Iodine Treats Breast Cancer, Overwhelming Evidence

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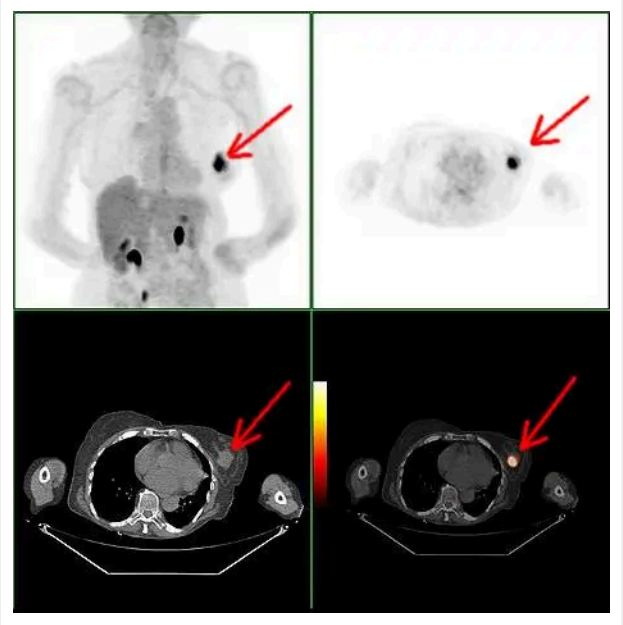


— Arrow points to Breast Cancer on PET SCAN

Iodine Treats Breast Cancer, the Overwhelming Evidence

by Jeffrey Dach MD

This article is Part Two of a series. For Part One , Click Here.

Spontaneous Regression of Breast Cancer

David Brownstein MD reports three cases of spontaneous regression of breast cancer after women take iodine supplementation.(1) (This is reported on page 63 of the Iodine Book by David Brownstein MD.)

Joan, an English Teacher

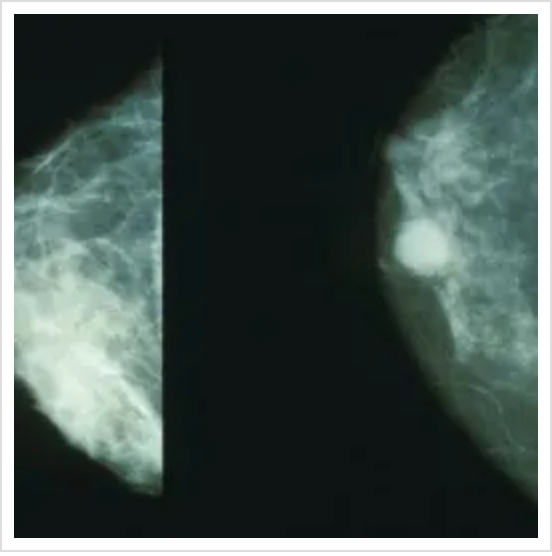
The first patient, Joan a 63 year old English teacher, was diagnosed with breast cancer in 1989, declined conventional treatment, and took 50 mg per day of Iodoral, (Iodine). Six weeks later, a PET scan (left image) showed, "all of the existing tumors were disintegrating". *Upper Left Image: Upper two frames is a PET scan showing breast cancer)(red arrows). Lower two frames is a CAT scan showing enhancing breast cancer mass, red arrow. Courtesy of Wikimedia Commons.*

Delores

The second patient, 73 year old Delores, was diagnosed with breast cancer in 2003. She declined conventional treatment with radiation and chemotherapy. Instead, Dolores took 50 mg of Iodoral daily. A follow up ultrasound of the breast 18 months later showed," It appears that these malignancies have diminished in size since the last examination. Interval improvement is definitely seen," Two years later a follow up mammogram and ultrasound failed to show any abnormality and were read by the radiologist as normal.

Joyce

The third patient, 52 year old Joyce was diagnosed with breast cancer two years prior (left image), and started on Iodoral 50 mg per day. Three years after starting Iodoral, her follow up mammograms and ultrasound exams show decreasing size of the tumor with no progression.(1)



Left Image: Mammogram showing breast cancer courtesy of wikimedia commons.

Iodine Deficiency Causes Breast Cancer – The Overwhelming Evidence

Human Studies of Areas with Low Iodine

Iodine deficiency is associated with a higher rate of goiter and breast cancer. Similarly, higher dietary Iodine intake is associated with less goiter and breast cancer. For example, Japan has the highest dietary intake of iodine (13

mg per day), and the lowest rates for goiter and breast cancer. However, when Japanese women immigrate and change dietary intake of Iodine to the lower 150 mcg/day in America, breast cancer rates increase.(1)

Iceland is another country with high Iodine intake and low rates for goiter and breast cancer. The high dietary iodine came from the fishing industry before WWI. In those days, the fish meal was fed to dairy cows providing milk with high iodine content. After WWI, the fish meal was eliminated from the dairy cows, and breast cancer rates soared ten-fold. (2)

Animal studies

Iodine deficient diets in animals induces breast cancer and goiter.(1)

Iodine Research from Mexico, India and Japan.

India

The Shrivastava group in India reported molecular iodine induces apoptosis (programmed cell death) in human breast cancer cell cultures. *"Iodine showed cytotoxic effects in the cultured human breast cancer cells".* (3)

Mexico

From Mexico, the Carmen Aceves Velasco Group reported Iodine to be safe, with no harmful effects on thyroid function, and an anti-proliferative effect on human breast cancer cell cultures. (5)(6)(7) Their 2009 paper reported the mechanism by which Iodine works as an anti-cancer agent. Iodine binds to membrane lipids called lactones forming iodo-lactones which regulate apoptosis (programmed cell death). Iodine causes apoptosis which makes cancer cells undergo programmed cell death.(4) Dr. Aceves concluded that continuous molecular iodine treatment has a

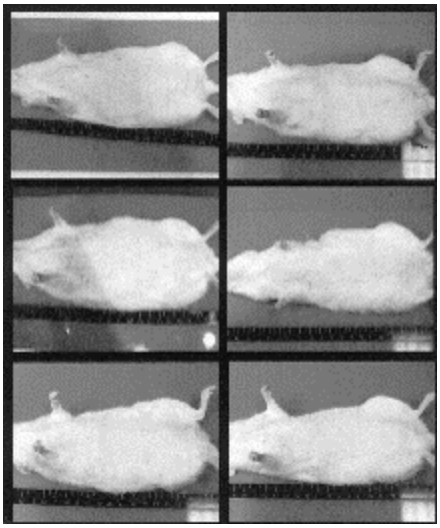
> "*potent antineoplastic effect*" on the progression of mammary cancer. (10)

Japan

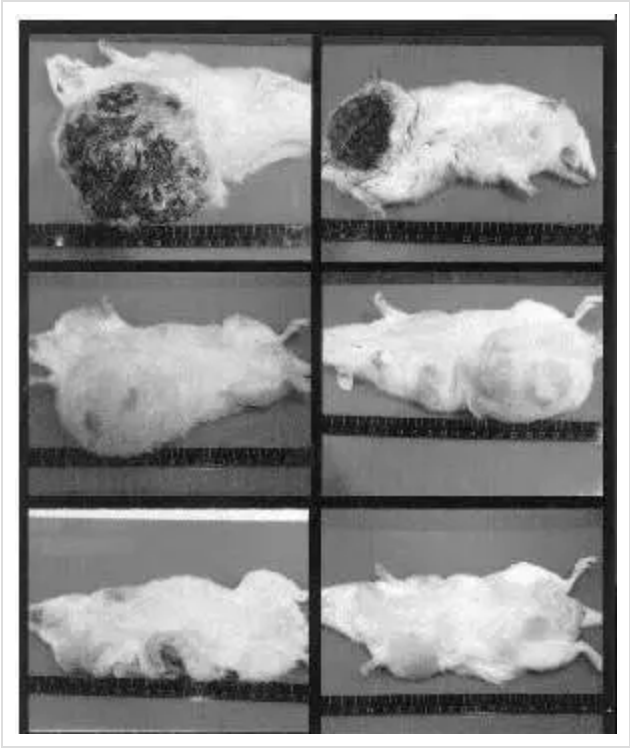
From Japan, Dr Funahashi reported a common seaweed food containing high iodine content is more beneficial than chemotherapy on breast cancer . He found that

> *"administration of Lugol's iodine or iodine-rich Wakame seaweed to rats treated with the carcinogen dimethyl benzanthracene suppressed the development of mammary tumors. The same group demonstrated that seaweed induced apoptosis in human breast cancer cells with greater potency than that of fluorouracil, a chemotherapeutic agent used to treat breast cancer."*(8)

Fig 2 from Funahashi 2001. Breast cancer induced in rats with DMBA a carcinogenic chemical.



Above Images are rats pre-treated with Iodine (mekabu seaweed) showing no breast cancer tumors. Below Images are untreated (Control) rats with large breast cancer tumors visible at the (red arrows) . From Funahashi_2001_Jpn_Journal_Cancer_Research (8)



Mechanism of Action-Altering Gene Expression

A 2008 paper by Bernard A. Eskin MD showed that Iodine actually altered gene expression in breast cancer cells, inducing programmed cell death. (9)

Lung Cancer and Iodine

A 2003 study by Ling Zhang showed that molecular Iodine caused lung cancer cells to undergo programmed cell death (apoptosis). These lung cancer cells had been genetically modified to increase iodine uptake.(12)

Interestingly, a 1993 case report describes spontaneous remission of lung cancer in a patient incidentally treated with Amiodorone which contains iodine (about 9 mg per day)(13)

Using Adjunctive Iodine Combined with Chemotherapy

A 2013 breast cancer study in mice by Dr Alfaro shows synergy of molecular iodine combined with conventional chemotherapy (doxorubicin). (38) He writes:

> *The DOX-I2 (Doxorubicin / Iodine) combination exerts antineoplastic, chemosensitivity, and cardioprotective effects and could be a promising strategy against breast cancer progression.(38)*

In 2018, Dr Zambrano-Estrada studies the Iodine Doxorucin combination in dogs with canine mammary carcinoma finding excellent synergy. (40) He writes:

> *The mDOX+I2 (Doxorubicin .Iodine) scheme improves the therapeutic outcome, diminishes the invasive capacity, attenuates the adverse events and increases disease-free survival.* ***These data led us to propose mDOX+I2 as an effective treatment for canine mammary cancer.****(41)*

A 2019 Breast Cancer study by Dr Mendieta using both in vitro (cell proliferation and invasion assay) and in vivo (xenografts of athymic nude mice) find results so promising, Dr Mendieta proposes Iodine as adjuvant treatment for breast cancer. (31) He writes:

> *I_2 ,Molecular Iodine, decreases the invasive potential of a triple negative basal cancer cell line, and under in vivo conditions the oral supplement of this halogen activates the antitumor immune response, preventing progression of xenografts from laminal and basal mammary cancer cells. These effects allow us to* ***propose iodine supplementation as a possible adjuvant in breast cancer therapy.*** *(31)*

Adjuvant Treatment with Iodine in Breast Cancer

In 2019 Dr Moreno-Vega does a randomized human study on adjuvant use of iodine with chemotherapy for breast cancer.(36) Thirty women were in early stage, and thirty women advanced stage breast cancer. He writes:

> ***Five-year disease-free survival rate*** *was significantly higher in patients treated with the I2 supplement before and after surgery compared to those receiving the supplement only after surgery* ***(82% versus 46%). I2-treated tumors exhibit less invasive potential, and significant increases in apoptosis, estrogen receptor expression, and immune cell infiltration.*** *Transcriptomic analysis indicated* ***activation of the antitumoral immune response.*** *The results led us to register a phase III clinical trial to analyze chemotherapy + I2 treatment for advanced breast cancer.(36)*

Iodine Combined with Zoledronic Acid for Breast Cancer Bone Metastasis

Breast cancer has a predilection for metastatic spread to bone, frequently the ribs. This is routinely treated by mainstream oncology with an IV bisphosphonate drug called Zolendronate. This 2016 study Dr Tripathi uses a mouse xenograft model to show molecular Iodine (Iodoral) has good synergy when combined with Zoledronate. (41). He writes:

We analyzed the effect of combination of I2 (molecular iodine) with Zol (Zolendronate) as a potent adjuvant therapeutic agent for triple negative breast cancer cells (MDA-MBA-231) and in the mice model of breast cancer. …We report that Zol potentiates the efficacy of I2 by inducing non-mitochondrial intrinsic apoptosis by increasing intracellular calcium and ER stress. We show that MDA-MB-231cells register minimal hypodiploidy in response to individual treatment with either I2 or Zol, but synergistically enhances apoptosis when given in combination. Similar potentiating effect as reflected by enhanced apoptotic index on I2-mediated cell death was also reported in these cells by addition of ***chloroquine*** *(12) and by addition of* ***doxorubicin*** *in other animal tumor models and cancer cells (11,19).*

In Conclusion: Current research suggests molecular Iodine as treatment for all patients with breast cancer. (10) (11) Other cancers such as lung and prostate may also benefit. Iodine my serve as adjunct along with conventional chemotherapy and/or with IV Zoledronate. Further research on Iodine for breast cancer should receive top priority for NIH funding.

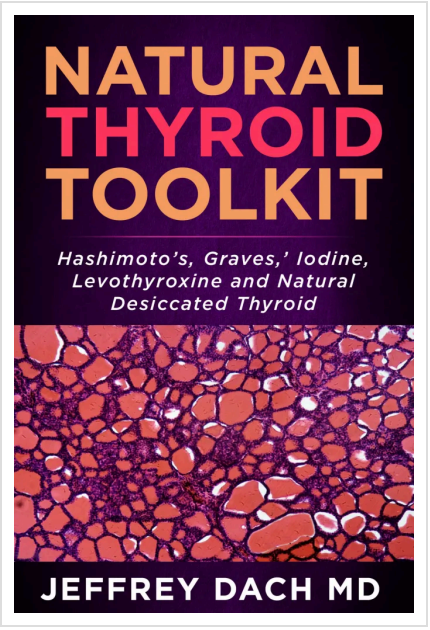
How to Obtain Iodine Tablets

Iodine (Iodoral) is available on the internet without a prescription.

Financial **Disclosure:** The author has no financial interest in the Iodine Book mentioned above, or in any company that manufactures Iodine Supplements.

Disclaimer: This article is not intended as medical advice. Make sure to discuss with your doctor and follow your own personal doctor's recommendations when contemplating any changes to your diet, medications, or medical treatment programs.

Click Here for WHO Iodine Testing Guidelines: Iodine Guidlines: WHO_Urinary Excretion 2013 World Health Organization



Natural Thyroid Toolkit

If you liked this article, you might like my new book, **Natural Thyroid Toolkit** available on Amazon. If you purchase a book, remember to leave a favorable review. That would be much appreciated. See the book cover, left image.

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Links and References

1) Iodine, Why You Need It , Why You Can't Live Without It, by David Brownstein MD
Fourth Edition 2009, Medical Alternatives Press.

2) Breast Cancer and Iodine by David M Derry MD PhD 2001 Trafford.

Human Breast Cancer – India 2006 full text

3) Shrivastava, Ashutosh, et al. "Molecular iodine induces caspase-independent apoptosis in human breast carcinoma cells involving the mitochondria-mediated pathway." Journal of Biological Chemistry 281.28 (2006): 19762-19771.

The iodine-induced apoptotic mechanism was studied in MCF-7 cells. DNA fragmentation analysis confirmed internucleosomal DNA degradation. Terminal deoxynucleotidyl transferase-mediated dUTP nick-end labeling established that iodine induced apoptosis in a time- and dose-dependent manner in MCF-7 cells.

We propose a detailed mechanism of the molecular iodine (I2)-induced apoptosis in human breast cancer cells that may explain iodine-induced breast cancer regression in experimental rat models as well as beneficial effects observed in human fibrocystic breast subjects (3-6). Iodine showed cytotoxic effects in the cultured human breast cancer cells

C Aceves, Arroyo-Helguera

4) Aceves, Carmen, et al. "Antineoplastic effect of iodine in mammary cancer: participation of 6-iodolactone (6-IL) and peroxisome proliferator-activated receptors (PPAR)." Molecular Cancer 8.1 (2009): 1-9.

This report confirms our previous observations that I2 treatment reduces mammary cancer incidence [7], decreases the proliferative rate (PCNA), and induces apoptosis (TUNEL and caspases) in cancerous mammary cells in vitro [14,15] or in vivo without any secondary adverse effect on the thyroid or general health . …Our previous observation that tumor growth resumes if I2 treatment is suspended. [7]. In conclusion, these data support our notion that I2 supplement could be an adjuvant in the therapy of mammary cancer, where the high concentration of AA characteristic of tumoral cells serves as substrate to form 6-IL, which in turn triggers the activation of apoptotic and anti-invasive pathways by modulating PPAR receptors.

Safety of Molecular Iodine

5) Anguiano, B., et al. "Uptake and gene expression with antitumoral doses of iodine in thyroid and mammary gland: evidence that chronic administration has no harmful effects." Thyroid: official journal of the American Thyroid Association 17.9 (2007): 851. Uptake and gene expression with antitumoral doses of iodine in thyroid and mammary gland no harmful effects Anguiano American Thyroid Association 2007

Several studies have demonstrated that moderately high concentrations of molecular iodine (I2) diminish the symptoms of mammary fibrosis in women, reduce the occurrence of mammary cancer induced chemically in rats (50–70%), and have a clear antiproliferative and apoptotic effect in the human tumoral mammary cell line MCF-7.

Nevertheless, the importance of these effects has been underestimated, in part because of the notion that exposure to excess iodine represents a potential risk to thyroid physiology. In the present work we demonstrate that uptake and metabolism of iodine differ in an organ-specific manner and also depend on the chemical form of the iodine ingested (potassium iodide vs. I2). Further, we show that a moderately high I2 supplement (0.05%) causes some of the characteristics of the ''acute Wolff-Chaikoff effect''; namely, it lowers expression of the sodium=iodide symporter, pendrin, thyroperoxidase (TPO), and deiodinase type 1 in thyroid gland without diminishing circulating levels of thyroid hormone.

Finally, we confirm that I2 metabolism is independent of TPO, and we demonstrate that, at the doses used here, which are potentially useful to treat mammary tumors, chronic I2 supplement is not accompanied by any harmful secondary effects on the thyroid or general physiology. Thus, we suggest that I2 could be considered for use in clinical trials of breast cancer therapies

I2 increases expression of NIS, PEN, and lactoperoxidase (LPO) in tumoral mammary tissue without any alteration in thyroid physiology. These data indicate that the uptake and metabolism of iodine are organ-specific and differ depending on the chemical form in which it is ingested, and they provide additional evidence that a chronic, moderately high I2 supplement causes no harmful secondary effects on health (e.g., body weight, thyroid

economy, or reproductive cycle). Thus, we propose that I2 supplementation should be considered for use in clinical trials of breast cancer therapies.

Breast Cancer Cell Line and Iodine

6) Arroyo-Helguera, O., et al. “Uptake and antiproliferative effect of molecular iodine in the MCF-7 breast cancer cell line.” Endocrine-related cancer 13.4 (2006): 1147-1158.

In conclusion, these results demonstrate that I2 uptake does not depend on NIS or PDS; they suggest that in mammary cancer cells, I2 is taken up by a facilitated diffusion system and then covalently bound to lipids or proteins that, in turn, inhibit proliferation.

7) Arroyo-Helguera, O., et al. “Signaling pathways involved in the antiproliferative effect of molecular iodine in normal and tumoral breast cells: evidence that 6-iodolactone mediates apoptotic effects.” Endocrine-related cancer 15.4 (2008): 1003-1011.

Previous reports have documented the antiproliferative properties of I2 and the arachidonic acid (AA) derivative 6-iodolactone (6-IL) in both thyroid and mammary glands. In this study, we characterized the cellular pathways activated by these molecules and their effects on cell cycle arrest and apoptosis in normal (MCF-12F) and cancerous (MCF-7) breast cells.

Low-to-moderate concentrations of I2 (10–20 μM) cause G1 and G2/M phase arrest in MCF-12F and caspase-dependent apoptosis in MCF-7 cells. In normal cells, only high doses of I2 (40 μM) induced apoptosis, and this effect was mediated by poly (ADP-ribose) polymerase-1 (PARP1) and the apoptosis-induced factor, suggesting an oxidative influence of iodine at high concentrations. Our data indicate that both I2 and 6-IL trigger the same intracellular pathways and suggest that the antineoplasic effect of I2 in mammary cancer involves the intracellular formation of 6-IL. Mammary cancer cells are known to contain high concentrations of AA, which might explain why I2 exerts apoptotic effects at lower concentrations only in tumoral cells.

Seaweed Japan

8) Funahashi, Hiroomi, et al. “Seaweed prevents breast cancer?.” Japanese journal of cancer research 92.5 (2001): 483-487.

To investigate the chemopreventive effects of seaweed on breast cancer, we have been studying the relationship between iodine and breast cancer. We found earlier that the seaweed, wakame, showed a suppressive effect on the proliferation of DMBA (dimethylbenz(a)anthracene)-induced rat mammary tumors, possibly via apoptosis induction. In the present study, powdered mekabu was placed in distilled water, and left to stand for 24 h at 4°C. The filtered supernatant was used as mekabu solution. It showed an extremely strong suppressive effect on rat mammary carcinogenesis when
used in daily drinking water, without toxicity. In vitro, mekabu solution strongly induced apoptosis in 3 kinds of human breast cancer cells. These effects were stronger than those of a chemotherapeutic agent widely used to treat human breast cancer. Furthermore, no apoptosis induction was observed in normal human mammary cells. In Japan, mekabu is widely consumed as a safe, inexpensive food. Our results suggest that mekabu has potential for chemoprevention of human breast cancer.

Iodine Alters Gene Expression in Breast Cancer Cells.

9) Stoddard II, Frederick R., et al. “Iodine alters gene expression in the MCF7 breast cancer cell line: evidence for an anti-estrogen effect of iodine.” International journal of medical sciences 5.4 (2008): 189.
The protective effects of iodine on breast cancer have been postulated from epidemiologic evidence and described in animal models. The molecular mechanisms responsible have not been identified but laboratory evidence suggests that iodine may inhibit cancer promotion through modulation of the estrogen pathway.

To elucidate the role of iodine in breast cancer, the effect of Lugol’s iodine solution (5% I2, 10% KI) on gene expression was analyzed in the estrogen responsive MCF-7 breast cancer cell line. Microarray analysis identified 29 genes that were up-regulated and 14 genes that were down-regulated in response to iodine/iodide treatment. The altered genes included several involved in hormone metabolism as well as genes involved in the regulation of cell cycle progression, growth and differentiation.

Molecular Iodine to Treat Breast Cancer

10) http://findarticles.com/p/articles/mi_pwwi/is_200610/ai_n16809836/
Research Calls for Use of Molecular Iodine to Treat Breast Cancer. Market Wire, October, 2006

11) Cann, Stephen A., Johannes P. Van Netten, and Christiaan van Netten. “Hypothesis: iodine, selenium and the development of breast cancer.” *Cancer causes & control* 11.2 (2000): 121-127. Hypothesis iodine selenium and breast cancer Cann Stephen A Cancer Causes Control 2000

Lung Cancer and Iodine

12) Zhang, Ling, et al. “Nonradioactive iodide effectively induces apoptosis in genetically modified lung cancer cells.” Cancer research 63.16 (2003): 5065-5072.

We assessed a nonradioactive approach to induce apoptosis in non-small cell lung cancer by a novel iodide uptake and retention mechanism. To enhance tumor apoptosis, we transduced non-small cell lung cancer cells with retroviral vectors containing the sodium iodide symporter (NIS) and thyroperoxidase (TPO) genes. Expression of NIS and TPO facilitated concentration of iodide in tumors. As a consequence of the marked increase in intracellular levels of iodide, apoptosis was seen in >95% of NIS/TPO-modified lung cancer cells. Intraperitoneal injection of potassium iodide resulted in significant tumor volume reduction in NIS/TPO-modified tumor xenografts without apparent adverse effects in SCID mice. Iodide induced an increase in the level of reactive oxygen species. Iodide-induced apoptosis is sensitive to N-acetylcysteine inhibition, suggesting an important role by reactive oxygen species in this apoptotic process. In addition, iodide-induced apoptosis is associated with overexpression of CDKN1A (p21/Waf1)and down-regulation of survivin at both mRNA and protein levels. This is the first report demonstrating that a therapeutic dose of nonradioactive iodide has potent efficacy and high selectivity against lung cancer when used in combination with genetic modification of cancer cells to express the NIS/TPOgenes.

Spontanous Remission of Lung Cancer With Iodine Treatment

13) Hercbergs, Aleck, and John T. Leith. “Spontaneous remission of metastatic lung cancer following myxedema coma—an apoptosis-related phenomenon?.” *JNCI: Journal of the National Cancer Institute* 85.16 (1993): 1342-1343.

Patient was incidently treated with Amiodorone which contains iodine 9 mg day

14) Tazebay, Uygar H., et al. “The mammary gland iodide transporter is expressed during lactation and in breast cancer.” Nature medicine 6.8 (2000): 871-878.

15) Venturi, Sebastiano, et al. “Role of iodine in evolution and carcinogenesis of thyroid, breast and stomach.” *Advances in clinical pathology* 4 (2000): 11-18.

16) Venturi, Sebastiano. “Is there a role for iodine in breast diseases?.” The Breast 10.5 (2001): 379-382.

Iodine for breast pain

17) Kessler, Jack H. “The effect of supraphysiologic levels of iodine on patients with cyclic mastalgia.” *The breast journal* 10.4 (2004): 328-336. Effect of Supraphysiologic Levels of Iodine on Patients with Cyclic Mastalgia Kessler Jack H Breast Journal 2004

Patients recorded statistically significant decreases in pain by month 3 in the 3.0 and 6.0 mg/day treatment groups, but not the 1.5 mg/day or placebo group; more than 50% of the 6.0 mg/day treatment group recorded. A clinically significant reduction in overall pain. All doses were associated with an acceptable safety profile. No dose-related increase in any adverse event was observed.

Peter PA Smyth

18) Smyth, Peter. “The thyroid, iodine and breast cancer.” *Breast Cancer Research* 5.5 (2003): 1-4.

Previous studies [2,3] that showed both increased goiter rates and increased prevalence of thyroid enlargement by ultrasound in patients with breast cancer [4].

It has been postulated that formation of iodolipids such as iodolactones or iodoaldehydes represents a form of thyroidal autoregulation [26], which may be the mode of action of iodide inhibition of thyroid function in the Wolff–Chaikoff effect [27-29]. In addition to their role in inhibiting thyroid function, these compounds may act as antiproliferative agents in the thyroid [26].

An anticarcinogenic role for iodine in experimental animals was suggested by the work of Funahashi and coworkers [33], who found that administration of Lugol’s iodine or iodine-rich Wakame seaweed to rats treated with the carcinogen dimethyl benzanthracene suppressed the development of mammary tumours. In further studies [34], the same group demonstrated that seaweed induced apoptosis in human breast cancer cells with greater potency than that of fluorouracil, a chemotherapeutic agent used to treat breast cancer. This finding led the authors to speculate that ‘seaweed may be applicable for prevention of breast cancer’. This hypothesis is in accord with the relatively low breast cancer rate reported in Japan [35], where the normal diet is seaweed rich, and with increasing breast cancer rates in Japanese women who emigrate [36] or consume a western style diet [37]. The frequent coexistence of iodine and selenium deficiencies and the importance of replacing both to maintain thyroid function is well established

Iodine Resolves Fibrocystic Breast Disease – Ghent and Eskin 1993

19) Ghent, W. R., et al. “Iodine replacement in fibrocystic disease of the breast.” Canadian journal of surgery. Journal canadien de chirurgie 36.5 (1993): 453-460..

OBJECTIVE: To determine the response of patients with fibrocystic breast disease to iodine replacement therapy. CONCLUSIONS: Molecular iodine is nonthyrotropic and was the most beneficial.

Iodine Project

20) Guy Abraham Iodine Project Optimox Resources for Iodine Research

George Flechas MD

21) David Brownstein MD Iodine Book

22) Jacob Teitelbaum MD on Iodine Deficiency An Old Epidemic is Back

23) Extrathyroidal Benefits of Iodine by Donald W. Miller, Jr., M.D.

Iodine Links

24) Breast Cancer Choices.org
Iodine and the Breast IODINE AND THE BREAST

25) http://www.hacres.com/diet/articles/Iodine.pdf
Recent Advances in Iodine Nutrition by Michael Donaldson, PhD

26) http://www.thorne.com/altmedrev/.fulltext/13/2/116.pdf
Iodine: Deficiency and Therapeutic Considerations Lyn Patrick, ND
Alternative Medicine Review Volume 13, Number 2 2008

27) Dwyer, Roisin M., et al. “In vivo radioiodide imaging and treatment of breast cancer xenografts after MUC1-driven expression of the sodium iodide symporter.” Clinical Cancer Research 11.4 (2005): 1483-1489.

Results: **A 58-fold increase in iodide uptake was observed in infected MUC1-positive T47D cells with no significant increase observed in MUC1-negative MDA-MB-231 cells** or in cells infected with the control virus. The in vivo study yielded clear images of Ad/MUC1/NIS-infected tumor xenografts using Iodine-123. Administration of a therapeutic dose of Iodine-131 resulted in an 83% reduction in tumor volume, whereas control tumors continued to increase in size ($P < 0.01$).

28) Carroll, Candace E., et al. “Curcumin delays development of MPA-accelerated DMBA-induced mammary tumors.” *Menopause (New York, NY)* 17.1 (2010): 178.

29) Aldaz, C. Marcelo, et al. “Medroxyprogesterone acetate accelerates the development and increases the incidence of mouse mammary tumors induced by dimethylbenzanthracene.” *Carcinogenesis* 17.9 (1996): 2O69-2O72.Medroxyprogesterone MPA accelerates mouse mammary tumors induced by dimethylbenzanthracene DMBA Aldaz Marcelo

30) Lanari, Claudia, et al. “The MPA mouse breast cancer model: evidence for a role of progesterone receptors in breast cancer.” *Endocrine-related cancer* 16.2 (2009): 333-350. The MPA mouse breast cancer model Lanari Claudia 2009

31) Mendieta, Irasema, et al. “Molecular iodine exerts antineoplastic effects by diminishing proliferation and invasive potential and activating the immune response in mammary cancer xenografts.” *BMC cancer* 19.1 (2019): 261.

The immune system is a crucial component in cancer progression or regression. Molecular iodine (I_2) exerts significant antineoplastic effects, acting as a differentiation inductor and immune modulator, but its effects in antitumor immune response are not elucidated.

METHODS:
The present work analyzed the effect of I_2 in human breast cancer cell lines with low (MCF-7) and high (MDA-MB231) metastatic potential under both in vitro (cell proliferation and invasion assay) and in vivo (xenografts of athymic nude mice) conditions.

RESULTS:
In vitro analysis showed that the 200 μM I_2 supplement decreases the proliferation rate in both cell lines and diminishes the epithelial-mesenchymal transition (EMT) profile and the invasive capacity in MDA-MB231. In immunosuppressed mice, the I_2 supplement impairs implantation (incidence), tumoral growth, and proliferation of both types of cells. Xenografts of the animals treated with I_2 decrease the expression of invasion markers like CD44, vimentin, urokinase plasminogen activator and its receptor, and vascular endothelial growth factor; and increase peroxisome proliferator-activated receptor gamma. Moreover, in mice with xenografts, the I_2 supplement increases the circulating level of leukocytes and the number of intratumoral infiltrating lymphocytes, some of them activated as CD8+, suggesting the activation of antitumor immune responses.

CONCLUSIONS:
I_2 decreases the invasive potential of a triple negative basal cancer cell line, and under in vivo conditions the oral supplement of this halogen activates the antitumor immune response, preventing progression of xenografts from laminal and basal mammary cancer cells. These effects allow us to propose iodine supplementation as a possible adjuvant in breast cancer therapy.

32) Rappaport, Jay. “Changes in dietary iodine explains increasing incidence of breast Cancer with distant involvement in young women.” Journal of Cancer 8.2 (2017): 174
The incidence of breast cancer with distant involvement at diagnosis is increasing in young women, age 25-39, possibly at an accelerating rate, as previously demonstrated by Johnson et al.1 This disturbing trend was also observed in women age 40-54, albeit to a letter extent. Understanding the causes for these changes in cancer demographics is particularly important in view of the young age of these women, as well as the poor survival rate for women with distant metastatic breast disease. There have been explanations proposed in several letters, including vaccination 2, advanced-age at first pregnancy 3 as well as folate supplementation 4, however these explanations have not been particularly satisfying. In response, the original authors 5 suggested causal association need explain: 1) why a proposed agent or risk factor would cause advanced disease, 2) why it would preferentially affect the youngest women, and 3) how temporal trends in the proposed causative agent have changed since the mid-1970s 5. The hypothesis that iodine deficiency in the United States, plays a role in the

increased incidence of breast cancer with distant involvement, is discussed here in the context of these three factors.

33) Aceves, Carmen, Brenda Anguiano, and Guadalupe Delgado. "Is iodine a gatekeeper of the integrity of the mammary gland?." *Journal of mammary gland biology and neoplasia* 10.2 (2005): 189-196.

34) Anguiano, Brenda, and Carmen Aceves. "Iodine in mammary and prostate pathologies." Current Chemical Biology 5.3 (2011): 177-182.

35) How Molecular Iodine Attacks Breast Cancer
Oncology Times: December 25, 2016 – Volume 38 – Issue 24 – p 34

36) Moreno-Vega, Aura, et al. "Adjuvant effect of molecular iodine in conventional chemotherapy for breast cancer. Randomized pilot study." Nutrients 11.7 (2019): 1623.

This study analyzes an oral supplement of molecular iodine (I2), alone and in combination with the neoadjuvant therapy 5-fluorouracil/epirubicin/cyclophosphamide or taxotere/epirubicin (**FEC/TE**) in women with Early (stage II) and Advanced (stage III) breast cancer. In the Early group, 30 women were treated with **I2 (5 mg/day)** or placebo (colored water) **for 7–35 days before surgery.** For the **Advanced group, 30 patients received I2 or placebo, along with FEC/TE treatment**. After surgery, all patients received **FEC/TE + I2 for 170 days**. **I2 supplementation showed a significant attenuation of the side effects and an absence of tumor chemoresistance.** The control, I2, FEC/TE, and FEC/TE + I2 groups exhibited response rates of 0, 33%, 73%, and 100%, respectively, and a pathologic complete response of 18%, and 36% in the last two groups. **Five-year disease-free survival rate** was significantly higher in patients treated with the I2 supplement before and after surgery compared to those receiving the supplement only after surgery **(82% versus 46%). I2-treated tumors exhibit less invasive potential, and significant increases in apoptosis, estrogen receptor expression, and immune cell infiltration.** Transcriptomic analysis indicated **activation of the antitumoral immune response.** The results led us to register a phase III clinical trial to analyze chemotherapy + I2 treatment for advanced breast cancer.

37) Cuenca-Micó, Olga, et al. "Effects of Molecular Iodine/Chemotherapy in the Immune Component of Breast Cancer Tumoral Microenvironment." Biomolecules 11.10 (2021): 1501.

Molecular iodine (I2) induces apoptotic, antiangiogenic, and antiproliferative effects in breast cancer cells. Little is known about its effects on the tumor immune microenvironment. We studied the effect of oral (5 mg/day) I2 supplementation alone (I2) or together with **conventional chemotherapy (Cht+I2)** on the immune component of breast cancer tumors from a previously published pilot study conducted in Mexico. RNA-seq, I2 and Cht+I2 samples showed significant increases in the expression of Th1 and Th17 pathways. Tumor immune composition determined by deconvolution analysis revealed significant increases in M0 macrophages and B lymphocytes in both I2 groups. Real-time RT-PCR showed that I2 tumors overexpress T-BET ($p = 0.019$) and interferon-gamma (IFNγ; $p = 0.020$) and **silence tumor growth factor-beta (TGFβ;** $p = 0.049$), whereas in Cht+I2 tumors, GATA3 is silenced ($p = 0.014$). Preliminary methylation analysis shows that I2 activates IFNγ gene promoter (by increasing its unmethylated form) and silences TGFβ in Cht+I2. In conclusion, our data showed that I2 supplements **induce the activation of the immune response and that when combined with Cht, the Th1 pathways are stimulated.** The molecular mechanisms involved in these responses are being analyzed, but preliminary data suggest that methylation/demethylation mechanisms could also participate.

38) Alfaro, Yunuen, et al. “Iodine and doxorubicin, a good combination for mammary cancer treatment: antineoplastic adjuvancy, chemoresistance inhibition, and cardioprotection.” Molecular cancer 12.1 (2013): 1-11. Although mammary cancer (MC) is the most common malignant neoplasia in women, the mortality for this cancer has decreased principally because of early detection and the use of neoadjuvant chemotherapy. Of several preparations that cause MC regression, doxorubicin (DOX) is the most active, first-line monotherapeutic. Nevertheless, its use is limited due to the rapid development of chemoresistance and to the cardiotoxicity caused by free radicals. In previous studies we have shown that supplementation with molecular iodine (I2) has a powerful antineoplastic effect in methylnitrosourea (MNU)-induced experimental models of MC. These studies also showed a consistent antioxidant effect of I2 in normal and tumoral tissues.
Methods

Here, we analyzed the effect of I2 in combination with DOX treatment in female Sprague Dawley rats with MNU-induced MC. In the first experiment (short) animals were treated with the therapeutic DOX dose (16 mg/kg) or with lower doses (8 and 4 mg/Kg), in each case with and without 0.05% I2 in drinking water. **Iodine treatment began on day 0, a single dose of DOX was injected (ip) on day 2, and the analysis was carried out on day 7. In the second experiment (long) animals with and without iodine supplement were treated with one or two injections of 4 mg/kg DOX (on days 0 and 14) and were analyzed on day 56.**

Results **At all DOX doses, the short I2 treatment induced adjuvant antineoplastic effects (decreased tumor size and proliferating cell nuclear antigen level) with significant protection against body weight loss and cardiotoxicity (creatine kinase MB, cardiac lipoperoxidation, and heart damage).** With long-term I2, mammary tumor tissue became more sensitive to DOX, since a single injection of the lowest dose of DOX (4 mg/Kg) was enough to stop tumor progression and a second DOX4 injection on day 14 caused a significant and rapid decrease in tumor size, decreased the expression of chemoresistance markers (Bcl2 and survivin), and increased the expression of the apoptotic protein Bax and peroxisome proliferator-activated receptor type gamma. **Conclusions The DOX-I2 combination exerts antineoplastic, chemosensitivity, and cardioprotective effects and could be a promising strategy against breast cancer progression.**

39) Elliyanti, Aisyah, et al. “An Iodine Treatments Effect on Cell Proliferation Rates of Breast Cancer Cell Lines; In Vitro Study.” Open Access Macedonian Journal of Medical Sciences 8.B (2020): 1064-1070.

BACKGROUND: Iodine can reduce breast tumor progression by mediates an antiproliferative effect.**AIM:** This study aimed to investigate the effect of **iodine (I2), Lugol (I3K)**, and the combination of both on cell proliferation of three different types of breast cancer cell lines.**METHODS:** The samples were MCF7, SKBR3, and MDA-MB 213 cell lines. Cell proliferation rate was measured using colorimetric and clonogenic assays.**RESULTS:** The cell proliferation rate of MDA-MB 231 cells was reduced significantly by treatment I2, I3K, and combination of both with $p = 0.046$, $p = 0.00$, and $p = 0.00$, respectively. In MCF7 cells, I2 reduced the cell proliferation of 54–94% and I3K reduced the proliferation of 74–94%. The effectiveness of I3K treatments in slowing cell proliferation rate was dose-dependent. In SKBR3 cells, **I2 reduced proliferation cell up to 85% and I3K 4%-94% depending on the dose. Clonogenic assay results showed a discontinue of the cell proliferation by all doses of I2 and I3K (10 μM and 20 μM).**

CONCLUSION: Breast cancer cell lines, representing subtypes of luminal A, HER2+, and triple-negative, show an excellent response to iodine treatments and I3K response shows in a dose-dependent manner. Further studies are needed to investigate the effective *in vivo* doses.

40) Zambrano-Estrada, Xóchitl, et al. “Molecular iodine/doxorubicin neoadjuvant treatment impair invasive capacity and attenuate side effect in canine mammary cancer.” BMC veterinary research 14.1 (2018): 1-14.

Mammary cancer has a high incidence in canines and is an excellent model of spontaneous carcinogenesis. Molecular iodine (I2) exerts antineoplastic effects on different cancer cells activating re-differentiation pathways. In co-administration with anthracyclines, I2 impairs chemoresistance installation and prevents the severity of side effects generated by these antineoplastic drugs. This study is a random and double-blind protocol that analyzes the impact of I2 (10 mg/day) in two administration schemes of Doxorubicin (DOX; 30 mg/m2) in 27 canine patients with cancer of the mammary gland. The standard scheme (sDOX) includes four cycles of DOX administered intravenously for 20 min every 21 days, while the modified scheme (mDOX) consists of more frequent chemotherapy (four cycles every 15 days) with slow infusion (60 min). In both schemes, I2 or placebo (colored water) was supplemented daily throughout the treatment.
Results

mDOX attenuated the severity of adverse events (VCOG-CTCAE) in comparison with the sDOX group. The overall tumor response rate (RECIST criteria) for all dogs was 18% (interval of reduction 48–125%), and no significant difference was found between groups. I2 supplementation enhances the antineoplastic effect in mDOX, exhibiting a significant decrease in the tumor epithelial fraction, diminished expression of chemoresistance (MDR1 and Survivin) and invasion (uPA) markers and enhanced expression of the differentiation factor known as peroxisome proliferator-activated receptors type gamma (PPARγ). Significant tumor lymphocytic infiltration was also observed in both I2-supplemented groups. The ten-month survival analysis showed that the entire I2 supplementation (before and after surgery) induced 67–73% of disease-free survival, whereas supplementation in the last period (only after surgery) produced 50% in both schemes.
Conclusions

The mDOX+I2 scheme improves the therapeutic outcome, diminishes the invasive capacity, attenuates the adverse events and increases disease-free survival. These data led us to propose mDOX+I2 as an effective treatment for canine mammary cancer.

41) Tripathi, Ranu, et al. “Zoledronate and molecular iodine cause synergistic cell death in triple negative breast cancer through endoplasmic reticulum stress.” Nutrition and Cancer 68.4 (2016): 679-688.

We analyzed the effect of combination of I2 with Zol as a potent adjuvant therapeutic agent for triple negative breast cancer cells (MDA-MBA-231) and in the mice model of breast cancer. Cell viability, terminal deoxynucleotidyl transferase dUTP nick end labeling staining, Western blotting, real-time PCR, flow cytometry, and other assays were performed for assessing cell death, calcium levels, and migration potential, respectively, in treated cells. The increased caspase 8, increased [Ca(2+)]c levels, and endoplasmic reticulum (ER) stress resulted in apoptosis. Real time and fluorescence-based analysis demonstrated that the combination treatment targets ER Ca(2+) homeostasis chaperons leading to apoptosis. Combination therapy reduces metalloproteinases 2 and 9, inhibits invasion/migration of cells, and prevents growth of tumor in mice. I2 + Zol combination treatment induces synergistic increase in ER-mediated apoptosis, reduces invasion/migration potential of MDA-MB-231 cells, and exhibits antiproliferative property in vivo demonstrating its potential as combination therapy.

To the best of our knowledge, this is the first report on the potential of I2 as an adjuvant combination with Zol for the treatment of metastatic breast cancers

We report that ZoI potentiates the efficacy of I2 by inducing non-mitochondrial intrinsic apoptosis by increasing intracellular calcium and ER stress. We show that MDA-MB-231cells register minimal hypodiploidy in response to individual treatment with either I2 or ZoI, but synergistically enhances apoptosis when given in combination. Similar potentiating effect as reflected by enhanced apoptotic index on I2-mediated cell death was also reported in these cells by addition of chloroquine (12) and by addition of doxorubicin in other animal tumor models and cancer cells (11,19).

41) Nava-Villalba, Mario, and Carmen Aceves. "6-Iodolactone, key mediator of antitumoral properties of iodine." Prostaglandins & other lipid mediators 112 (2014): 27-33.

An iodinated derivative of arachidonic acid, 5-hydroxy-6-iodo-8,11,14-eicosatrienoic acid, □-lactone (6-IL) has been implicated as a possible intermediate in the autoregulation of the thyroid gland by iodine. In addition to antiproliferative and apoptotic effects observed in thyrocytes, this iodolipid could also exert similar actions in cells derived from extrathyroidal tissues like mammary gland, prostate, colon, or the nervous system. In mammary cancer (solid tumors or tumor cell lines), 6-IL has been detected after molecular iodine (I2) supplement, and is a potent activator of peroxisome proliferator-activated receptor type gamma (PPAR□). These observations led us to propose I2 supplement as a novel coadjutant therapy which, by inducing differentiation mechanisms, decreases tumor progression and prevents chemoresistance. Some kinds of tumoral cells, in contrast to normal cells, contain high concentrations of arachidonic acid, making the I2 supplement a potential **"magic bullet"** that enables local, specific production of 6-IL, which then exerts antineoplastic actions with minimal deleterious effects on normal tissues.

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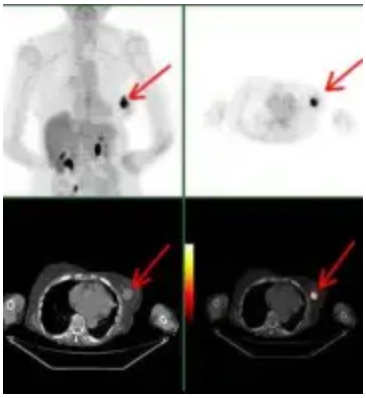

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Iodine Treats Breast Cancer Overwhelming Evidence

Description

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flitesurgn
on **December 28, 2016 at 10:34 PM** said:

Overwhelming evidence? Where? !i didn't see anything that came close to that claim.

Jeffrey Dach MD
on **January 4, 2017 at 1:06 PM** said:
Take a closer look.

Michel de Bruijn
on **July 28, 2017 at 6:50 AM** said:

Ik maak THC en CBD olie zelf, volgens de Simpson methode. Dat doe ik, sinds de kanker van mijn oma. Het bleek toen erg lastig, aan THC olie te komen of tegen absurde prijzen. De overheid zou het daarom meer toegankelijk moeten maken…
Zelf gebruik ik het voor mijn slaapproblemen en mijn partner voor de spierziekte. Het is goed tegen vele ziektes en gewoon plantaardig!
Mocht u ook niet aan de THC of CBD olie kunnen komen, kunt u altijd mailen: mi**********@ho**.nl .
wellicht dat ik u kan helpen ermee

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Pingback: Iodine- the in's the out's the answers? - Advice from Allie

rogersan
on **November 4, 2017 at 3:24 PM** said:

Not sure about the above studies but I use Lugol's iodine for myself all the time with vitamins. My friend came to visit and he used some on some large cancerous looking cysts on his skin. After about 2 weeks the largest one ruptured and drained and the rest have since cleared up. He is a believer too in iodine the miracle cure.

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Jeffrey Dach MD
on **December 27, 2020 at 4:55 AM** said:

Dr Jonathan V. Wright Tahoma Clinic in a video discussion of iodine as breast cancer preventive:
https://tahomaclinic.com/a-drop-a-day-keeps-breast-cancer-away/