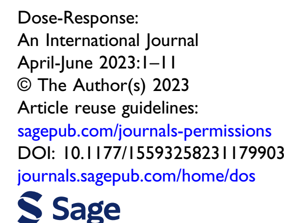
Original Article

Differential In Vivo Effects on Cancer Models by Recorded Magnetic Signals Derived From a Healing Technique


Dose-Response:
An International Journal
April-June 2023:1–11
© The Author(s) 2023
Article reuse guidelines:
sagepub.com/journals-permissions
DOI: 10.1177/15593258231179903
journals.sagepub.com/home/dos

William Bengston[1], Paul Cizdziel[2], Akane Tanaka[3], and Hiroshi Matsuda[4]

Abstract

Previous research on "healing-with-intent" has reasonably demonstrated the validity of the phenomenon at least when a human healer is present and involved. However, in order for healing to be adopted into more conventional therapies, it must be able to be made scalable. The present study tests the effects of a scalable recording of the Bengston Healing Method on 3 cancer models. BalbC mice engrafted with 4T1 breast cancer cells, C57BL mice with melanoma B16 cells, and C3H mice with bladder MBT-2 wells were exposed to a recording of healing intent for 4 hours/day for approximately 1 month. In the breast cancer model, there was significant tumor suppression and a reduction of anemia marker HCT in treated vs control mice. In the melanoma model, there were no significant differences except for a reduction in platelet count among the treated mice. For unknown reasons, tumor growth never became evident in the bladder cancer model. While the effects of the recording seem to vary by model, there appears reason to pursue scalable delivery systems in multiple models and with multiple doses.

Keywords

alternative and complementary medicine, integrative medicine, healing, magnetic signals

Introduction

The father of Western medicine, Hippocrates, recognized that certain individuals appear to be able to heal, and described this as "the force which flows from many people's hands."[1] A list of the methods used by these purported healers have included laying on of hands, prayer, and induced altered states of consciousness, to name a few. Today, of course, these types of purported healing methods would not be widely or officially sanctioned by Western medicine, though anecdotally more physicians and other health care providers are becoming increasingly open to the possibility.

Historically, the source and mechanism of this healing evaded systematic research even as the number of clinical practitioners and cases greatly increased. The foundation of modern Western healing research can be traced to the pioneering experimental work in the 1950s and 1960s by the late biologist Bernard Grad at McGill University. Grad found that selected healers could influence the germination of plant seeds, the growth rate of plants, and the curing of seeds that had been shocked by saline solution. In addition, he was able to measure the ability of healers to reduce goiter and stimulate wound healing in mice.[2–4]

Since Grad's work, there have been innumerable experimental studies of healing, oftentimes categorized by the target of the intended healing. For example, Benor, in one of the first systematic compilations of controlled experimental studies,

[1] St. Joseph's University, Port Jefferson NY, USA
[2] Society for Scientific Exploration, Tokyo, Japan
[3] Division of Animal Life Science, Tokyo University of Agriculture and Technology, Tokyo, Japan
[4] Institute of Agriculture, Tokyo University of Agriculture and Technology, Tokyo, Japan

Received 23 August 2022; accepted 17 May 2023

Corresponding Author:
William Bengston, St Joseph's University, 112 Peninsula Drive, Port Jefferson, NY 11777, USA.
Email: wbengston@sjny.edu

Creative Commons Non Commercial CC BY-NC: This article is distributed under the terms of the Creative Commons Attribution-NonCommercial 4.0 License (https://creativecommons.org/licenses/by-nc/4.0/) which permits non-commercial use, reproduction and distribution of the work without further permission provided the original work is attributed as specified on the SAGE and Open Access pages (https://us.sagepub.com/en-us/nam/open-access-at-sage).

discusses healing action on enzymes, eukaryotic cells in the laboratory, fungi/yeasts, bacteria, plants, single-cell organisms, and animals that have been subjected to controlled study.[5]

Many of the early experimental studies were accompanied by unresolved issues of reliability. Snel, for example, reported significant growth inhibition of mouse leukemia cells in tissue culture but not in attempted replication.[6] Even Grad found that experienced healers were unable to reproduce significant effects on the enzyme trypsin when they were not in the proper frame of mind.[7] This deficiency in replication has not only emboldened skeptics, but has also made widespread application of healing methods sometimes problematic.

Systematic reviews on the quality of healing studies took a leap forward in a seminal 2003 issue of *Alternative Therapies in Health and Medicine.* That issue published the results of a Samueli Institute conference which systematically assessed the quality of healing research up to that date. Crawford et al. reviewed the quality of healing studies in 45 laboratory and 45 clinical studies between 1955 and 2001.[8] Among the conclusions were that laboratory studies tended to be of higher methodological rigor than clinical studies, and specific recommendations were made to advance the field.

In the same issue of *Alternative Therapies in Health and Medicine*, Schlitz et al.[9] summarized replicable effects of healing on enzymes,[10] fungi,[11] yeast,[12] bacteria,[13] cancer cells,[14] and hemolysis of red blood cells under osmotic stress.[15] The authors also made 38 specific recommendations regarding experimental protocols for studying healing, addressing issues of proper randomization, sensory shielding, blinding, and fraud prevention.

More recently, *Global Advancements in Health and Medicine* published papers from a 2014 conference hosted by the Consciousness and Healing Initiative and sponsored by the Miraglow Foundation, the Institute for Noetic Sciences, the Samueli Institute, and the Chopra Foundation.[16] At this conference, a wide variety of researchers examined the implications of healing for the physical, biological, and social sciences, along with methodological assessments of the state of the art.

This conference oftentimes noted the need for more "second order" questions. Instead of the simple first order demonstration of the phenomenon of healing, there needs to be an increase of the proportion of "second order" studies looking for correlates to the healing. These include the use of multiple simultaneous targets, dose response, and the like. For example, Gronowicz et al.[17] assessed the dose dependent effects of Therapeutic Touch on the proliferation of different types of human cells in culture. Fibroblasts, tendon cells (tenocytes), and bone cells (osteoblasts) were treated with therapeutic touch, sham treatment, or no treatment for 10 minutes per treatment with varying frequencies of treatment each week. They found that tenocytes and fibroblasts but not osteoblasts demonstrated significant increases in cell proliferation in the first week of treatment, while osteoblast did so only after 2 weeks of treatment. All 3 cell types responded to treatment per week for 2 weeks, suggesting a threshold for treatments that affect proliferation in multiple cell types.[17]

In similar vein, multiple targets were also used by Abe et al.[18] to explore the effect of Johrei on the viability and proliferation of cultured human cancer cells. Loss of cancer cell viability was significantly higher than in control groups, even though the responsiveness to Johrei varied with 9 different cancer types. The human gastric cancer cell line AGS and the uterine cervix epitheliod carcinoma HeLa proved most susceptible to Johrei, while the prostate cancer PC-3 and PPC-2 were the least susceptible. More studies like these second order articulations of healing variability by cell line may provide a basis for more robust theoretical understanding. For example, genetic mutations of different expression patterns of ion channels unique to a particular cell type and associated with altered responsiveness to healing therapies might provide clues regarding molecular pathways mediating the effects.

In recent years, there has been a significant increase in the number of studies on healing. Several peer-reviewed journals are devoted exclusively to this burgeoning field of complementary and alternative medicine, including the Journal of Alternative and Complementary Medicine; Alternative Therapies in Health and Medicine; and Explore: The Journal of Science and Healing. These journals publish both preclinical and controlled clinical studies of healing on a wide variety of conditions. In addition, an increasing number of peer-reviewed journals that are not focused exclusively on complementary and alternative medicine are open to publishing controlled studies in healing, such as the Journal of Scientific Exploration. Some of these, such as Dose Response, have previously only published articles with more conventional subjects.

Virtually nothing is known about how the various methods of healing converge or diverge in terms of healing efficacy. Do the different healing methods produce similar results? Also, there are no data to determine whether different healing methods have different mechanisms of action. As previously noted, researchers have spent an inordinate amount of time and effort trying to demonstrate the "fact" of healing to the so-called skeptical community and too little effort looking for what might be termed the "secondary correlates" of healing, such as dose response curves, the combined effects of conventional and unconventional healing, the role of belief, and so on.

Transition to the Present Study

Many of these second order correlations have been previously studied using the Bengston Healing Method[19,20] even as they await independent replication. The healing method involves very rapid imaging techniques which the participant healer cycles through. This is a relatively mechanical process which requires practice but not belief, and has been described elsewhere in great detail.[19,20] This healing method has been

tested in 16 in vivo cancer experiments on mice using standard models of mammary adenocarcinoma, methylcholanthrene-induced sarcomas, naturally occurring oncogenic tumors, immune-deficient nude mice, and innumerable other in vitro experiments on human leukemia and breast cancer cells.[21–25,26] These experimental models have a long history of known predictable outcomes with conventional empirical research.[27] In many of the studies using the Bengston healing method, volunteer healers, both students and faculty, have been pre-screened to have no experience in alternative healing, nor were they in any way "believers" in the validity of alternative healing. Variations in many parameters have been examined, such as distance, dose, and frequency of treatment; the subjective experiences of the volunteer healers; human physiological correlates using electroencephalograms (EEG) at a private lab, and functional magnetic resonance imaging (fMRI) carried out independently at 2 medical schools; and physical changes in the space adjacent to the healings.[25,28] An abridged summary of a selection of the results of these experiments include the following:

- Demonstration of a reliable full lifespan cure of cancer in experimental mice, including an apparent immunity to reinjection of the same cancer.[21]
- A dose response to healing. Some minimum amount of healing time is necessary to affect a cure. Interestingly, the only predictor of the aggregate speed of cure is the number of mice in an experiment, the quicker cures being associated with more mice being treated.[22]
- Healing proceeds in a non-linear fashion, with sudden bursts of healing that resemble "phase transitions."[21,22]
- There is a fluid, measurable "resonant bond" between healer and healee. Successful healing is associated with "connection," and healing failure is associated with "disconnection."[22]
- Healing efficacy apparently has no relationship to distance. It appears to be fundamentally about "information" despite the popular belief that it is primarily related to "energy."

The present study seeks to expand research into yet another under studied area. That is, can healing which could be considered outside the scientific mainstream transition towards more conventional application? An affirmative answer would require, at the very least, the ability to separate the vagaries of human intervention with some standardized application mechanisms. Simply put, the present study addresses the question of whether healing can be "stored" in such a way that it can be "delivered" on demand and hence be made widely available. This would require the demonstration of a scalable delivery technology analogous to the manufacture of many conventional therapies.

Previous work has already demonstrated that healing can be stored in cotton.[28] Proximity of treated cotton has induced measurable changes in cancer cells in vitro using MDA-MB-231. Carcinoma cells screened for markers using a qRT-PCR assay comprising 84 genes involved in cell growth and cancer were exposed to cotton treated by the healing method for 24 hours. The control consisted of cells not exposed to cotton. Significant change in expression was found in 6 genes: CASP9, E2F4, HMOX1, IGFBP3, MCM2, and PPP1R15A.[28]

The present study further explores whether healing can also be stored in a recording, and the effect reproduced in vivo without the further need of a human healer. Previous work has already demonstrated an affirmative answer on cancer cells in vitro.[28]

Materials and Methods

Recording of Healing Method

Three people practiced the Bengston healing method on cotton at the Institute for Noetic Sciences inside a solid steel, double-walled, electromagnetically shielded chamber (Series 81 Solid Cell; ETS-Lindgren, Cedar Park, Texas) for 5 minutes. The cotton per se had no experimental value, but rather was used as a means for the volunteer healers to use as a target for their healing intentions. As the treatment of cotton took place, magnetic and electromagnetic signals were recorded using 4 types of sensors: (1) 11 three-axis magnetoresistive sensors (Honeywell model HMC2003, sensitivity DC-1 KHz), (2) 2 antennas recording electromagnetic fields above 10 KHz, (3) a geomagnetometer (model IDR-321, sensitivity 8 DC-500 Hz; Integrity Design & Research, Essex Junction, Vermont), and (4) 2 custom-fabricated "Caduceus" coils, designed to cancel out transverse electromagnetic waves. Each of these 38 analog signals was digitized by a 24-bit analog-to-digital converter at 44.1 KHz (model Motu 24ai; Motu, Inc, Cambridge, Massachusetts) and custom PC software converted and saved the incoming sensor signals into the .wav audio format.

Cell Lines

Murine breast cancer cell line 4T1 cells were purchased from the American Type Culture Collection (Manassas, VA) and cultured in RPMI-1640 medium supplemented with 10% FCS and antibiotics. Murine bladder carcinoma line MBT-2 cells were provided from the American Type Culture Collection and cultured in RPMI-1640 medium supplemented with 10% FCS and antibiotics. Murine melanoma cell line B16 cells were obtained from Japan Health Science Foundation and cultured in alpha-MEM medium supplemented with 10% FCS and antibiotics.

Passages were performed once every 3–4 days in T150 culture flasks. Culture medium was aspirated and cells were washed with PBS. Adherent cells were treated with trypsin-EDTA in 37°C for 10 minutes. Cells were washed with fresh medium supplemented with 10% FCS and collected by a centrifugation. Cell numbers and viability were checked with

trypan blue dye exclusion test and 2×10^5 cells/ml were passaged to the new culture flasks.

Animals

5 week old BALB/c mice (female), C3H/HeN mice (male), and C57BL/6J mice (male) were acclimated for a week before they were exposed to the experimental recording. Because of space limitations, the entire study was broken up into two phases, with each phase using 10 mice per cell line. Phase 2 essentially became a replication of phase 1. Ten mice of each strain were purchased from Japan SLC Inc. for each phase of the experiments. In each phase, mice were divided into 2 groups (5 mice each), one for the control and another for audio treatment. The control mice were kept in the air-conditioned mouse breeding room with 12-hour light and dark cycle located at the ground floor. Mice in the treatment group were kept in an isolated air-conditioned compartment with 12-hour light and dark cycle located at the second floor. All mice were maintained in each air-filtered clean room and allowed free access to food (pellet) and water. Temperature and humidity of animal rooms during pilot and phase studies were kept at 23–25°C and 40–60%, respectively. All experiments with animals were complied with the standards in the guidelines of the University Animal Care and Use Committee in Tokyo University of Agriculture and Technology (Approval No. R02-84).

Engraftment of Tumor Cells

Cells were washed and harvested by trypsin-EDTA treatment. Cells were washed 3 times in ice-cold PBS and collected by centrifugation. Numbers of cells and their viability were evaluated by a trypan blue dye exclusion test. Cells with more than 95% viability were used for transplantation. Breast cancer 4T1 cells were suspended in PBS (5×10^5 cells/100 μl/site) and subcutaneously injected into the back of female BALB/c mice at the age of 6 weeks. Bladder carcinoma MBT-2 cells were suspended in PBS (1×10^6 cells/100 μl/site) and subcutaneously injected to the back of male C3H/HeN mice at the age of 6 weeks. Melanoma B16 cells were suspended in PBS (2×10^5 cells/100 μl/site) and subcutaneously injected to the back of male C57BL/6J mice at the age of 6 weeks. Low-dose syringes and needles were used for the cell injection.

Measurement

Before the first injection, blood samples and complete blood counts (CBC) from each mouse were examined. The body weight of each mouse was measured. After the cell injection, tumor size (width and length) was measured once a week estimating tumor volume (mm^3) using tumor volume = $[(\text{width})^2 \times \text{length}]/2$.[29] The body weight of each mouse was also monitored and images of each mouse (overhead and side) were taken.

Treatment With Audio Recording

Mice in the treatment group were kept in a clean compartment room with a HEPA filter located at the ground floor. The treatment consisted of 4 hours of exposure to the recording played daily between 10:00 and 14:00. The audio system playing the recording was placed on a rack above the mice cages with two passive speakers (PCB4 K; Pyle Home, Brooklyn, New York) pointing down toward the cages. The speakers were connected in phase with 18AWG speaker wires (Southwire, Carrollton, Georgia) to an amplifier (PTAU45, Pyle). During the period of exposure, the recording was repeatedly played on a loop by the amplifier connected directly to the speakers. The recorded audio file (.wav) was present on a USB drive that was inserted into the amplifier USB port. The volume of the computer and the player, software, as well as the amplifier were set at 50%. The importance of the 50% setting is unknown, as no frequency per se could be heard from the speakers despite this being a .wav file. The amplifier was under the control of a timer, which allowed the recording to be played continuously for 4 hours per day. Following engraftment, mice in the room with the recording were housed in the dedicated room. Mice in the control group were housed in a distant general housing room. All mice were in a 12 hour light and 12 hour dark cycle. Animal room caretakers for the control group and treatment group were separate technicians to minimize potential "contamination" effects due to resonant bonding influences.

Sample Collection

At the end of the experiments, all mice were euthanized, whole blood was collected by heart-puncture, and CBC examined. Formed tumors were enucleated, weight-measured, and divided into two portions. One section was immediately stored in buffered-formalin for histological analysis and the other section was cut into pieces smaller than 5 mm blocks and stored in RNALater™ at −20°C.

Other organs (lung section, whole heart, whole kidney, and liver lobe) were harvested and stored together in a formalin sample jar. The entire spleen was also harvested and weighed. After weighing, one-half of the spleen was added to the other organs in the same formalin sample jar. The other spleen half was stored in RNALater™ at −20°C.

Statistical Analysis

Graph Pad Prism version 9.2.0 (283) was used for the data analysis. Two-way ANOVA followed by Tukey's multiple comparisons test was applied for the surface mass measurement data and the body weight data. Unpaired t test with Welch's correction was applied to the statistical analysis of the extracted tumor weight data, the spleen weight data, and CBC data. A P value < .05 was regarded as statistically significant.

Results

Recording Output

In some cases, interesting short-term temporal effects were observed compared to controls, and these may be the subject of further study. However, the focus here is on the more consistently observed spectral changes where 3 healers were engaged in treating pieces of cotton.

Spectral analyses covered the frequency range from below .1 Hz to about 20 KHz. For the recording, the only region where significant differences were observed was at frequencies below 20 Hz, and in particular, below 5 Hz. For all 11 3-axis magnetometers used to record the activity, 1 data channel (y-axis) indicated significantly elevated spectral content in the frequency range .25 Hz to about 3 Hz, as compared to control recordings. These elevations in spectral levels between R18 and control results were typically 6 dB or more, and in some cases, 20 dB or more. Figure 1 illustrates the spectral differences in the very low frequency range for 2 independent magnetometers.

These frequencies are far *below* the audible range of human hearing.

Tumor Growth in BALB/c Mice Injected With 4T1 Breast Cancer Cells

The results of the three strains of cancer, breast, melanoma, and bladder, are analyzed separately.

Tumor development and growth of 4T1 cells in syngeneic BALB/c mice were confirmed in both Phase I and Phase II experiments. Tumors of 4T1 cells sometimes showed erosion at the injected sites. In the Phase I study, one mouse in the control group and one mouse in the treatment group died between 3 and 4 weeks after injection of 4T1 cells. In the Phase II study, 3 mice in the control group died 4 to 5 weeks after injection. No mouse died until the end point in the treatment group in the Phase II study.

Surface Mass. Weekly surface mass measurements in the combined Phase I and Phase II experiments are shown in Table 1 and Figure 1. Data in Table 1 show averages ±standard error (SE) of mice in the control group and the treatment group. Figure 1 shows the average mm2 ±SE of the growth curve in each group. At Week 5, tumor growth in the mice of the treatment group was significantly suppressed compared to the control mice ($P < 0.0001$).

In the control mice, 4T1 cells formed larger tumors than those in the treatment mice.

Exposure to the audio recording significantly delayed the growth of 4T1 tumors ($P < .0001$). Autopsies were performed after euthanasia of the BALB/c mice. Many metastatic spots were found in the lung of mice in treatment group as well as mice in the control group. Also, most of mice had opaque ascites in the peritoneal cavity. (See Table 1 and Figure 2 below.)

CBC DATA. CBC data before and after the combined Phase I and Phase II experiments are shown below (Table 2). Data show average ± SE of 8 mice in the control group and 9 mice in the treatment group. The upper and lower tables show data collected before or after experiments, respectively. Mice in both groups showed marked increase in WBC at the end point. Anemia was recognized in all mice at the end point; however, it tended to become more severe in control mice with significantly lower HCT than those of mice in treatment group ($P < .05$). No statistical difference was identified between the control group and the treatment group in WBC ($P = .88$), RBC ($P = .19$), Hbg ($P = .28$), and PLT ($P = .16$).

Tumor and Spleen Size. At the end point of the experiment, all mice were euthanized, and samples were collected. Parenchyma of enucleated tumors and isolated spleens of each mouse were weighed. The extracted tumor size was smaller in the treated mice. The weight of spleen of each group was almost the same, though they were both larger compared to the spleens of BALB/c mice without tumor transplantation. Columns in Figure 3 show average ± SE of each group. Extracted tumor weight in the treatment group was significantly smaller than that in the control group ($P < .05$).

Body Weight. Changes in body weight are given in Table 3.

Tumor Growth in C57BL/6J Mice Injected With B16 Melanoma Cells

Tumor development and growth of B16 melanoma cells in syngeneic C57BL/6J mice were confirmed in both Phase I and Phase II experiments. Tumors engrafted with B16 cells showed soft round tumors at the injected sites and sometimes accompanied edematous areas around the tumors. In the Phase I study, no mouse was lost until 5 weeks after injection of B16 cells. In the Phase II study, 2 mice in the control group and 2 mice in the treatment group were lost 4 to 5 weeks after injection of B16 cells.

Surface mass measurement data every week in Phase I and Phase II experiments are shown below. Data in Table 4 show averages ±SE of mice in the control group and the treatment group.

In surface measurement data, no significant differences between groups were identified. Autopsies were performed after euthanasia. No metastatic spots were found in the lungs of mice in either group. No mouse had ascites in the peritoneal cavity (Figure 4).

CBC data before and after the Phase I and Phase II experiments are shown below. Data show the average ± SE of 10 C57BL/6J mice with B16 tumors in both control and treatment groups. Upper and lower tables show data collected before or after experiments, respectively. WBC in mice of both groups was almost the same before and after experiments. However, serious anemia was

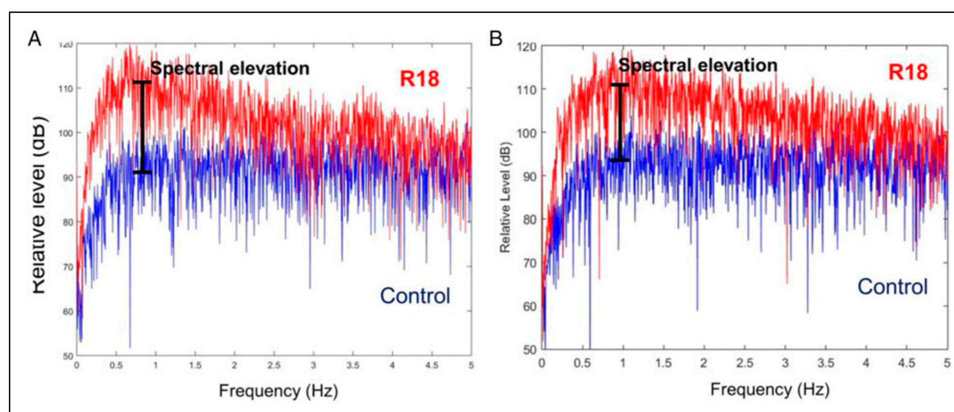


Figure 1. Illustration of differences observed at low frequencies in the recording compared to control recording. Energy from 3 healers simultaneously charging cotton inside a Faraday chamber (red) was recorded on 38 channels and data were converted into an audio format as .wav. The spectra from these recordings were compared to a control situation (control, blue, Faraday chamber containing a crystal). The graphs represent the spectra of the recording and control from sensor A (A) and sensor B (B). The vertical bar indicates a spectral elevation of approximately 20 dB for sensor A and 15 dB for sensor B in the frequency range between .5 and 1 Hz. This elevation was observed for all 11 magnetometers used in the recording apparatus.

Table 1. Surface Mass Volume (mm^2) of 4T1 Breast Cancer Cells in BALB/c Mice.

	Week-1	Week-2	Week-3	Week-4	Week-5
Control	40.1 ± 13.1	548.1 ± 80.5	1210.8 ± 106.0	1967.1 ± 273.8	3271.0 ± 580.4
Treatment	18.6 ± 11.0	341.7 ± 84.1	854.2 ± 128.4	1360.7 ± 160.8	1972.7 ± 224.6

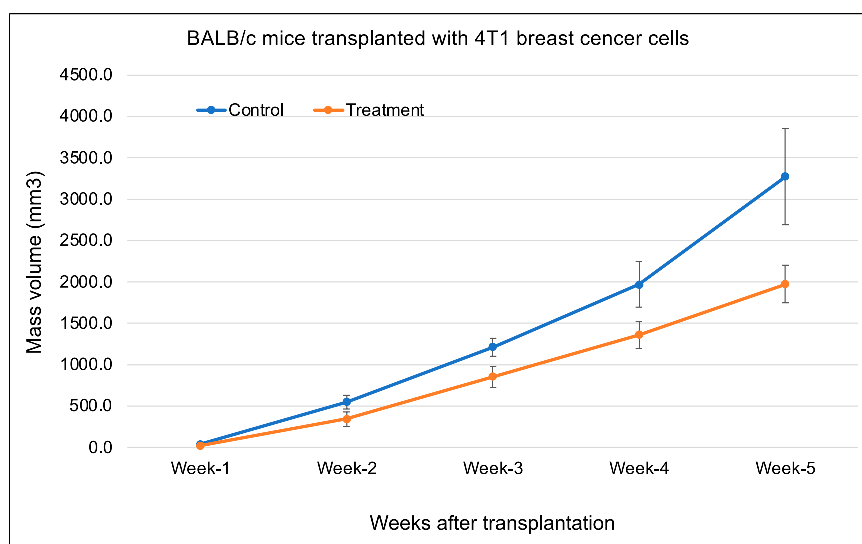


Figure 2. Surface mass measurement data of BALB/c mice with 4T1 cell transplantation.

Table 2. CBC Data *Before* (Upper) and *After* (Lower) the Experiments.

	WBC ($\times 10^3/\mu l$)	RBC ($\times 10^6/\mu l$)	Hbg (g/dl)	HCT (%)	PLT ($\times 10^3/\mu l$)
Control	8.3 ± 0.4	10.3 ± 0.4	14.0 ± 0.5	53.0 ± 2.4	802 ± 64
Treatment	8.9 ± 0.4	11.0 ± 0.4	14.6 ± 0.3	56.4 ± 2.5	945 ± 72
Control	115.6 ± 6.7	9.0 ± 1.3	11.5 ± 1.7	44.3 ± 1.6	574 ± 89
Treatment	116.9 ± 5.2	11.0 ± 0.6	13.2 ± 0.7	55.8 ± 3.5	737 ± 64.7

WBC: white blood cells, RBC: red blood cells, Hbg: hemoglobin, HCT: packed cell volume or hematocrit, PLT: platelets.

Table 3. Changes in Body Weight (g).There Were No Significant Differences (P > .05).

	Week-0	Week-1	Week-2	Week-3	Week-4	Week-5
Control	19.2 ± 0.5	19.8 ± 0.2	21.4 ± 0.3	20.6 ± 0.8	20.7 ± 1.3	23.8 ± 0.6
Treatment	18.2 ± 0.3	19.2 ± 0.4	20.5 ± 0.4	20.5 ± 0.7	21.3 ± 1.0	23.0 ± 1.2

Table 4. Surface mass volume of B16 Melanoma Cells in C57BL/6J Mice. There were no significant differences (P > .05).

	Week-1	Week-2	Week-3	Week-4	Week-5
Control	10.0 ± 3.6	295.9 ± 142.9	2952.6 ± 1045.3	10506.0 ± 2669.8	10000.8 ± 2325.7
Treatment	17.9 ± 5.2	290.0 ± 93.2	2478.5 ± 712.8	8452.6 ± 2159.5	11529.7 ± 2712.6

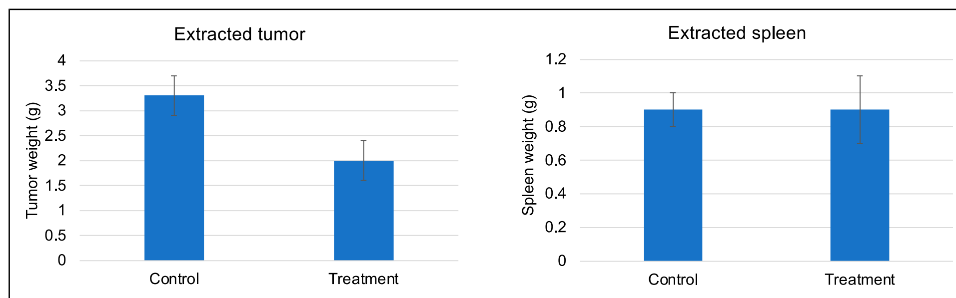


Figure 3. Weight data of extracted tumors and spleens collected from BALB/c mice with 4T1 breast cancers.

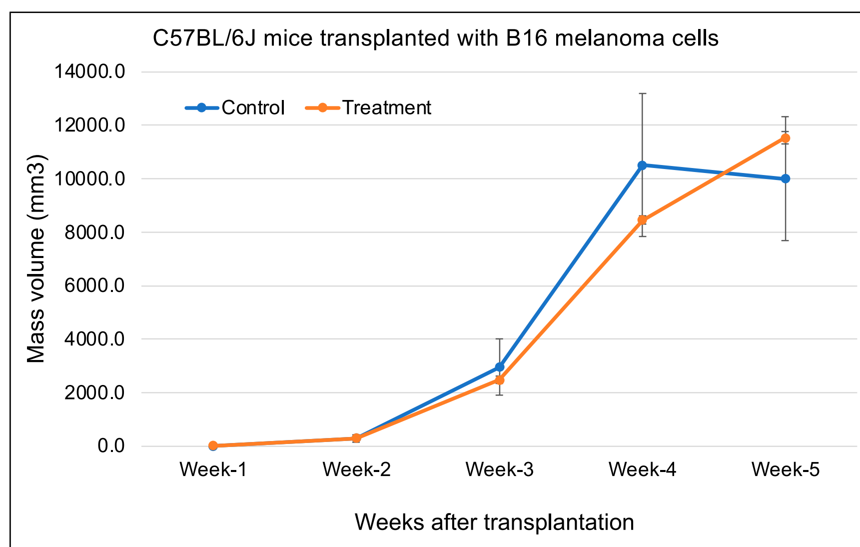


Figure 4. Surface mass measurement data of C57BL/6J mice with B16 cell transplantation.

recognized in all mice of both groups at the end point. PLT also became lower in mice of both groups at the end point. PLT in the treated group was significantly lower than that in the control group ($P < .05$). No significance differences were identified between treated and control groups in WBC ($P = .23$), RBC ($P = .65$), Hbg ($P = .68$), and HCT ($P = .73$) (Table 5).

At the end point of the experiment, all mice were euthanized and samples were collected. Parenchyma of enucleated tumors and isolated spleens were weighed. Extracted tumor size tended to become larger in the control mice than in treated mice. No significances differences between groups were found. The weight of spleen of each group was almost the same. Columns in Figure 5 show average ± SE of each group.

Body weight change is shown in Table 6. Data represent the average ± SE of each group. There were no significant differences between groups.

Experiments With C3H/HeNJ Mice and MBT-2 Bladder Carcinoma Cells

Despite a successful pre-test on two mice before the Phase I study began, tumor formation in C3H/HeNJ mice was not confirmed in either Phase I or Phase II. This part of the experiment was terminated at 4 to 5 weeks after injection of MBT-2 cells. The exact cause of the failure to form tumors is unknown.

Discussion and Conclusions

Treatment with daily exposure to the audio recording showed suppressive effects on tumor growth in the syngeneic mouse model for breast cancer. Statistical significance was found between treated and control groups in surface mass measurement data and extracted tumor weight data. BALB/c mice in the control group tended to show serious anemia in CBC data at the end point, compared to the treatment mice, suggesting that the general conditions of mice in the treatment group might be relieved from serious tumor cachexia. HCT at the end point was significantly lower compared to that in the treated mice, indicating that the control mice showed more severe anemia. No statistical significance between groups was found in the other CBC data.

In contrast, exposure to the audio recording generally showed no suppressive effect on the mouse model of melanoma with the exception of a difference only on platelet count. Serious progression of tumors in C57BL/6J mice injected with B16 melanoma cells was identified in both control and treatment groups.

MBT-2 cells did not form tumors in C3H/HeNJ mice in this study, although they grew as expected in the pilot study. The reason for this is unclear, but since the pilot study was conducted

Table 5. CBC Data *Before* (Upper) and *After* (Lower) the Experiments.

	WBC (× $10^3/\mu$l)	RBC (× $10^6/\mu$l)	Hbg (g/dl)	HCT (%)	PLT (× $10^3/\mu$l)
Control	9.1 ± 0.7	9.1 ± 0.5	12.7 ± 0.6	49.8 ± 1.5	576 ± 70
Treatment	8.4 ± 0.3	9.7 ± 0.3	13.1 ± 0.5	49.5 ± 1.1	553 ± 82
Control	9.9 ± 1.3	5.2 ± 1.0	5.7 ± 1.1	26.7 ± 5.3	284 ± 36
Treatment	7.6 ± 1.3	4.5 ± 1.2	5.1 ± 1.2	23.9 ± 6.0	162 ± 20

WBC: white blood cells, RBC: red blood cells, Hbg: hemoglobin, HCT: packed cell volume or hematocrit, PLT: platelets.

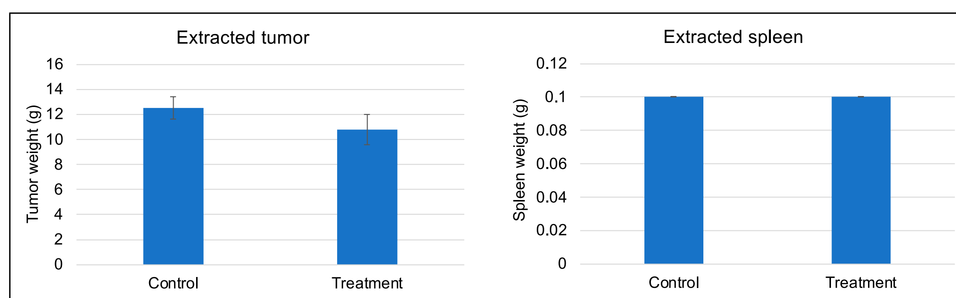


Figure 5. Weight data of extracted tumors and spleens collected from C57BL/6J mice with B16 melanoma.

Table 6. Body weight change.

	Week-0	Week-1	Week-2	Week-3	Week-4	Week-5
Control	20.8 ± 0.2	21.7 ± 0.3	22.5 ± 0.4	24.0 ± 0.9	28.9 ± 1.8	32.4 ± 2.2
Treatment	20.6 ± 0.3	21.1 ± 0.3	21.4 ± 0.4	23.8 ± 0.6	29.3 ± 1.5	33.0 ± 1.9

immediately after the purchase of the cells and the present study used cells that had been cultured and stored in the freezer, some sort of change in cell properties may have occurred.

This discussion necessarily will only address findings from the breast cancer and melanoma models.

In such a newly emerging field, it is not surprising that more questions are raised than answers provided. It should be remembered that the main focus of this research is relatively new: to reproduce the already established effects of healing intention without the healer, that is, without human intervention. This is quite different from most studies which have looked at the experimental effects of healing either in vitro or in vivo with some success.[20-22,30,31]

Using the same recording as found in this study, Beseme et al.[28] have previously established a reliable effect on breast cancer cells in vitro, though the strength of the effect was somewhat less than that produced by live healers.[28] An attempt to reproduce the effect using the same breast in vivo cancer model used in this study produced significant results, but the experiment had to be prematurely aborted because tumor volume exceeded IACUC guidelines.[32]

Among the questions that need to be addressed include the reason that the recording "worked" on the breast cancer model in the first place, as well as why the recording did not produce significant effects on the melanoma model. A short list of questions that need addressing include the following:

1. By what mechanism might the recording produce tumor suppression?
2. Why might that mechanism affect breast cancer but not melanoma?
3. Can the recording more properly be seen as an "alternative" or as a "complementary" therapy?

Some speculation follows:

By what mechanism might the recording produce tumor suppression? As illustrated in Figure 1, significant changes in spectral elevation were observed in the recording. And signal analysis suggested that all significant effects were found at extremely *low* frequencies (3.5-5 Hz). This contrasts with some current therapies that involve *high* frequencies. Image-guided focused ultrasound (FUS) has been successfully employed as a treatment for solid malignancies in the same cancer models used in this study.[33,34] Why would both extremely *low* and *high* frequencies produce tumor suppression, and why would the low frequency intervention work only on the breast cancer model yet the high frequency intervention also produce significant effects on the melanoma? The answer is currently unknown, even though low frequencies have been shown to work, at least in vitro, on other conditions. Ross and Harrison, for example, demonstrated that a similar low frequency (5 Hz) played to an in vitro inflammation model significantly reduced the amount of inflammation.[35]

Studies such as these are usually put under the term "energy medicine," even though the actual energetic mechanisms of action which have been proposed remain widespread and oftentimes controversial.[36] Indeed, whether it is even "energy" or "information" which is involved in healing remains an open question.

What mechanism might affect breast cancer but not melanoma? The microenvironment surrounding a tumor is the interface between the tumor cells and the immune system of the host. Tumor-produced factors and tumor growth dynamics can affect the microenvironment in a variety of ways. The audio treatment used in this study is a non-thermal physical therapy as well as infrasound-based cancer treatment. Cohen *et al.* reported in an in vivo study with 4T1 breast cancer and B16 melanoma that Pulsed-Focused Ultrasound (pFUS) suppressed the cancer progression in each syngeneic mouse strain.[33,34] In pFUS treatment, the numbers of immune cells infiltrating the microenvironment surrounding the tumor was more obvious in B16 tumors than in 4T1 tumors, suggesting that the antitumor effect of pFUS on B16 tumors might depend on host-derived immune cells in the tumor microenvironment. In contrast, in the 4T1 tumors, there was no significant increase in host-derived immune cells in the tumor microenvironment, suggesting that the antitumor effect of pFUS might be a direct effect on 4T1 tumor cells. In the present study, audio treatment was effective against 4T1 tumors but not against B16 tumors, suggesting that the target of audio treatment may not be host-derived immune cells, but rather a direct effect on tumor cells.

Whether the recording can primarily be seen as an "alternative" or as a "complementary" treatment remains unknown. Some past in vivo studies which used live healers on breast cancer models have produced interventions that result in full lifespan cure.[21,22] In the present study, the recording produced statistical tumor suppressive effects but not complete elimination of the cancer. This implies that the recording has not completely captured the healing effect.[28,32] Speculative possibilities for this include the necessity for more or different kinds of detectors; the translation of analog signals that were reduced to a digital format so there was some information lost. The possibilities are endless, and at this point, it is important that research into various forms of recordings take place.

Previous studies on "live" healers suggest that that model of therapy can be applied as an "alternative" one. But using one-on-one therapies obviously eliminates the chance to widely scale the treatment, which would be necessary in order to gain more conventional acceptance. The recording, which is immediately scalable, at this point seems not to be sufficient to be an alternative treatment. It may in fact be useful as a "complementary" therapy, but at this point, what complementarity would practically mean is unknown.

There are ample data here to suggest that a technology of healing intention can be developed. At the same time, more work needs to be done on increasing its efficacy.

Acknowledgments

The authors thank Jason Yotopoulos from The Emerald Gate Charitable Trust for his invaluable input into the project, and research fellows and technicians from TUAT Kaoru Karasawa, DVM, PhD, Mayumi Shibayama, and Mayuko Nagasaki.

Declaration of Conflicting Interests

The author(s) declared the following potential conflicts of interest with respect to the research, authorship, and/or publication of this article: WB is the inventor of the Bengston Healing Method and author of works describing the method.

Funding

The author(s) disclosed receipt of the following financial support for the research, authorship, and/or publication of this article: This research was fully funded through generous donations from The Emerald Gate Charitable Trust.

ORCID iD

William Bengston https://orcid.org/0000-0002-0164-5025

References

1. Schiegl H. *Healing Magnetism*. Freiburg, Germany: Hermann Verlag KG; 1983.
2. Grad B, Cadoret R, Paul G. An unorthodox method of treatment on wound healing in mice. *Int J Parapsychol*. 1961;3:5-24.
3. Grad B. A telekinetic effect on plant growth. *Int J Parapsychol*. 1964;6:473-498.
4. Grad B. A telekinetic effect on plant growth II experiments involving treatment of saline in stoppered bottles. *Int J Parapsychol*. 1963;5:114-133.
5. Benor D. Healing research: Holistic medicine and spirituality. In: *Research in Healing, Vol 1*. Munich, Germany: Helix-Editions; 1992.
6. Snel F. PK influences on malignant cell growth. *Research Letter No. 10*. Netherlands: Parapsychology Laboratory, University of Utrecht; 1980.
7. Smith J. First international education conference in health sciences. *Human Dimensions*. 1972;135:15-19.
8. Crawford CC, Sparber AG, Jonas WB. A systematic review of the quality of research on hands-on and distance healing: Clinical and laboratory studies. *Altern Ther Health Med*. 2003;9(3 Suppl):A96-A104.
9. Schlitz M, Radin D, Malle B, Schmidt S, Utts J, Yount GL. Distant healing intention: Definitions and evolving guidelines for laboratory studies. *Altern Ther Health Med*. 2003;9(-3 Suppl):A31-A43.
10. Bunnell T. The effect of "healing with intent" on pepsin enzyme activity. *J. Scientific Exploration*. 1999;13(2):139-148.
11. Barry J. General and comparative study of the psychokinetic effect on a fungus culture. *J or Parapsychology*. 1968;32:237-243.
12. Haraldsson E, Thorsteinsson T. Psychokinetic effects on yeast: an exploratory experiment. In: Roll WG, Morris RL, Morris JD, eds. *Research in Parapsychology*. Matuchen: Scarecrow Press; 1973:10-21.
13. Nash C. Test of psychokinetic control of bacterial mutation. *J. American Society for Psychical Research*. 1982;51:217-221.
14. Rein G. *Quantum Biology: Healing with Subtle Energy*. Palo Alto: Quantum Biology Research Labs; 1992.
15. Braud WG. Distant Mental Influence on the rate of hemolysis of human red blood cells. *J American Soc Psy Res*. 1990;84(1):11-24.
16. *Global Advances in Health and Medicine*. Consciousness and Healing Initiative. 2015;4(suppl).
17. Gronowicz GA, Jhaveri A, Clarke LW, Aronow MS, Smith TH. Therapeutic touch stimulates the proliferation of human cells in culture. *J Altern Complement Med*. 2008;14(3):233-239.
18. Abe K, Ichinomiya R, Kanai T, Yamamoto K. Effect of a Japanese energy healing method known as Johrei on viability and proliferation of cultured cancer cells in vitro. *J Altern Complement Med*. 2012;18(3):221-228.
19. Bengston WF. Commentary: A method used to train skeptical volunteers to heal in an experimental setting. *J Altern Complement Med*. 2007;13(3):329-331.
20. Bengston W. *Hands on Healing: A Training Course in the Energy Cure (CD Audio Set)*. Colorado: Sounds True; 2010.
21. Bengston W, Krinsley D. The effect of the laying-on of hands on transplanted breast cancer in mice. *J Sci Explor*. 2000;14(3):353-364.
22. Bengston WF, Moga M. Resonance, placebo effects, and type II errors: Some implications from healing research for experimental methods. *J Altern Complement Med*. 2007;13(3):317-327.
23. Bengston WF. Spirituality, connection, and healing with intent: Reflections on cancer experiments on laboratory mice. In: Miller L, ed. *Science and Spirituality*. New York: Oxford University Press; 2012.
24. Moga M, Bengston W. Anomalous magnetic field activity during a bioenergy healing experiment. *J Sci Explor*. 2010;24(3):397-410.
25. Bengston W. Some reflections on consciousness, intention, and healing. In: Dunne B, Jahn R, eds. *Being and Biology: Is Consciousness the Life Force?* Princeton: ICRL Press; 2017:101-118.
26. Bengston W. Methodological difficulties involving control groups in healing research. *J Alter and Compl Med* 2007;13(3):317-327.
27. Lerner LJ, Bianchi A, Dzelzkalns M. Effect of hydroxyurea on growth of a transplantable mouse mammary adenocarcinoma. *Cancer Res*. 1966;26(11):2297-2300.
28. Beseme S, Bengston W, Radin D, Turner M, McMichael J. Transcriptional changes in cancer cells induced by exposure to a healing method. *Dose Response*. 2018;16(3):1559325818782843-1559325818782848.
29. Tanaka A, Muto S, Konno M, Itai A, Matsuda H. A new IkappaB kinase beta inhibitor prevents human breast cancer progression through negative regulation of cell cycle transition. *Cancer Res*. 2006;66:419-426.
30. Yang P, Jiang Y, Rhea P, et al. Human biofield therapy and the growth of mouse lung carcinoma. *Integr Cancer Ther*. 2019;18:1-12. DOI: 10.1177/1534735419840771

31. Yang P, Rhea P, Conway T, et al. Human biofield therapy modulates tumor microenvironment and cancer stemness in mouse lung carcinoma. *Integr Cancer Ther*. 2020;19: 1-11.
32. Beseme S, Fast L, Bengston W, Turner M, Radin D, McMichael J. Effects induced In Vivo by exposure to magnetic signals derived from a healing technique. *Dose Response*. 2020;18:1-10.
33. Cohen G, Chandran P, Lorsung R, et al. Pulsed-focused ultrasound slows B16 melanoma and 4T1 breast tumor growth through differential tumor microenvironmental changes. *Cancers*. 2021;13(7):1546.
34. Cohen G, Chandran P, Lorsung RM, et al. The impact of focused ultrasound in two tumor models: Temporal alterations in the natural history on tumor microenvironment and immune cell response. *Cancers*. 2020;12:350.
35. Ross C, Harrison B. Effect of pulsed electromagnetic field on inflammatory pathway markers in RAW 264.7 murine macrophages. *J Inflamm Res*. 2013;6:45-51.
36. Oschman J. *Energy Medicine: The Scientific Basis*. NY: Elsevier; 2016.
37. Hendricks L, Bengston W, Gunkleman J. The Healing connection: EEG Harmonics, Entrainment, and Schumann's Resonances. *J Sci Explor*. 2010;24(4):655-666.