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A Study of the Effect of Energy Healing on *In Vitro* Tumor Cell Proliferation

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ABSTRACT

This study examined the effect of energy healing on *in vitro* tumor cell growth using the cell culture model similar to that embraced by oncologists to assess the effect of chemotherapeutic agents. After selecting an energy healer based on his ability to influence this model, we assessed the effects of energy treatment compared to cells left at ambient temperature and to a control treatment consisting of a medical student mimicking the healer. A chi-square test comparing a medical student's and the practitioner's ability to inhibit tumor cell growth by 15% associates our practitioner with inhibition of tumor cell proliferation ($p = 0.02$). We also found that the magnitude of change was too close to the assay's intrinsic margin of error, thus making our quantitative data difficult to interpret. Although energy healing appears to influence several indices of growth in *in vitro* tumor cell proliferation, these assays are limited in their ability to define and prove the existence of this phenomenon. More sensitive biological assays are needed for further study in this field.

INTRODUCTION

Allopathic practitioners of Western medicine attribute ailments to observable dysfunctions of organs or to the body's regulatory mechanisms. Although the placebo effect suggests that the mind can cause myriad physical changes in the body, the mind-body relationship still remains a mystery. Medical scientists would concede that a person's mental state can influence his or her physical state, but are more skeptical of the idea that a person can mentally influence aspects of another person's body. Western science does not currently have a paradigm with which to understand therapies in which practitioners purport to deliver or channel energy to a patient, or therapies in which practitioners, using physical or metaphysical means, manipulate energy fields. For this reason, healthcare practitioners are generally more open to physically tangible forms of complementary medicine such as massage therapy or yoga, but are suspect of healers who claim to treat patients through intense concentration or meditation. This negative bias persists despite the presence of over 150 studies (see Benor, 1992 for a review) claiming the effect of intentional healing.

These studies often fail to convince physicians practicing at large academic centers. One problem is inconsistent adherence to strict scientific method and analysis. For example, a sham treatment, a critical control of nonspecific or unintended interactions, was omitted from one of the most compelling studies (Wirth, 1992). Another study with impressive results

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failed to report thoroughly their experimental protocols (Chien et al., 1991). Secondly, there is diminished credibility of studies in which investigators have published without trying to generate an adequate sample size (Rein, 1995) or have failed to report their findings in peer-reviewed journals (Chien et al., 1991; Rein, 1995). Finally, we found that many allopathic physicians are biased against studies that are performed in small privately funded centers for alternative medicine. Though the data and methods of these studies may be sound, their replication at large allopathic academic centers could boost their credibility in the eyes of traditional allopathic physicians.

This study attempts to address some of these methodological problems by documenting the effect of one form of intentional healing on a simple biological model. We chose an *in vitro* tumor cell proliferation assay because it is specific and quantifiable, and because previous studies suggest that energy healers may be able to retard tumor cell growth (Rein, 1995; Snel et al., 1995; Chien et al., 1991). Furthermore, one proposed mechanism of intentional healing is that healers are emitting electromagnetic (or other) energy fields. Low-frequency electromagnetic fields have been shown to affect oncogenic mechanisms (Dees et al., 1996; Liburdy et al., 1993).

METHODS

Study overview

Phase I. Initially, healers were screened in order to find one that had the highest likelihood of efficacy within the constraints of our model. Prospective healers were given one of two identical plates of MCF-7 cells (see below) to treat using their modality of choice. Additionally, a student performed a sham treatment on an identical plate of cells by mimicking the actions of the healer. This plate served as a negative control. After 24 hours of incubation, the cells were counted and compared. We considered a healer effective if he or she could two out of the three times demonstrate at least a 15% decrease in cell number when compared to the control.

Phase II. The healer with the most impressive results from the screening phase was used in a more extensive experimental phase consisting of four cell lines and two more conditions: (1) a plate of cells which remained in the incubator at 37°C, termed "untouched"; and (2) another left out in the laboratory at room temperature, termed "ambient." The four experimental conditions are as follows: (E): experimental (treatment); (C): control (sham treatment); (U): untouched; and (A) ambient.

Treatment lasted 1 hour. At treatment time, the cells were removed from the incubator, brought to the experimental room, and placed on a desk with a chair in front of it. Our practitioner then held his hands over the cells, sometimes pointing at the wells, sometimes holding his palms over the cells. Once or twice during the treatment, he gently shook the cells without lifting them, using a slow back-and-forth movement. Also, he would often make a wiping motion above cells that looked like he was pulling something out of the air. For approximately the last 20 minutes of treatment, the practitioner sat down in the chair and mediated. During each treatment, a student watched the practitioner from the other side of the room and noted specific movements. At no time did the practitioner remove the protective covering of the cell plates. After treatment, the student replaced the cell cultures in the incubator. To provide a negative control, either before or after the experimental treatment (in a randomized fashion), a student performed a sham treatment, in the same room and position and for the same amount of time. Afterwards, the student replaced the cells in the incubator. Once all the cells were back in the incubator, a scientists not involved in the study labeled the plates in order to blind the investigators in all subsequent steps. The plates and their corresponding treatments were matched only after all the counts were read and analyzed.

Practitioner

Our practitioner, Frank Huo, has studied Yuanji medicine for 18 years and currently practices under the Yuanji Science Worldwide Corporation. Yuanji medicine is a traditional Chinese science dedicated to healing the dis-

eased body and mind. The Yuanji practitioner focuses on collecting universal energy and transferring that energy from practitioner to subject. Although bodily balance is the goal of Yuanji practices, our practitioner felt that he could transfer energy to any subject. Additionally, he has received a B.S. in Material Science and an M.S. in Plastics and Processing Engineering (Beijing University of Aeronautics and Astronautics).

Culturing cells

Fresh cells (p0) were frozen in multiple aliquots and stored at −80°C in fetal bovine serum (Gibco BRL, Gaithersburg, MD) supplemented with 10% dimethyl sulfonate (DMSO) (Sigma, St. Louis, MO). Cells were cultured in RPMI medium supplemented with 10% fetal bovine serum and 100 U/mL penicillin and streptomycin (Gibco BRL). The cells were incubated at 37°C in 5% CO_2 and were maintained for ten passages before a new aliquot would be thawed from the parent batch.

Protocol for practitioner screening (phase I)

Twenty-four hours before an experiment, MCF-7 cells were trypsinized (Trypsin in 0.05% EDTA, Gibco BRL) and passed on to two 6-well dishes at a concentration of 30,000 cells per well. One dish would be used as the experimental and one would be the control. Twenty-four hours after treatment, cells were trypsinized against, spun (1000 rpm for 5 minutes) and resuspended in 1 mL of phosphate-buffered saline (PBS). Cels were stained with trypan blue (1:10 trypan blue:cell suspension) and counted using a hemacytometer (Sigma). Assays were done in sets of six. The mean counts for the six wells were taken to be the total cell count for that condition.

Protocol for experimental trial (phase II)

Cells were trypsinized (except K562, which are nonadherent), counted, and passed on to 6 wells of four 96-well dishes at a concentration of 3,000 cells per well. On the day of the experiment, one plate was left out in the experimental room for 5 hours, one plate was left in the incubator, and two others were used as the experimental and the control. Eighteen to 20 hours before the time of assay 0.2 mCi/well of 3-methyl thymidine (Sigma) was added. At this point (24 hours after treatment), cells were harvested (Wallac 1295-001 Cell Harvester, Gaithersburg, MD) and counted (Wallac 1205 Betaplate Liquid Scintillation Counter). Raw numbers from six independent wells pertaining to a particular cell type and treatment were averaged to obtain a single data point, the mean cell count. In the case of the positive control, a cocktail of 4 micromolar cis-platinum (II)-diamine dichloride and 4 micromolar tamoxifen (Sigma) was added to the cells prior to adding thymidine.

Cell lines

The following four human cell lines were used: MCF-7, K562, MEL6, and LNCaP. MCF-7 cells have been reported to respond to weak electromagnetic fields (Dees et al., 1993; Liburdy et al., 1996) and is an ER+, breast cancer line. MEL6 is a melanoma line with a p53 knockout. LNCaP is a prostate metastatic line that was cultured from a patient's lymph node. K562 is a leukemia line that responds particularly well to natural killer cell activity. MCF-7, K562, and LNCaP are commercially available (ATCC). MEL6 cells were a kind gift of Dr. Tony Raffo, Department of Oncology, Columbia University.

Statistical methods/data analysis

When averaging the counts for the six wells, any number that was more than two standard deviations away from the mean of the remaining five numbers was discarded. If more than half of the wells were outliers, the entire experiment was considered unreliable and was discarded. Mean counts were applied to the following equation (1-experimental/untouched) × 100 to obtain a value for effect. A negative number indicated growth relative to the incubator; a positive number indicated inhibition (or cell death) relative to the incubation.

A two-tailed paired *t*-test compared control and experimental groups for significant differences. A one-way analysis of variance

Table 1. Results of Practitioner Screening

Practitioner type	*Trial 1*	*Trial 2*	*Trial 3*
Therapeutic touch 1	25[a]	−10	0
Therapeutic touch 2	−17	−36	3
Energy healer	15	−15	−6
Yuanji healer	31	29	18

[a]Numbers are expressed as percent inhibitions calculated with the following equation: (1-treated/control)*100.

(ANOVA) demonstrated whether there were significant differences between the four groups. To evaluate whether the experimental treatment was significantly associated with a decrease in cell number (relative to the untouched plate) the data was analyzed by converting our continuous data into binary data and then using a chi-square test.

RESULTS

Table 1 demonstrates that the first three practitioners did not meet our study requirements. The Yuanji healer, on the other hand appeared to consistently inhibit proliferation. The first four columns of Table 2 show the raw data for each experiment in Phase II. The two columns

Table 2. Raw Data and Calculated Percent Inhibitions for Each Trial

		Raw data			*Percent inhibition*		
	Untouched (U)	*Ambient (A)*	*Control (C)*	*Experimental (E)*	*C/U*[a]	*E/U*	*E/C*
MCF7 (n = 7)	1	513483[b]	370554	523792	367433	−2	28.4	29.9
	2	222617	230787	240883	240555	−8.2	−8.1	.1
	3	154585	157179	161581	145943	−4.5	5.6	9.7
	4	617311	545501	570789	557955	7.5	9.6	2.2
	5	113414	69093	103825	101915	8.5	10.1	1.8
	6	554141	474517	589549	619468	−6.4	−11.8	−5.1
	7	67768	54125	63535	67065	6.2	1	−5.6
	Mean	320474.1	271679.4	321993.4	300047.7	0.2	6.3	4.7
	SEM	87803.2	73848.5	87412.2	83653.4	2.7	5.0	2.1
K562 (n = 7)	8	228691	241775	242937	229458	−6.2	−0.3	5.5
	9	28739	19428	33200	25183	−15.5	12.4	24.1
	10	409636	565457	584563	560361	−42.7	−36.8	4.1
	11	156326	200674	203713	203247	−30.3	−30	.2
	12	301664	148202	295489	248945	2	17.5	15.8
	13	71509	43589	63104	67211	11.8	6	−6.5
	14	89958	109854	112501	101380	−25.1	−12.7	9.9
	Mean	183789.0	189854.1	219358.1	205112.1	−15.1	−6.3	7.6
	SEM	51898.1	69430.3	70788.1	67413.3	7.2	7.9	3.8
MEL 6 (n = 8)	15	151210	x	130329	103358	13.8	31.6	20.7
	16	953	969	1147	1031	−20.4	−8.2	10.1
	17	4034	3926	4742	3504	−17.6	13.1	26.1
	18	21456	21379	21140	19338	1.5	9.9	8.5
	19	18775	17227	18051	12193	3.9	35.1	32.5
	20	15489	8983	8433	12203	45.6	21.2	−44.7
	21	1292	974	1458	1565	−12.8	−21.1	−7.3
	22	9915	8884	11164	13269	−12.6	−33.8	−18.9
	Mean	27890.5	8906.0	24558.0	20807.6	0.2	6.0	3.4
	SEM	17834.2	2990.2	15327.0	12013.8	7.7	8.8	9.1
LNCaP (n = 4)	23	165937	125727	165315	122680	0.4	26.1	25.8
	24	92873	117845	115007	114945	−23.8	−23.8	.1
	25	11797	6165	14262	8151	−20.9	30.9	42.8
	26	14417	14904	17982	11287	−24.7	21.7	37.2
Mean		71256.0	66160.3	78141.5	64265.8	−17.3	13.7	26.5
SEM		36739.9	32205.3	37258.2	31538.6	5.9	12.6	9.5

[a]Percent inhibition for X/Y is the inhibition caused by caused by X relative to Y. Values are calculated with the following formula: $(1 - X/Y) \times 100$.

[b]Values are expressed as counts per minute.

following the raw numbers contain values for percent inhibition calculated by comparing both the control and the experimental to the untouched cells. In the last column, percent inhibition was calculated by directly comparing the experimental cells to the control cells.

An ANOVA demonstrated no significant difference between any of the four groups when looking at them either within a particular cell line, or when looking at the numbers as a complied set (data not shown). One can see in Figure 1 that the differences between the groups are slight when compared to their standard error. On the other hand, for each cell line, the experimental counts were consistently lower than the control counts. Using a paired *t*-test (Table 3) to compare these groups, we found that our practitioner was not able to cause a significant change ($p < 0.05$), although in the K562 and the LNCaP cells, the difference approached significance ($p = 0.08$ for both).

TABLE 3. CONTROL AND EXPERIMENTAL PERCENT INHIBITION (± STANDARD DEVIATION) FOR EACH CELL LINE AND THEIR *p* VALUES CALCULATED WITH A TWO-TAILED PARIED *t*-TEST

Cell line	n	*control*	*Experimental*	p
MCF7	7	.2 ± 7.7	5.0 ± 9.2	0.34
K562	7	−15.1 ± 19.1	−6.3 ± 21.0	0.08
MEL6	8	.2 ± 21.8	6 ± 24.9	0.24
LNCaP	4	−17.3 ± 11.9	13.7 ± 25.3	0.08

We (by means of a chi square test) then asked if inhibition was significantly associated with a treatment by our practitioner (Tables 4A and 4B). The results for all cell lines were combined and subsequently divided into two groups: inhibition or no inhibition. Inhibition was defined using two standards, the experimental error of the assay (5%) and the inhibitory effect of the chemotherapeutic agents used as a positive control (15%). The results of analyses un-

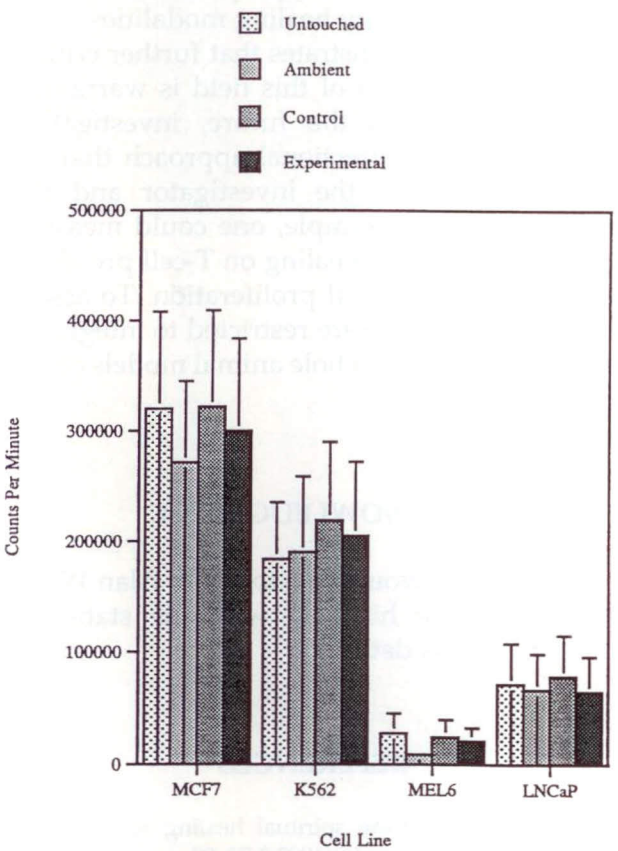

FIG. 1. Mean (±SEM) counts per minute for each of the four groups and each of the four cell lines.

TABLE 4. CHI SQUARE TEST ANALYZED IN VARIOUS WAYS

a) Inhibition is defined as greater than 5% inhibition

$p = 0.012$

	Inhibition	*No Inhibition*
Experimental	16	10
Control	7	19

b) Inhibition is defined as greater than 15% inhibition

$p = 0.024$

	Inhibition	*No Inhibition*
Experimental	8	18
Control	1	25

c) Growth is defined as greater than 5% growth

$p = 0.160$

	Growth	*No Growth*
Experimental	9	17
Control	15	11

d) Growth is defined as 15% growth

$p = 0.349$

	Growth	*No Growth*
Experimental	5	21
Control	9	16

der both criteria are significant ($p = 0.012$ and $p = 0.024$, respectively).

DISCUSSION

Figure 1 demonstrates a trend associating inhibition of tumor cell growth with Yuanji treatment. The chi-square tests establish the nonrandomness of this relationship. The insignificant results of the paired *t*-tests may be attributable to the sample size particularly in the LNCaP and K562 cell lines where $p = 0.08$ for both.

Figure 1 also demonstrates a weakness in the experimental model. We added the untouched group and the ambient group to the study to give us a sense of the precision of the assay and so we could see the effect of moving the cells out of the incubator. We expected the untouched cells, which never left the incubator, to have the highest rate of proliferation as they were grown in optimal conditions. Similarly, we expected the ambient group, kept at room temperature for 5 hours, to have the lowest rate of proliferation. Accordingly, if the ambient and the untouched groups demonstrated similar levels of proliferation, it would be evident that these cell lines are not sensitive to movement and moderate changes in temperature. In fact, for three of the four cell lines, the ambient group did show the least amount of proliferation.

This would suggest that the control (sham) group, which was exposed to ambient conditions for 1 hour, should have proliferated less than the untouched group. However, in two of the four cell lines, the control group showed higher rates of proliferation than the untouched group and thus from our data, it is impossible to make global statements about the effect of removing cells from the incubator and this example illustrates the inherent imprecision in the model that may have contributed to our inability to quantify the energy healing effect.

This imprecision does not invalidate the results of the chi-square test. It is highly improbable that random error alone could generate the association we observed between tumor cell inhibition and our healer's treatment. However, the large random error does preclude us both logically and statistically from making conclusions about the magnitude of the healer's effect.

CONCLUSION

Our study would have been more informative if we could have asked our practitioner to demonstrate both a positive and negative effect on the same cells. We intended to have a second experimental group in which our practitioner would attempt to potentiate tumor cell growth, but he was uncomfortable with the thought of using his abilities to cause "harmful change." In fact, all of the practitioners we interviewed felt that they would only be capable of effecting a therapeutic change. Most practitioners also expressed misgivings about their abilities to affect an isolated biological system like cultured tumor cell as they usually work towards restoring health at the organismal level. Even though simple models lend themselves to rigorous scientific experimentation, they might not be appropriate when evaluating certain energy healing modalities.

This study demonstrates that further critical scientific evaluation of this field is warranted and necessary. In the future, investigators could adopt a bidirectional approach that accommodates both the investigator and the practitioner. For example, one could measure the effect of energy healing on T-cell proliferation versus tumor cell proliferation. To assess energy healers who are restricted to integrated biological systems, whole animal models could be used.

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