TOWNSEND LETTER
The Examiner of Alternative Medicine
Wednesday, September 7, 2022

Salicinium Clinical Pearls and Caveats

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Many fine articles on salicinium have been published in the *Townsend Letter* in 2013, 2014, 2015, 2016, 2017, and 2021, and are available in the Archives. I would like to present a brief overview of my journey in the world on integrative oncology using salicinium and my current perspective on the task of successful problem solving for cancer patients.

The field of integrative oncology is a new frontier for medical problem solving. Cancer is the epidemic no one is talking about. Even five year "disease free" survival is increasingly difficult to achieve. We are seeing a huge increase in new cases of first-time diagnosis at Stage IV cancer in patients under the age of 35. Worldwide hope for a "cure" remains paramount in patient consciousnesses, yet even disease-free survival is difficult to attain despite our advances in drug development and technology. When we look at the traditional treatment options of chemotherapy, radiation therapy, surgery, hormonal modulation, and immunotherapy, these modalities continue to evolve. Nonetheless, many patients are seeking treatment outside of these options.

There is increasing discontent on the part of patients and families when faced with these choices. With the instances of cancer being so high in the population, it seems everyone knows several people who have cancer; and many have watched their friends and family suffer and die on traditional regimens. A week does not pass when I interview a new patient who tells me of someone they know who died after the first dose of chemotherapy or was told "your only hope is to receive chemotherapy until you die." Thus, many are turning away from the "standard of care" since they feel it will not produce the result they want and hope for, namely life and vitality beyond cancer. Fear of death is one thing, but patients state that fear of death by torture is unacceptable.

As a family member I walked with my sister through her journey with leukemia that transformed into an aggressive B-cell lymphoma. She chose the traditional route of chemotherapy with Rituximab and all of her tumors would disappear only to reoccur 3-4 months later. Her diet of sugar and carbs never changed. Eventually, her cancer became resistant to Rituximab and she began taking an experimental oral chemo agent and continued with Rituximab. Only three weeks after starting, she developed a complication of the drug called, "Richter's transformation" where the bone marrow pours all formed white blood cells at once. She developed bilateral pneumonia and died 48 hours later. After this life experience, I decided I could do better. A spinal cord injury subsequently stopped my career as a vascular surgeon and I turned back to my life passion and combined all my experiences to applying my knowledge of cell biology and biochemistry to medical problem solving. My intention was to discover how to treat cancer from multiple aspects of its unique metabolism and to use

traditional methods of treatment combined with metabolic based modalities in a synergistic and non-conventional way.

I have been studying and treating patients using this strategy for many years. I noticed cancer is increasingly difficult to eradicate, and environmental challenges such as EMFs (5G), long term side effects of pesticide use, toxicity from synthetic materials, and untreated surreptitious viral and bacterial infections have become prominent confounding factors. If not addressed, patients have no hope of achieving disease-free survival.

The genius of Otto Warburg, a Nobel prize winning biochemist who lived from 1883-1970, brought to our attention the profound observation that normal cells will convert from oxidative metabolism (mitochondria produced energy) to anaerobic fermentation or glycolysis, when exposed to low oxygen tension for an extended period of time. We experience this phenomenon on a temporary basis, when we exercise heavily and work our muscles to "failure" with repeated reps. Anyone who has undergone a strenuous workout has had muscle soreness caused by lactate release that can last for hours, if not a day or two.With rest and hydration our muscles revert back to oxidative phosphorylation and the lactate washes out, relieving pain. This phenomenon is called facultative anaerobic metabolism because the cells can convert back to normal. However, Warburg demonstrated that all sick cells, which include those infected with bacteria, viruses, fungi, candida, parasites, and cancer cells all use glycolysis as a primary mode of energy production forever.

Glycolysis is an energy inefficient process that can only yield 2 ATP vs. 34 ATP, which is produced by mitochondrial oxidative phosphorylation. Furthermore, "perfect storm" conditions required for glycolysis points towards how cancer can develop. In a situation of low oxygen tension, moist dark environment, low energy frequency, access to excess glucose and iron, these factors all play a part in developing an aerotolerant anaerobe. Concurrent with Otto Warburg's research, Japanese researchers identified another key fact—that these sick cells can secrete nagalase (Alpha-N-acetylgalactosaminidase), which "cloaks" the cells from immune system recognition and repels phagocytes (like a hormonal trophoblastic cell). The combination of these observations led people to conclude that nagalase production allowed cancer cells to thrive. Permanent conversion to anaerobic fermentation metabolism combined with nagalase release made cancer cells cloaked from immune system surveillance and, thus, could not be eliminated.

This information opened the door for changing our thinking about how to treat cancer from many different perspectives besides poisoning, cutting, or burning with radiation. Specifically, if you want to kill a cancer cell just destroy the environment it needs to thrive and create metabolic tricks "that will enter only sick cells and wreak havoc on their metabolism."

Salicinium is the Trojan horse for cancer cells. In 2005, Joe Brown collaborated with Professor Darrell Lemaire, head of the Department of Chemistry at the University of Nevada, Reno, to investigate the potential for phytochemicals. In the early 1970s, Japanese researchers demonstrated the anti-tumor effects of benzaldehyde via several different mechanisms. Joe Brown and his team created a stable glucoside/complex glycome of 4-hydroxy-benzaldehyde extracted from the plant *Helicia nilagirica* (from fig) and conjugated it to a glucose molecule (salicinium).

Indirect and Direct Actions of Salicinium

Anaerobic metabolizing cells are "needy" for excess glucose to fuel their energy inefficient anaerobic metabolism. To that end, the GLUT transport "pores" are overly expressed on the surface of cancer cells to take in as much glucose as possible. Thus a benzaldehyde ring, which would not normally penetrate a cell, enters readily because it is conjugated with glucose. Once inside the cell, glucose is removed and used as an energy substrate.

Benzaldehyde is now able to donate its hydrogen and complexes with NADPH to form NADP-benzaldehyde. This reaction blocks forward progression of glycolysis, and changes intracellular pH just enough to cause quantum change in intracellular energy, which blocks acid extrusion and lactate formation. In this manner, these sick cells starve and lack energy to produce nagalase, the fibrin coat that "cloaks" cancer cells from recognition by macrophages.

The intracellular pH changes that occuralso destroy mitochondrial function by causing their membranes to become "leaky."

Another factor of great interest is that the benzaldehyde ring causes a reduction of phosphorylation of the "hub signaling protein 14-3-3ζ [14-3-3 zeta]." The 14-3-3 ligand has a number of differential configurational states that regulate all normal cell functions. In cancer cells a mutant form of 14-3-3ζ is present. When benzaldehyde reduces the phosphorylation of 14-3-3ζ, its interaction with many "client proteins" is blocked—e.g., familiar mutations mTOR, c-RAD, STAT3, FOXO, NFKβ, RicToR, and TGF-β to name a few.[1] It would appear that as a result of these observations salicinium does more than disrupt glycolysis, starve cancer cells, and block nagalase production. It may well be able to prevent expression and function of many mutational pathways and ultimately cause apoptosis from even more avenues.

The topic of studying 14-3-3ζ is an active area for new ongoing research. Discoveries about the complex show 14-3-3ζ is implicated in causing chemoresistance and, thus, contribute to poor patient outcomes. Specifically, 14-3-3ζ overexpression confers chemoresistance and promotes oncogenic pathways PI3k/AKT, IGF-IR, ERK/MAPK, TGF-Beta, Beta-Catenin (related to WnT) and h-TERT in breast, lung, multiple myeloma, head and neck, glioblastoma. The question that needs to be answered *in vitro* is "does salicinium show specificity for multiple 14-3-3 isoforms and bind to phosphate binding pockets or to the divergent to C-terminal tail of different 14-3-3 isoforms." That results in blocking these mutational functions.

Salicinium can cross the blood-brain barrier.

Administration of IV Salicinium

- 3 grams per dose daily in 5 ccs in DMSO added to 0.9 NS 250 ccs over a span of 45 minutes-1 hour.
- Advise patient they will exude an odd/strong odor during the IV saturation phase, which disappears when patient converts to Orasal.

- IVs must be given five days per week consecutively, with Orasal on weekends, for a minimum of three weeks to ensure maximum tissue saturation before converting to oral only.
- Patient should follow accompanying protocol with pHenomenal, Impact, and React (https://forperfectbalance.com) daily.
- Orasal alone without IV loading may not give a significant result in adults.
- Avoid ozone, H2O2, DCA (dichloroacetate), arteminisin, high-dose vitamin C, SOD (superoxide dismutase), high-dose curcumin on salicinium protocol.
- Keep in mind salicinium is a food additive, a natural phytochemical and not chemotherapy.

Salicinium Application Do's and Don'ts

- Due to saturation of bone, tissue, and lymph nodes, the IV salicinium is an excellent option to combine with IPT for bone metastases and hematologic origin cancers
- Remember, tumor mass may dissolve, soften, and/or puff up as it is dying. Thus, palpable masses may not diminish in size and yet microscopically die on pathology analysis.
- Due to the metabolic effects on other sick cells that predispose a patient to developing or perpetuating cancer, Salicinium is an effective adjuvant for patients with EBV, HPV, HSV, parasites, fungus, and candida.
- If possible, obtain a PET/CT scan prior to treatment to assess intensity of tumor activity. Be aware, subsequent scans may not show full resolution SUV because even dying tumors may show metabolic activity. The key is that the SUV should decrease with treatment.
- Salicinium can be an excellent protocol for preoperative optimization of a cancer patient having surgical removal of the tumor. Salicinium tends to cause the tumor to coalesce and localize to allow resection with clear margins.
- Salicinium can be used alone or in combination with other natural agents.
- If angio-embolization of a tumor mass is planned, do not use salicinium before the procedure because feeding vessels may become tortuous and cannot be catheterized.

Clinical Cases

Stage I & II: Utilize the opportunity to treat pre-op and post-op, when desired, to "shrink" a large lesion prior to surgery. Treating the whole abnormal metabolic environment includes viral infections.

Fifty-eight-year-old female, with triple negative breast cancer, 5 cm lesion with two satellite lesions (1cm+2cm) plus positive axillary adenopathy on pre-op. Treated with six weeks of IV salicinium and IV mistletoe, interspersed with four sessions of AIPT. At operation (total mastectomy, no immediate reconstruction, limited axillary dissection). Pathology revealed 0.7 cm residual lesion in the breast plus all nodes negative in the axilla. Patient to complete post-op IV salicinium and IV mistletoe plus two more AIPT sessions before transitioning to Orasal/SubQ Mistletoe/herbal treatments.

Stage III: 45-year-old GoPo, ER-, PR-, HER2 Neu+ breast cancer with positive axillary nodes. Palpable 4 cm tumor disappeared completely with salicinium, mistletoe, IPT, and herceptin. Pathology at the operation showed no carcinoma, residual DCIS with negative sentinel lymph nodes after 12 months of herceptin. The patient is still disease free at three years.

Stage IV: Stage IV breast cancer diagnosed in 2015. No surgery. No chemotherapy. No ERT. Large bilateral axillary tumors (8×7 cm) and 8 liters ascites weekly (due to 2 small (2 cm) peritoneal implants). Patient had received >20 high-dose infusions of IV vitamin C, which had no positive effect on the tumors, and only increased ascites production. After five weeks of IV salicinium and IV mistletoe, bilateral axillary tumors were 100% reabsorbed, breast tumor volume shrank >50%, and ascites was gone. F/U PET Scan showed no SUV in right breast and low residual SUV in left breast. Patient is alive and well on Orasal and subQ mistletoe to date.

References

1. Pennington KL, et al. The Dynamic and Stress-Adaptive Signaling Hub of 14-3-3: Emerging Mechanisms of Regulation and Context-Dependent Protein–Protein Interactions. *Nature News*, 18 June 2018, https://www.nature.com/articles/s41388-018-0348-3.
2. Jonathan Collin <townsendpublisher1@yahoo.com>

Virginia Von Schaefer, MD, relies on more than 30 years of clinical experience in general and trauma surgery, endocrinology, and oncology to create strategies to solve her patients' complex medical problems. Her background in cell biology and biochemistry at Columbia University in New York City, established a special problem-solving mindset and framework that allows her to integrate "the best of both worlds" of conventional medical and surgical practice, along with an understanding of the fundamental biochemical/scientific basis for all current modalities. Continued study and shared knowledge with colleagues make it possible to evaluate and integrate the ever evolving, new advances for optimal patient care.
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