]

NK Cell Cytotoxicity

The natural killer cell cytotoxicity can be demonstrated in several related ways. The primary and most important known mechanism when attacking cancer cells is based on the release of cytotoxic granules from their cytoplasm after becoming active in response to cytokines.

First, NK cells attach themselves to receptors on the membrane of cancer cells and inject granules called perforin, which is a membrane-disrupting protein that perforates the target cell membrane and forms pores, creating an aqueous channel that permits the entry of another cytotoxic protease.[9] (See Figure 2)

When the pores are formed, NK cells release and inject, via this channel, granzymes serine proteases,[10-11] which cleave caspases 3-7-9 and subsequently induce apoptosis in target cells by degrading protein involved with DNA integrity. This is the main channel utilized by NK cells to destroy cancer cells. One NK cell can kill a cancer cell in five minutes time and repeat this 27 times before dying. It shows the great capacity of this unique cell to kill cancer. As we have already indicated, cancer patients have a low NK cell activity compared to healthy individuals. Both in vitro and in vivo tests confirmed that the number of cytotoxic granules increase with NK cells stimulated by RBAC.

It seems that our lifestyle and diet can also increase the activity of NK cells with a balanced diet that included a variety of vegetables, fruits, meat, and fish being associated with higher levels of perforin and granzymes, efficiently serving as protection against cancer cells.[12]

Natural Killer Cells and Cytokines

Not only can NK cells target and kill cancer cells through cytotoxic granules, but when activated they have one other interesting indirect targeting mechanism. When activated NK cells release pro-inflammatory cytokines and interferons such as IFN-γ and TNFa[8] that can cause direct tumor necrosis by inflicting tumor-associated capillary injury but also (and

this is important) generate an adaptive immune response by activating other immune cells such as macrophages, T-cells, and dendritic cells.[13] The secretion of IFN-γ by NK cells plays a key role in the stimulation and maturation of dendritic cells and thus increasing, even more, the killing of cancer cells.[14] Also, one other advantageous factor is the fact that IFN-γ secreted by the NK cells contributes to inhibiting angiogenesis and blood vessels, a major step required for tumor growth and expansion. Therefore, NK cells seem to have a wide targeting effect besides killing cancer cells directly.

Perforin-Independent Apoptotic Channel

NK cells use another apoptotic weapon channel to kill cancer cells. This is a perforin-independent mechanism using the death receptor ligand. This cytotoxic pathway relies on tumor necrosis factor (TNF) receptor superfamily members.[15] The two main TNF receptors used in apoptotic induction are FAS (CD95) and Trail (TNF-related apoptosis-inducing ligand).[16] Trail is an apoptotic molecule that interacts with the death receptors Dr4 and Dr5 and activates intracellular apoptosis through cleavage of caspase 8, which in turn can directly activate caspases 3-9. Literature has shown that Trail can kill any cancer or leukemia cell regardless of their degree of malignancy.

Trail is also considered a natural anti-tumoral cytokine expressed on a wide variety of tissue[17,18] and on the surface of NK cells, T-cells, macrophages, and dendritic cells. IFN-γ released by NK cells induces Trail expression that in turn induces apoptosis or lysis of cancer cells. It was proven that many types of tumor cells express high levels of ligands for NK cells receptors, which leads to their recognition and killing by NK cells.

Factors That Depress NK Cell Activity

NK cell populations can be affected by several factors that decrease their capacity to secrete perforin and granzyme in cancerous patients. For instance, surgery and chemotherapy may contribute to decreased immune system activity, which is already reduced in cancer patients.[19,20] In breast cancer patients, there is a strong association between depression and the disease, especially associated with breast cancer recurrence. Depressive disorder was associated with a higher risk of breast cancer recurrence among patients after breast surgery.[21] Professor Ron Herbeman (NCI), now in charge of the Institute of Oncology at the University of Pittsburgh, demonstrated with a group of 116 surgically treated breast cancer patients that the more the NK cells are active in the week that follows the surgery, the better are the chances of long term survival.[22] Evidence also demonstrates that radiotherapy significantly decreases the number and function of cells from both innate and adaptive immune systems, including natural killer lymphocytes, TNFa, and interferon-γ cytokine activity.[23] Therefore, we may understand why repeated chemo/radiation can in many cases contribute to stimulating tumor growth.

A decline in NK cell count and function appears to be a biomarker of overall risk for disease and cancer.

Arabinoxylan Rice Bran

Rice Bran Arabinoxylan Compound

Rice bran arabinoxylan compound (RBAC) is a nutritional supplement developed by Daiwa Pharmaceuticals Co. Ltd. of Japan and sold as a functional food in Japan. RBAC is produced by enzymatic hydrolysis of hemicellulose B, a dietary fiber found in rice bran. The principal ingredient, arabinoxylan, which is a highly branched and complicated sugar composition of mainly arabinose and xylose, is obtained from modified bran rice with a hot water extraction consisting of *Lentinula edode, Coriolus versicolor,* and shitake mushroom mycelia culture. The process breaks long polysaccharide molecules into small ones. Hemicellulose B contains a relatively small amount of a molecule with a polymerization degree of approximately 200, which can be absorbed through the intestinal wall after oral intake.

Before the process, arabinoxylan itself has no immuno-stimulating activity. When the long polysaccharide molecules are broken up into smaller components, they can be rapidly absorbed by the small intestine in undigested form and enter the blood circulation with strong immuno-stimulating action, especially on natural killer cells (NK cells),[24] and additional oncogenic effects.

During the past 24 years, a large number of rigorous preclinical and clinical research studies have shown that RBAC's anticancer effects are founded on the ability of this natural compound to act as a potent biological response modifier. (See Figure 3) RBAC has been shown to possess immunomodulating anticancer effects and can work synergistically with chemotherapeutic agents in vitro.

Eleven studies, including non-randomized pre-post-intervention studies and six randomized controlled trials (RCTs), reported the following effects of RBAC:

- Enhanced immunoprofile,
- Reduced side effects,
- Decreased antigen tumor markers,
- Improved treatment outcomes, and
- Significantly increased lifespan.

This is what I am going to discuss in this article based on many reports, but also this is what basically I have observed with patients that we have treated using RBAC, as well as in long-term administration.

RBAC Boosts NK Cell Activity

When taken orally as a food supplement, RBAC has the ability to activate different arms of the immune system to attack cancer such as by the proliferation of T and B cells and macrophages,[25] and activation of dendritic cells[26] and especially NK cells function, while also enhancing the production of several cytokines.[27] Both in vitro and in vivo tests confirmed that RBAC increases the amount of cytotoxic granular content (perforin and granzyme B) of NK cells. This has been demonstrated morphologically and biochemically since NK cells of cancer patients are usually degranulated with no cytotoxic effect.[28] NK cells kill virally infected cells or cancer

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cells through the secretion of cytotoxic granules to target cells, causing their rapid death.[29]

The increase in the binding ability of NK cells was also investigated. The NK cells of a human who took 45 mg/kg/day of RBAC for 30 days were incubated with K-562, which were the target cells, and then the increase of the binding ability was measured. In a subject who took RBAC, this significantly increased to 38.5% compared with 9.4% before intake.[24]

Figure 3. How RBAC Boosts NK Activity Against Cancer Cells

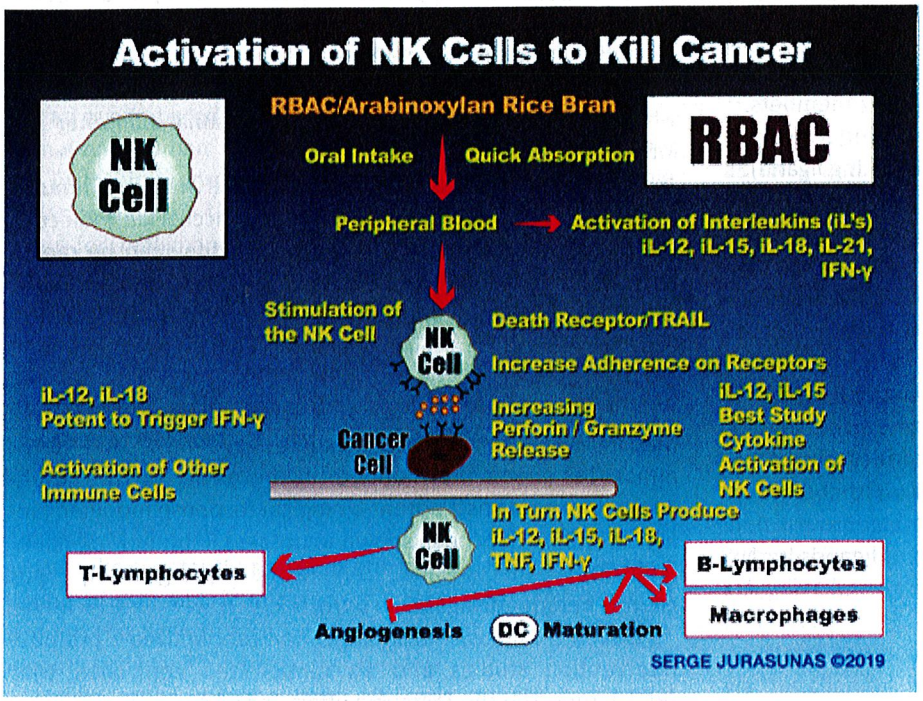
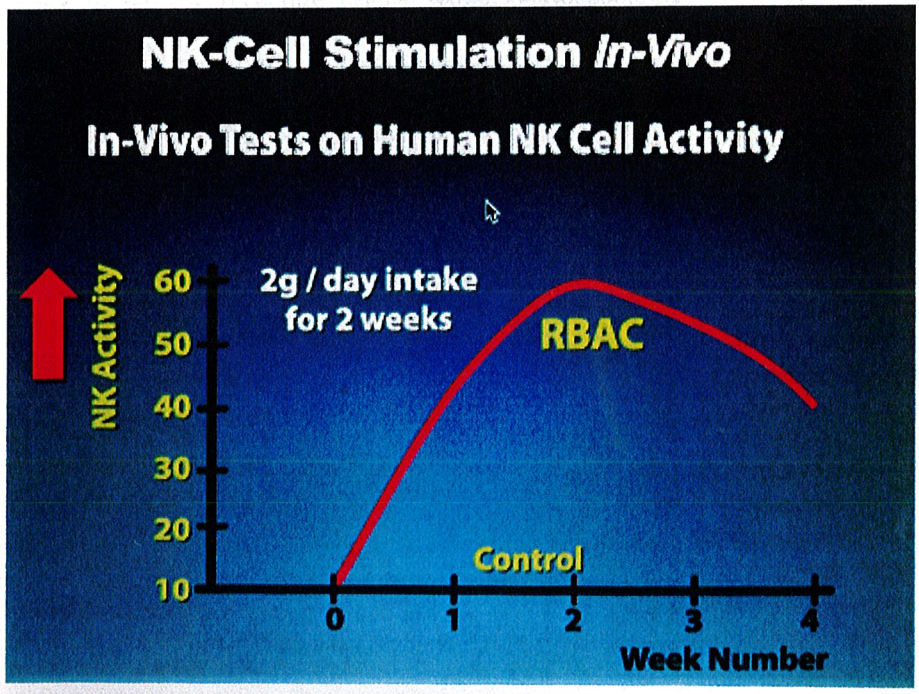


Figure 4. NK-Cell Stimulation In-Vivo



In NK cell stimulation in vivo with volunteers (without disease or cancer), we observed a remarkable increase of NK cell activity after two weeks with a 2 g/daily intake of RBAC, which was sufficient for this test. It then started to decrease, but remaining at the same dose enhanced NK activity, which continued to rise at three to six months after treatment. (See Figure 4)

In another study, 27 cancer patients with different types of advanced malignancies (7 patients with breast cancer, 7 with prostate cancer, 8 multiple myelomas (MM), 3 leukemia, and 2 cervical cancers) were examined. All patients were under treatment with conventional therapy and were given 3 g of RBAC daily; 3 g is an optimal dose for cancer patients since they have lower NK cell activity.

NK activity was then examined at two weeks, three months, and six months. NK cell activity was examined by a 51Cr-release assay using K-562 tumor cells as a target. Target ratios ranged from 12.1-100.1. The result showed that patients had a low level of basal NK activity. Treatment with RBAC caused a remarkable increase in NK activity after two weeks and the percentage of induction were as follows:

- Breast cancer: 154-332%.
- Prostate cancer: 174-385%.
- Leukemia: 100-240%.
- Multiple myeloma: 100-537%.
- Cervical cancer: 100-275%.

Enhancement of NK cell activity continued to rise both after three months and six months after treatment.[30] What is interesting is the fact that this increase in NK cell activity continued and was maintained for five years with continued supplementation, indicating that RBAC was still active with no hypo-responsiveness, which is the case with some other compounds.

An additional study to check on the immunomodulatory effects of RBAC included thirty-two cancer patients with different types of malignancies with depressed NK cell activity after post-conventional cancer treatment who were treated with RBAC for two weeks.[28] A significant increase in NK cell activity up to 10-fold was observed;

and an increase in NK cell granularity and binding capacity were detected in all types of cancer, and tumor markers decreased.

As explained above, most cancer patients have low NK cell activity, which can decrease even more after repeated cycles of chemotherapy and radiotherapy that may promote resistant tumors as well as local and systemic toxicity. I was not surprised that RBAC dramatically increased NK cell activity so high in prostate cancer but also MM. In the past, some of my cases of MM lived up to eight years taking a daily dose of RBAC. Professor Ron Herbeman stressed that there is considerable evidence that patients with cancer express an abnormal immune response, which has been observed with many types of cancers including breast cancer.

For example, a 44-year-old female was diagnosed with breast cancer in December 1994. She received surgery and chemotherapy, after which her NK cell activity baseline was 39.9% in May 1995. One month after starting RBAC/MGN3 supplementation, it was 48.9%. By October 1995 it was 83.5%. Since then this level has been maintained, and all follow-up mammograms have shown no sign of relapse.

As another example, five patients with breast cancer were given RBAC at 3 g/day and their NK cell activities were measured by 4-hr 51 Cr-release assay using K-562 tumor cells as a target. The results showed the following:

1. Patients had a low level of basal NK activity 12.7%-58.3%. Participants had their NK activity significantly enhanced by RBAC (41.8%-99.5%) on a ratio basis of 10%-100%.
2. The augmentation in NK activity was detected as early as one to two weeks post-treatment and was further increased with the continuation of RBAC at 3 g/day.
3. Two patients who participate early in the study (6-8 months) went into complete remission.[31]

In one other study in human patients with malignancies, RBAC showed remarkable results in 48 patients with multiple myeloma, whose median age was 65 years.[32] A dose of 2 grams per day for three months produced nearly an 84% increase in NK cell activity by the end of the second month of supplementation. This correlates with my own clinical experience and the example that I spoke about, not to mention an overall better quality of life.

A very interesting study has been conducted at the Sano Surgical Clinic by Kamataso Sano.[33] After surgery, 205 patients who had a recurrence and ➤

Figure 5.
Rice Bran Arabinoxylan Improves Survival Rate in Cancer Patients

Relation between total survival rate, NK activity and survival rates in 2 groups

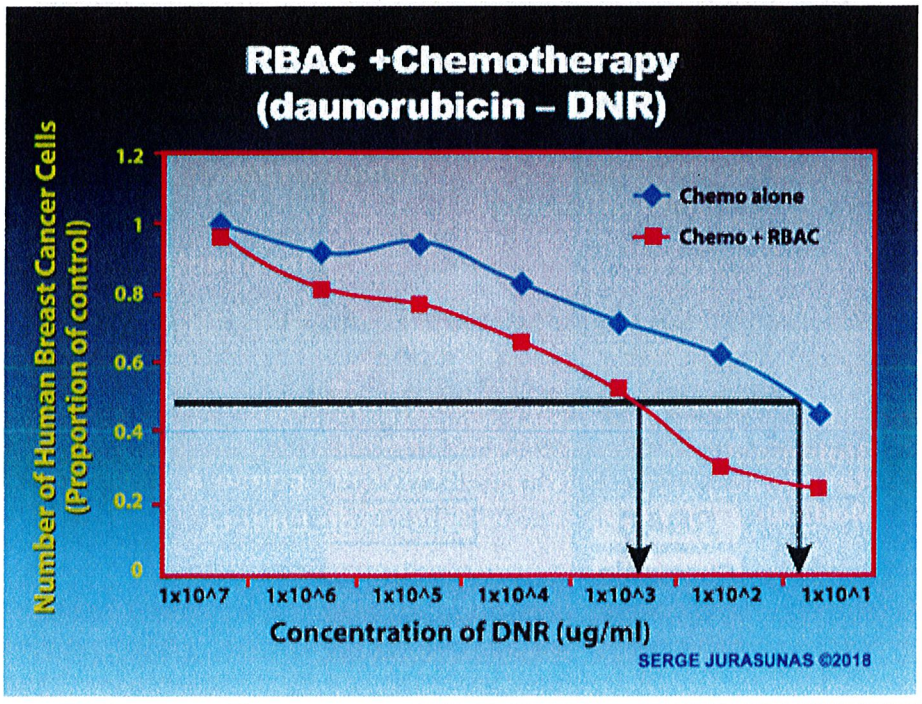
Group	RBAC Group	Control Group
Total survival rate	52/96 (54.2%)	19/63 (33.9%)
NK activity category		
Less than 19.9%	17/40 (42.5%)**	2/16 (12.5%)
20% - 40%	18/35 (51.4%)*	7/25 (28.0%)
More than 40%	17/21 (81.0%)	10/15 (66.7%)

% significant to the control group **p<0.01 *p<0.05

Takahara K. (Sano Surgical Clinic, JAPAN) : The Abstract of the 3rd Annnul Meeting of the Japanese Society for Complimentary & Alternative Medicine treatment, 2000

Figure 6. RBAC Improves Chemotherapy Effectiveness

Gollapudi, S, Ghoneum M. Modified arabinoxylan from rice bran, sensitizes human breast cancer cells to chemotherapeutic agent, daunorubicin. *Cancer Detecte Prev*. 2008.32.1-6 .



metastatic disease (late stages III-IV) were hospitalized and treated with complementary alternative medicines and anticancer drugs with lesser side effects. The 205 patients, hospitalized for six months, were divided into two groups: 109 patients were treated with the prescribed therapies (control group), and 96 patients were additionally treated with RBAC along with their prescribed therapies (the RBAC group) for 18 months. RBAC was given orally at 1 gram, three times per day after meals.

All patients were monitored for NK cell activity as an indication for the variation of immunoparameters. Patient QOL was also checked by observation and inquiry during the study. It had already been shown that patient NK cell activity after surgery was low. However, after administration of RBAC, NK cell activity increased as did the survival rate. As a result the 18-month survival rate after treatment was 54.2 % in the RBAC group and only 33.9 % in the control group after taking alternative medicine (mushroom, Gerson therapy, etc.). Increased NK cell activity was higher in the RBAC group than in the control group, resulting in a 1.5 times higher survival rate in the former group.

Figure 5 shows that the more NK cells that are activated, the higher is the survival percentage. Even with only a 20% NK cell increase, the survival rate is higher compared to the control group. Some patients with NK cell activity higher than 40% prior to entering the study had a very high survival rate compared to the control group. Also, QOL was greatly improved in the RBAC group in terms of less pain, fatigue, nausea, and diarrhea, along with having a better appetite.

Figure 7.

Comparative Treatment Groups – RBAC Sensitizes Metastatic Breast Cancer Cells to Paclitaxel In-Vitro. Co-culture of RBAC sensitized 4T1 cells to Paclitaxel causing an even greater decrease in cell survival. 4T1 cells were co-cultured with varying concentrations of RBAC 500 and 600 µg/ml and Paclitaxel for (24 hours and 48 hours). M. Ghoneum. *Anticancer Research*. January 2014.vol 34. N.1. 81.87

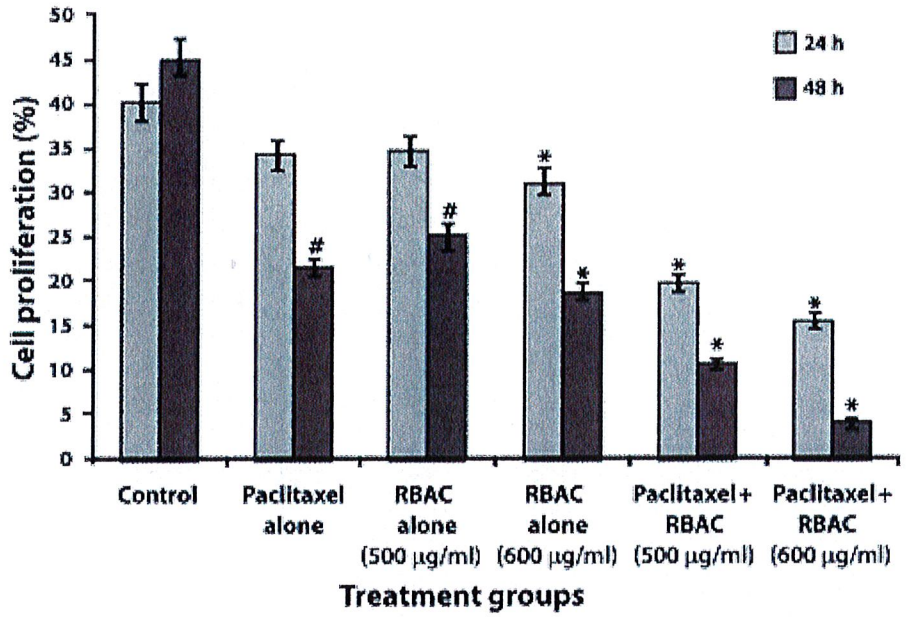
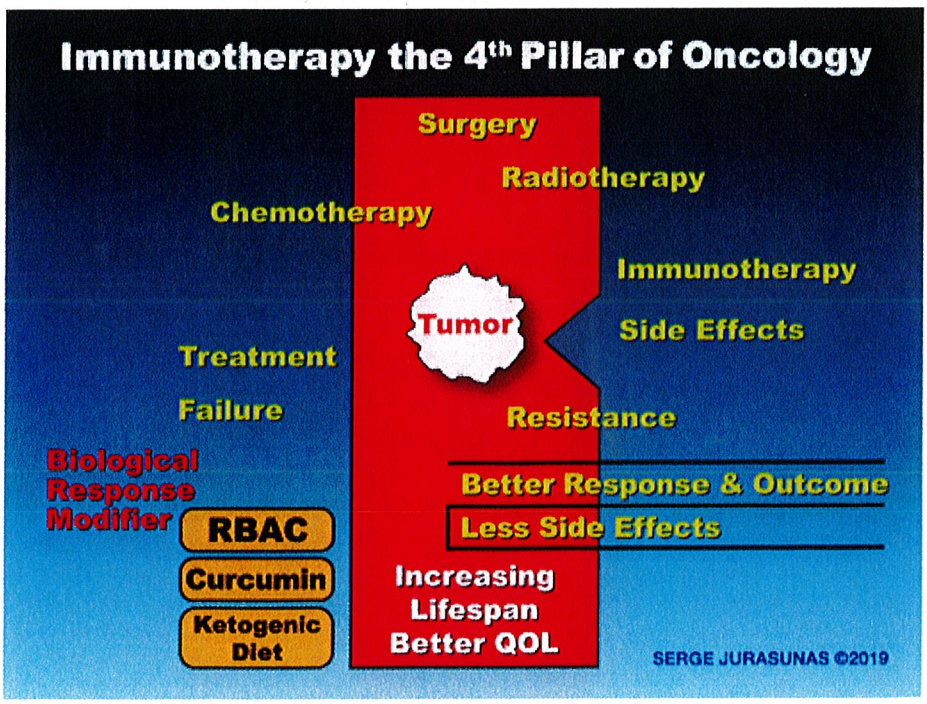


Figure 8. Immunotherapy and Biological Response Modifiers – The 4th Pillar of Oncology



RBAC and Chemotherapy

When working with chemotherapy, increasing its effectiveness to obtain better results and fewer side effects should be a main concern in integrative oncology. Many reports, studies, and also cases from my own clinical practice have proven that daily intake of RBAC may improve the efficacy of chemotherapy by reducing tumor size and decreasing tumor markers more quickly with better QOL. Citing more recent examples, a pancreatic tumor of 4 cm. was reduced to 1 cm. in about 30 days before surgery and chemotherapy by taking only RBAC at a daily dose of 3 grams. A female of 38

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Arabinoxylan Rice Bran

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years with a stage II breast cancer could not undergo chemotherapy because of heart dysfunction. Her CA 15.3 blood test at 67.7 decreased to 21.2 after four months of taking RBAC together with curcumin. A 5 cm-wide tumor in the case of an aggressive cancer was reduced to 13 mm. after three months with chemotherapy and daily intake of RBAC. We have other examples with CA 125 decreasing from 218 to 50, and then 11.5 and 5 when RBAC was taken together with the agent paclitaxel in a case of stomach cancer.

... RBAC works in synergy with curcumin to stop the cellular cycle at G0/G1, to induce apoptosis. Each one works in synergy with chemotherapy, and both substantially increase chemotherapy effectiveness.

In fact, several studies have shown how RBAC sensitizes metastatic breast cancer cells to paclitaxel[34] and to Daunorubicin[35] in vitro, via activation of caspase 3 and 8, increasing apoptosis of cancer cells. (See Figures 6 and 7) Paclitaxel is a powerful chemotherapeutic drug for the treatment of a number of cancers, including breast, ovary, and prostate, that is supposed to kill cancer cells by inducing cell cycle arrest and apoptosis. Its action, in fact, can be improved with a variety of natural compounds that also arrest cell cycles at G1 or G2 like curcumin, for instance. However, a high concentration is required to induce an apoptosis effect on cancer cells. Such a high dose, however, is associated with severe side effects, including cardiomyopathy and neutropenia. The study showed that RBAC potentially contributed to a reduction in chemo side effects while also reducing the dose yet offering the same efficiency in killing cancer cells.

One other interesting ability of RBAC is to help increase the accumulation of the chemotherapeutic agent Daunorubicin in the tumor. As we may know, some agents may not fully penetrate or accumulate into a tumor and therefore show less efficiency. Therefore, these studies show how RBAC sensitizes chemotherapy for a better result.

RBAC in Advanced Cancers

RBAC intake has been shown to extend lifespan with the improvement of quality of life for progressive cancer.[33,36] Cancer patients can take RBAC for several years without toxicity (LD50=36.0 g/kg) as demonstrated by extensive toxicity study that shows complete safety. RBAC is still efficient and without hypo-responsiveness, which is the characteristic of several other compounds, even when taken for few years.[37] I have observed in many advanced cancers with metastasis that taking RBAC has up to a five-year life extension period, improving patient QOL and physical condition and, for some, no further progression of the disease, meaning that RBAC is still active.

Conclusion

This article reviewed the ability of NK cells to target cancer cells from several options and examines key studies that were published examining the effect of RBAC on NK cells, dendritic cells, T and B cells, and on malignancy.[28] This is only a small review about scientific studies[38,39] citing the enormous capacity of RBAC as a biological response modifier and as an anticancer agent. It is also important to mention that RBAC is superior to various other compounds that claim to have anticancer properties but have little or no scientific evaluation – which is not the case with RBAC.

Chemotherapy and radiation treatment have not brought significant improvement to the longevity of cancer patients for the past 30 years or more. According to some official reports, most of the drugs don't even work with most patients and are apparently effective for about a quarter of patients.[40] Our aim is to support conventional therapy in a way that has better results with chemotherapy, such as by reducing tumor size, decreasing the number of metastatic lesions, decreasing antigen tumor markers, minimizing side effects, and having long-term remission – if not speaking of cure. This is what we can accomplish when associating RBAC with chemotherapy.

Now I would like to mention the fact that RBAC works in synergy with curcumin to stop the cellular cycle at G0/G1, to induce apoptosis.[41] Each one works in synergy with chemotherapy, and both substantially increase chemotherapy effectiveness. I have seen cases with breast cancer recurrence and multiple metastases getting much worse after new chemotherapy but rapidly improving by taking only RBAC and curcumin.

Therapeutic daily dose: RBAC - one sachet of 1 g three times per day, and liquid liposomal curcumin - from 3000 to 4500 mg per day.

Surely more studies about RBAC will be done in the coming years since immunology will take a most important place as a new weapon in the panoply of conventional therapy but also of alternative medicine.

RBAC is available as MGN-3 in Europe and was sold as MGN-3 in the past in the US and is now available as BRM4.

The research studies on RBAC can be found online at www.ipraxic.org.

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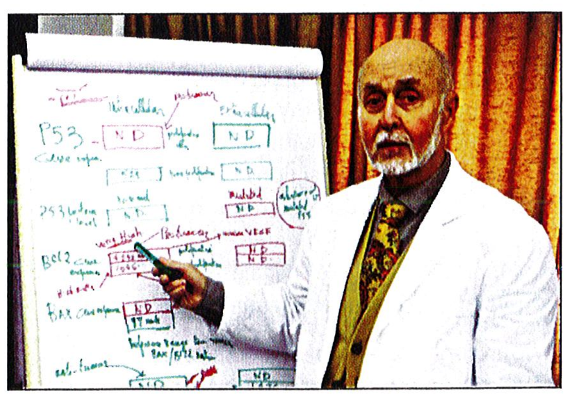

Serge Jurasunas is an internationally well-known practitioner and researcher in complementary oncology and molecular medicine besides being a naturopath and a fervent believer in nutrition and detox since 1967. He is a professor of naturopathic oncology at Pan Am University of Sciences and Natural Medicine and had been a former professor at Capital University of Integrative Medicine in Washington, D.C.

Professor Serge Jurasunas has devoted over five decades treating cancer of all types and grades, developing innovative therapies, being a pioneer in several approaches to cancer including cellular respiration and mitochondria immunotherapy, stem cell therapy, and live blood analysis and the oxidative dried blood test, since 1979.

He spent the past 12 years investigating, researching and putting into clinical application P53 tumor suppressor gene and other apoptotic players, telomerase etc. He delivered lectures in over 45 countries, including Russia, Australia, Poland, Hungary, Croatia, Germany, and Lithuania. He has been honored and decorated for his work, including the Silver Medal for Research and Invention by the French Academy and the Grand Academic Gold Collar of the Accademia Internazional Di Pontzen in the field of medical research, In 2017 he received the Lifetime Achievement Award (USA) for his 50 years of work and a pioneer in the field of education and natural health care.

He is the author of over 150 papers, lectures, articles, and eight books in three different languages and has been a frequent *Townsend Letter* contributor since 1999. Professor Serge Jurasunas maintains a private part-time practice only for cancer patients but also has outside patients from several countries via E-mail and Skype.

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