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ORIGINAL PAPERS

Resonance, Placebo Effects, and Type II Errors: Some Implications from Healing Research for Experimental Methods

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ABSTRACT

Background: Classical experimental design presupposes that subjects, randomly separated into experimental and control groups, are independent and distinct. Treatments given to the experimental group ought to have no effect on the control group, which functions as a baseline to illustrate "what otherwise would have happened." Any change in the control group is often labeled an "anomaly." Examples of these types of anomalous phenomena can be found in placebo research, which often shows proportional unexpected and unexplained changes in control subjects.

In four previously reported experiments on anomalous healing using "healing with intent" on mice injected with lethal doses of mammary adenocarcinoma (source, The Jackson Laboratories, Bar Harbor, ME; code, H2712; host strain, C3H/HeJ), a high percentage of both experimental *and* control mice exhibited an anomalous healing pattern, most often passing through stages of tumor ulceration to full life-span cure.

Objective: In order to explain tumor regression of control animals, I posit the formation of "resonant bonds," which can link spatially separate groups. Healing given to the experimental animals can result in an unintended treatment to the control animals, producing anomalous healing akin to placebo effects.

Materials and methods: A recently completed experiment at the Terre Haute campus of the Indiana University School of Medicine has produced a successful test of resonance theory. One group of mice (n = 30) was injected with mammary adenocarcinoma cells and randomly divided into a treated group (n = 15) and untreated control group (n = 15). A second group of age-matched controls (n = 25) was left uninjected. Mice from each group were intermittently sacrificed to measure hematologic values and spleen weight.

Results: As predicted by resonance theory, there were few differences between treated and untreated animals from the first group, but there were significant differences between these animals and the age-matched controls.

Conclusions: Some implications for placebo research and the way we normally conceptualize Type II errors will be discussed. Researchers are invited to reanalyze past data in light of resonance theory.

INTRODUCTION

Classical experimental design presupposes that subjects, randomly separated into experimental and control groups, are distinct and independent. Given this presupposition, the internal logic of an experiment is compelling: The random assignment of subjects ensures that the two groups are equivalent; a stimulus (independent variable) is given to one group and not to the other to compare outcomes. High-quality studies are double blinded to ensure that inadvertent experimenter bias does not creep into the results. Control subjects, especially in human subject designs, do not know to which group they have been assigned so that anticipation or belief cannot account for the outcome.

And so, at the end of an experimental protocol, given proper design and proper data analytical techniques, any dif-

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ferences between experimental and control groups must be the result of the given stimulus. If no significant differences occur between groups then we must conclude that the alleged "stimulus" had no real effect.

The logic of this ideal-type design is so well-established that, with only minor variations, it is applied to almost all fields that use experimental methods. Indeed, the randomized, placebo-controlled trial is the "gold standard" of experimental science.[1,2] Researchers are trained to follow proper experimental protocols, and the main criteria for empirical discovery are measurable differences between experimental and control groups.

This paper does not question the logic of experimental design. Rather, it suggests that, under some circumstances, for example, illustrated by placebo effects, the presupposition of experimental and control group independence is questionable. It suggests that this violation can occur via the creation of a "resonant bond" between groups. Resonance, in turn, can result in a macroscopic entanglement of experimental subjects, so that a stimulus given to one group also stimulates the other group.

If the present interpretation has validity, then there are implications for virtually all fields that use experimental design. Methodologically, for example, Type II errors occur when a false null hypothesis is incorrectly accepted. At the end of an experiment, if there is no difference between experimental and control groups because *all* subjects have been affected by a stimulus, then a Type II error of interpretation is likely. Simply put, the researcher will observe equivalent groups and conclude that nothing significant has occurred. This paper suggests that, when resonant bonds are formed, Type II errors will be commonplace.

In addition, some widely observed phenomena previously thought to be anomalous, become natural and expected. The commonly known placebo effect is an example. Currently, there is no explanation within pharmacokinetic theory for placebo effects,[3] and they have befuddled researchers for decades. This paper suggests that a placebo effect will result naturally when resonant bonds are formed.

The idea of resonant bonding among subjects came about from the first author's attempt to understand data from multiple experiments of anomalous healing in cancerous mice. To date the first author has been involved with six formal experiments on the effect of what has generically been called "energy healing" or "healing with intent" on mammary adenocarcinoma at three different institutions, and two informal experiments on methylcholanthrene induced sarcomas at another. The results of four of these experiments on mammary adenocarcinoma have been previously reported,[4–6] in which a significant percentage (87.9%) of cancerous mice were fully remitted by the healing-with-intent techniques. In three of these experiments, the remissions were accomplished by nonbelieving, previously inexperienced volunteers. The addition of a fifth experiment, reported later, brought the combined remission rate up to 91.7%.

These experimental remissions occurred in mice that normally have 100% fatality subsequent to injection, and so appear to be quite anomalous and in need of theoretical explanation. What is perhaps more perplexing, is that, in these previously reported experiments, a significant percentage (69.2%) of the *control* mice, who did not receive any direct healing-with-intent treatment, also remitted. The inclusion of the fifth experiment increased the control remission rate to 80.5% (Table 1). It is these remissions among the control mice which have led this author to the hypothesis of resonant bond formation, and which is the main focus of this paper.

The next sections briefly summarize the results of the first four experiments on mammary adenocarcinoma, with a concentration on the patterns of the control mice remissions. This will lead to the hypothesis of resonant bonding, which was recently demonstrated in an experiment to be reported here for the first time. The final sections deal with the implications of resonant bonding for placebo effects and se-

Table 1. Summary of Experimental and Control Remission Patterns

Experiment #	N	N *Remissions*	*% Remissions*
1—Experimental mice	5	5	100%
Control mice on site	6	4	66.7
2—Experimental mice	7	7	100
Control mice on site	6	4	66.7
3—Experimental mice	10	7	70
Control mice on site	6	3	50
Control mice off site	4	0	0
4—Experimental mice	11	10	90.9
Control mice on site	8	7	87.5
Control mice off site	4	0	0
5—Experimental mice	15	15	100
Control mice on site	15	15	100
Overall experimental	48	44	91.7%
Overall control on site	41	33	80.5%
control off site	8	0	0%

lected methodological issues involved in experimental research.

PREVIOUS RESEARCH ON MAMMARY ADENOCARCINOMA

In four separate procedures at two different institutions, disinterested biologists prepared experimental mice for use in the healing-with-intent research. From the Jackson Laboratories (Bar Harbor, ME) A "standard" mammary adenocarcinoma (code H2712; host strain C3J/HeJ; strain of origin C3H/HeHu) was obtained. The mice received subcutaneous injections of mouse mammary adenocarcinoma tumor cells sufficient to produce fatal tumors. The normal progression after the mouse is injected is the development of a nonmetastatic palpable and visible tumor that grows sufficiently large to crush the internal organs of the host. The conventional literature reports 100% expected fatality within approximately 1 month subsequent to injection.

As previously reported,[4] the healing-with-intent experimental protocol required that the volunteer healers practice mental and "directed energy" techniques taught to us by an experienced healer formerly based in Great Neck, New York. These techniques did not involve focused visualization, meditation, life changes, or belief of any sort. Although they are straightforward, the mental techniques required weeks of practice to master and involved a series of routine mental tasks that were to be practiced simultaneously while placing hands around the standard plastic mice cages for 1 hour per day.

The application of these techniques produced anomalous remission patterns. Approximately 14 days subsequent to injection, or 10 days into the healing procedure, the mice began to develop "blackened areas" on their tumors (Figs. 1 and 2). Approximately 1 week later, the blackened areas "ulcerated" as if they had been split open (Figs. 3 and 4). In some cases, the ulceration grew extremely large, then appeared to implode (not shown), and the wound closed. The mice then lived the normal lifespan of approximately 2 years.

In the figures, the index card notation "A-3" identifies the mouse, and the day number indicates elapsed time since injection. In Figure 1 (Day 14), the tumor is visible on the left posterior dorsal aspect of the mouse. On Day 22 (Figure 2), the tumor is obviously larger but has developed an encrusted area on its surface (the most posterior aspect of the tumor), indicating the earliest sign of tumor regression. Days 28 and 35 (Figures 3 and 4) illustrate the beginning stages of the tumor being internally resorbed. At no time during these remission stages did the mice appear to be sickly in any way.

As an aside, it appeared as though these anomalous healing stages did not proceed in a linear fashion. At times, there were minor day-to-day changes in the appearance of the tumors, and then, at other times, there would be sudden bursts of intensely accelerated healing. In some of these bursts, average-sized tumor ulcerations might completely disappear in only 6 days (Figs. 5 & 6).

Histology indicated that there were viable mammary adenocarcinoma cells present at all stages of remission. Only those mice whose ulcerations were completely closed were free of cancer. Furthermore, reinjection of selected remitted mice did not take, indicating that they had developed immunity to the cancer.

Overall, 29 of 33 (87.9%) experimental mice in the first four experiments went through this process of tumor regression to full cure. The 4 mice who died never developed the blackened area or ulceration.

CONTROL MICE REMISSIONS

The anomalous pattern of remissions in the experimental mice was clearly unexpected, but not as unexpected as the pattern of remissions among the *control* mice. To state the obvious, the healing-with-intent technique was never deliberately applied to these mice. We followed standard experimental protocol by which the mice were randomly assigned to be treated or not, and, in all cases, the control mice were housed separately in another laboratory, and sometimes in another building. Our intent was to keep the control mice separate for the duration of the experiment and to keep them particularly hidden from anyone who knew the healing techniques.

Our curiosity got the better of us, however, and, within several weeks of the first experiment, we violated protocol and visited the control mice. In hindsight, this may have proved fortuitous, because it inadvertently opened the door to unexpected phenomena. What follows focuses on a description of what happened to the control mice in each of the four initial experiments. More details on the experimental mice have been reported elsewhere.[4]

Experiment 1 summary

Seven (7) of 7 experimental mice were cured; 4 of 6 control mice were cured.

Bengston served as the first volunteer healer and followed the protocol of placing his hands around the mice cage for approximately 1 hour per day while practicing the healing techniques. Initially we thought that if the techniques were to be successful that the mice would develop tumors at a significantly slower rate than the control mice. Full cure was not seriously contemplated.

Approximately 10 days after injection, we first observed the growing tumors and ulcerations in the experimental mice, and so we thought that the healing techniques were failing and almost aborted the entire experiment. Then, between 14 and 17 days subsequent to injection, we received a report that 2 of the 6 control mice had died. With this re-

port, we relaxed protocol and Bengston went to see the 4 remaining control mice, who were housed in a separate building. They did not resemble the experimental mice, instead exhibiting normal tumor progression patterns without any blackened area or ulceration. These mice were huddled together and obviously sick and in the last stages of the disease.

After this one brief 10–15-minute visit to observe the 4 remaining control mice, unexpectedly, within days, each mouse developed a blackened area on the tumor, which then ulcerated and imploded to full cure.

Control Problem #1: What changed in the control mice so that they went from being in the last stages of the disease to exhibiting the blackened area to ulceration to full cure?

Experiment 2 summary

Seven (7) of 7 experimental mice were cured; 4 of 6 control mice were cured.

Six (6) control mice were again housed in a separate building. We explicitly set up the protocol to ensure that no-one who knew the healing technique came into contact with any of these mice.

Four (4) very skeptical people served as volunteer healers to be trained—2 were faculty members and 2 were undergraduate students. One (1) of the faculty volunteers for this experiment was in the biology department and 1 was in the geology department. One (1) student was an undergraduate child study major, the other majored in sociology.

The pattern of tumor regression was repeated, and once again, as the experimental mice were going through the various stages, we received word from a graduate assistant that 2 control mice had died. Unbeknownst to the rest of us at the time, the biology faculty member who was a volunteer healer broke protocol and went to visit the 4 remaining control mice several times per week. Once again, the 4 remaining controls then went through the remission pattern to full lifespan cure.

Experiment 3 summary

Seven (7) of 10 experimental mice were cured; 3 of 6 control mice on site were cured; 0 of 4 control mice offsite were cured.

This experiment produced the most puzzling patterns. Three (3) skeptical biology students and 2 skeptical nonbiology students were used as volunteer healers. One of the nonbiology students who had volunteered in the second experiment asked to repeat the procedure in disbelief that she actually did anything previously. After the first experiment ended she confessed to me that she really believed I had secretly been doing a study on student gullibility.

At the time we were attempting to "solve" the problem of the previous control group remissions and also to test whether each volunteer could individually remit mice. Each of the 5 volunteers was given a mouse to treat in the laboratory, and a mouse to treat at home. In the previous 2 experiments, mice exposed to someone who knew the healing techniques apparently had remissions, so we now assumed that, if any of the volunteers could produce a healing effect, then all of the experimental laboratory mice would remit. The new question was whether each person could remit their individual home mice that no-one else would see.

There were 2 control groups. Six (6) mice were kept in the same building in an adjacent laboratory approximately 25 feet away from the experimental mice. Four (4) control mice were sent to a laboratory in another city known only to the experimental biologist who was overseeing the experiment.

The very surprising results were that all of the volunteers were able to remit their home mice. The biology students' laboratory mice died within the expected 1-month period, even as the nonbiologist volunteers' mice, in adjacent cages no more than a foot away, remitted to full cure.

Against protocol, and after 3 control mice had died, the controls were "discovered" by the biology students in an adjacent laboratory, and they subsequently looked in on these mice regularly to observe tumor progression. The 3 remaining control mice observed by the biology students then went through the remission pattern to full cure, even as their experimental mice died in an adjacent laboratory. The 4 control mice sent to another city all died within the expected timeframe.

Control Problem #2: Why were the biology students able to remit their home mice but not their laboratory mice?

Control Problem #3: If the nonbiology students were able to remit their laboratory mice, why were the adjacent experimental mice of the biology students not also remitted as in previous experiments?

Control Problem #4: If the biology students were *not* able to remit their laboratory mice, why did the control mice in the adjacent laboratory remit, as the biology students were the only ones who knew the healing technique to see these mice?

Control Problem #5: Why did the control mice sent to another city die, unlike some control mice in the adjacent laboratory?

Experiment 4 summary

Ten (10) of 11 experimental mice were cured; 7 of 8 control mice on site were cured; and 0 of 4 control mice off site were cured.

Six (6) student volunteers were used, including 1 of the biology students who had failed to remit his laboratory mouse in the previous experiment. Two (2) other students who had done previous experiments volunteered because they were highly skeptical about their earlier results. Three (3) new, nonbiology students were also selected.

As in the third experiment, 2 control groups were used, 1 housed in an adjacent laboratory and another sent to a city

FIG. 1. Typical mouse 14 days after injection.

FIG. 4. Thirty-five (35) days after injection.

FIG. 2. Twenty-two (22) days after injection.

FIG. 5. Rapid healing sequence 1—day 22

FIG. 3. Twenty-eight (28) days after injection.

FIG. 6. Rapid healing sequence 2—6 days later (day 28)

known only to the experimental biologist. This time we did not even bother to ask the volunteers to avoid looking in on the adjacent laboratory, which housed the control mice.

All but 1 experimental mouse went through the remission pattern, as did all but one on-site control mouse. The control mice sent to another city all died within the expected timeframe. By this time, these patterns of remission came to be fairly predictable, even as we had no viable theory as to the mechanism or method by which mice actually got cured.

Control problem #6: Why did the biology student's laboratory mice remit in this experiment but not in the previous experiment?

Control problem #7: What was different about the 1 control mouse who died? (And, what was different about the one experimental mouse that died?)

THE HYPOTHESIS OF RESONANT BONDS

It should be apparent that within the presuppositions of conventional experimental design (i.e., that experimental and control groups are independent), these patterns of remission and nonremission defy logic. Ignoring for the moment the fact that the actual cause of *any* of the remissions remains essentially unexplained, the control mice, whom never directly received any treatment, should not have remitted.

Almost all of the seeming paradoxes of these remissions disappear if we allow for the possibility of "resonant bond formation" and "resonant bond dissolution," which may serve to entangle or *de*-entangle subjects. Certainly the notion of "entanglement," although still quite mysterious, is widely accepted and hailed for its predictive power on a quantum level in conventional physics.[7] What is being suggested here, however, is a form of entanglement on the macroscopic level.

Macroscopic entanglement, per se, is not a new idea,[8] and has been used to help explain experimental results on homeopathy.[9,10] Physicists are becoming more open to macroscopic entanglement, at least in principle.[11,12] Experiments that attempt to study the transition from the quantum to the classical world have demonstrated that molecules with more than 100 atoms can be made to interfere.[13]

In the "alternative" scientific literature, macroscopic entanglement has also been observed in previous experimental research. Tiller's et al.'s work[14,15] on "conditioned laboratory spaces," for example, reports of machines that have successfully been imbued with "intention" by expert meditators becoming information-entangled with each other so that the "control is lost." Jahn and Dunne's extraordinary experiments on the ability of human intent to alter the output of random event generators (REGs) at the Princeton Engineering Anomalies Research (PEAR) laboratory produced increased statistical success when volunteers subjectively reported a "resonant bond" with the machines.[16,17]

In both of these latter examples, the role of *consciousness* is paramount. Conventional science, of course, has had a bias toward a materialistic/reductionist approach to phenomena, which downplays the role of consciousness,[18,17] so that consciousness, if discussed at all, becomes a mysterious *effect* of other variables rather than a causal agent unto itself. In Tiller et al.'s work and Jahn and Dunne at the PEAR laboratory, the role of consciousness becomes central to the production of the various phenomena themselves. Perhaps it is because of this that their work is often labeled perjoratively "anomalous."

A resonance hypothesis also suggests that consciousness plays a crucial role in bond formation and dissolution, so that, on a macroscopic level entire organisms can become entangled, potentially blurring the distinction between experimental and control subjects. Consider two possible hypotheses: (1) shared experiences among experimental subjects can "bond" them together resonantly; and (2) consciousness itself, including that of the experimenter, can delimit the boundaries of experimental subjects, effectively defining those who are "in" and those who are "out." Those who are "in" form something akin to a larger "collective," analogous to those formed by colonies of insects, flocks of birds, and schools of fish. The resonance hypothesis extends this analogy to propose that what happens to 1 bonded subject affects directly what happens to the other. Also consider that these bonds are not permanent, but rather can also be broken, just as fish or birds can be broken off from larger schools or flocks.

The first hypothesis may initially appear similar to Sheldrake's hypothesis of morphic resonance,[19,20] in which members of a species come to share information with one another even though they are physically separated. Indeed, Sheldrake posits a "morphogenetic field" of memory generated from members. From this field, similar members can draw information and skills, so that successive generations of a wide variety of species learn tasks over diminishing intervals.

While, on the surface, there are parallels to resonant bonding, Sheldrake does not address how smaller collections of individuals of a species form delimited boundaries, so that some gain the necessary knowledge and some do not. In the mice experiments, whether we allow for the hypothesis of morphic resonance or a more generalized "field effect" produced by the healing-with-intent techniques, the patterns of selective remission remain unexplained. In the third experiment, for example, the biology students' mice died in the laboratory, indicating that they were unable to stimulate healing successfully. But, if the control mice, whom never received direct healing, were actually healed by some sort of field effect by the nonbiology students, then it follows that the biology students' experimental mice, located right next to the successfully

healed nonbiologists' mice, should have remitted by that same field. They were not.

The first hypothesis of resonant bond formation suggests that shared experiences, such as being inbred together, living together, and being housed together, will increase the probability of forming resonant bonds. Like schools of fish or flocks of birds whom have become "attuned" to one another, resonance can bond together formerly independent parts so that they begin to act as a collective.

The second hypothesis suggests that consciousness itself can form resonant bonds. Certainly, it is a universal experience that people feel emotionally "bonded" to selective people, places, and things, and that the experiential strength of that bond waxes and wanes over time. If we allow that these subjective experiences might reflect a real phenomenon of resonant connection, there are implications for experimental protocols. Just as human operators increase their success rate in producing statistical deviations in REGs at the PEAR laboratory when they feel bonded to the machine,[16] the conscious emotional experience of connection can resonantly bind and delimit experimental subjects. Conversely, the experience of emotional ebb and disconnection will be accompanied by a reduction in the strength of the resonant bond, so that what happens to one will have a diminished effect on the other.

Consider the previously outlined problems with the control mice from the perspective of resonant bonding via consciousness. The first "control problem" occurred in the first experiment, in which 2 control mice in another building had died, and the 4 remaining mice were following the normal tumor progression toward death until they were "visited" for a brief time. The subjective experience of seeing the control mice suffering produced an empathic bonding, so that future treatments to the experimental mice also inadvertently included treatments to the remaining controls. The resonant connection was made through the consciousness of the experimenter.

The second through fifth control problems involve the third experiment. Why were the biology students able to remit their home mice but not their laboratory mice? In the biology students' subjective logs, they reported feeling a very great sense of unease when they were standing around in their white coats while putting their hands around a cage in the middle of a laboratory. These were, after all, undergraduate biology students being trained in conventional biology, with all that this implies. At the very least, they reported feeling embarrassed, with an underlying fear of peer ridicule. At home, they were relaxed and at ease. I suggest that the emotional sense of fear and embarrassment served to break the resonant bond that their mice shared with the other mice. This would also explain the third control problem of why the nonbiology students, who were able to remit their mice in the laboratory, had no effect on the biology students' mice. The biology students' mice had lost the resonant bond with the group.

Control problem 4 asked how the 3 remaining control mice housed in an adjacent laboratory could be remitted if only seen by the biology students. The resonance solution is akin to the first control problem. The biology students who saw the remaining control mice were under no pressure to heal and under no fear of embarrassment for standing around and observing these mice. These students simply observed, and reported in their logs that, while treating their home mice, the students often thought of the mice in the adjacent laboratory, thus "pulling in" the control mice to the larger resonant group. This sense of ease also explains control problem six, why a biology student was unable to remit mice in the laboratory in the third experiment but was able to do so in the fourth experiment. The fourth experiment was carried out over the summer in an otherwise empty laboratory. The biology student reported experiencing camaraderie with other volunteers, who often came to the laboratory together for companionship and mutual support. No one else ever used the laboratory; hence, there was no embarrassment or sense of unease.

Control problem 5 presents a different sort of problem. That is, why did the control mice sent to another city die, unlike some control mice in an adjacent laboratory? There are at least three (speculative) possibilities. The physical act of shipping to another city with its associated stressors to the mice may have served to break the bond with the other mice. Perhaps distance matters. Or, perhaps the fact that no one who knew the healing techniques ever came into contact with these mice meant that they were never resonantly bonded in the first place. Anecdotally, the last explanation is the most problematic. In other informal experiments there have been second control groups not known to the volunteer healer, and those mice followed the same patterns as control groups who were known to exist. At this stage, more work is needed to determine whether stressors or distance can sever resonant bonds.

Control problem 7 remains unsolved. That is, if the healing techniques work, why do some mice die anyway? Certainly this same question can be asked in virtually all types of conventional research. Explanations of probabilistic natural variation in biologic systems are commonly assumed.

AN EXPERIMENTAL ILLUSTRATION OF RESONANCE

In a recently completed experiment at the Terre Haute campus of the Indiana University School of Medicine, using the same mammary adenocarcinoma model, 30 mice were subcutaneously injected (0.2–0.3 mL) with H2712 mouse mammary adenocarcinoma tumor cells (10^5 cells/mL diluted in RPMI-1640 cell culture medium and 20% normal mouse serum) and split into experimental (n = 15) and control (n = 15) groups. There was an additional group of mice (n = 25) who were not injected and served as age-matched controls. The 3 groups were housed in separate rooms in the animal facility.

The treatment model differed from previous experiments in that: (1) treatment began 1 day after injection rather than 3 days after injection; (2) the mice received a limited amount of direct "healing with intent," namely 3 1-hour sessions over 2 days; and (3) the balance of the treatment was done by "distant healing" from approximately 760 miles away by 2 people trained in the healing techniques. These distant healings involved the same healing techniques, except that hands were not placed directly on the sides of the cages.

At the beginning of the experiment and at 5, 9, and 13 weeks postinjection, 5 animals from each group were sacrificed. Hemoglobin levels were measured, and spleens were collected and weighed as an indicator of immune activation.

Conventional biologic research dictates that any evidence of successful intervention of the healing-with-intent techniques would be found in the *difference* between the experimental and control groups. The resonance hypothesis would predict that *if the healing techniques were to be successful, then the experimental and control mice would exhibit similarities.*

The results supported the resonance hypothesis. With palpation and visual inspection, there were no tumors in either the experimental or control groups, and (obviously) none in the age-matched controls. At 5 weeks, hemoglobin was marginally higher ($p = 0.05$) in experimental and control groups as compared to the uninjected group (Fig. 7). At all time points, spleen weight was significantly higher than in the age-matched controls (Fig. 8). *At weeks 9 and 13, there was no significant difference between the experimental and control groups.*

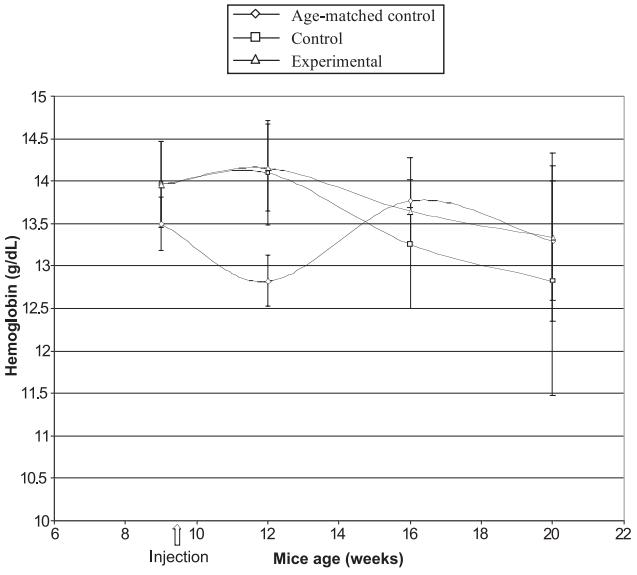

FIG. 7. Hemoglobin comparisons.

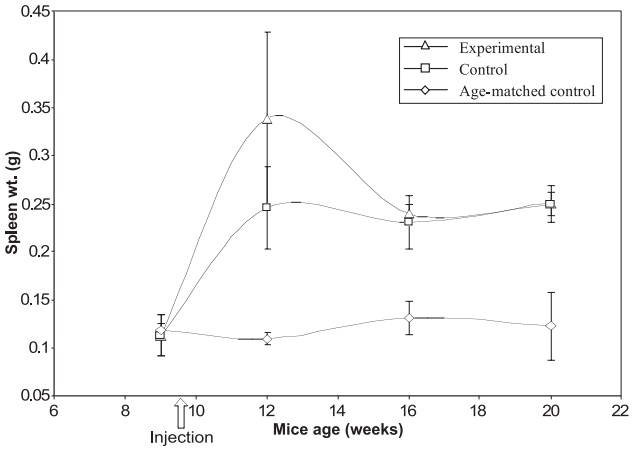

FIG. 8. Spleen Weight comparisons.

RESONANCE IMPLICATIONS— TYPE II ERRORS

Statistical analyses begin with the generation of a null hypothesis (i.e., that nothing significant has happened). Tests for statistical significance indicate the probability (p-value) of the results occurring if the null hypothesis is true. By convention, statistical significance means that it is unlikely (a p-value less than 0.05) that the test results would have occurred by chance alone, and therefore the null hypothesis is probably false.

As previously discussed, Type II errors in research occur when a false null hypothesis is incorrectly accepted. In simple terms, something unlikely or important has happened but the null hypothesis has not been rejected.

Figure 8 above might serve as an illustration. The experimental and control groups converge to a single point of spleen weight, which is a measure of immune activation. With no difference between groups, the conventional interpretation would be that nothing significant has occurred, when, in reality, something has been stimulated in *both* groups of mice.

Type II errors can also be easily illustrated by the previously discussed problems with the control groups. A visual inspection comparing selected experimental and control mice at various days subsequent to injection can show remarkable similarities. (See Figs. 9–11.)

It should be noted that there is substantial variance in the rate at which individual mice go through the remission pattern and that these pictures have been selected because of their similarities. Nonetheless, the aggregate conventional interpretation, a comparison of experimental and control groups, would result in a Type II error, although *none of these mice should have gone through a pattern of remission.*

FIG. 9. Comparison of Experimental and Control Mice at Day 14

FIG. 10. Comparison of Experimental and Control Mice at Day 38

FIG. 11. Experimental and Control Fully Cured

RESONANCE IMPLICATIONS—PLACEBO EFFECTS

Placebos have had an interesting and controversial history. Their effects were first ignored, then treated as a problem contaminant to be controlled, and, only relatively recently, investigated as variables of interest in their own right.[21] Fifty (50) years ago, the idea that an inert pill could produce physiologic change was unthinkable.[22] While there is no consensus on exactly how to define placebos or their effects, at present, there is little controversy that, however defined, they *do* work. Indeed, there have been serious scientific attempts to come to terms with placebo effects. Yet, significant controversies remain regarding how they work (mechanism of action), to what degree they work (explained variance), and under what circumstances they work.[21]

Conventional logic is stymied in the face of fairly common claims that placebos duplicate up to 80% of the effectiveness of conventional drugs[23] and can even mimic the effects of some conventional surgery.[24] In fact, placebo effects are so powerful at reproducing the effects of conventional drugs that tests of the efficacy of new treatments have had to alter methodologies in randomized clinical trials. Investigators have now begun not necessarily to show that a new treatment is superior to a placebo (superiority trial), but rather that it is not less effective than another (noninferiority trial) existing treatment.[22] Without this methodological shift, Type II errors would be rampant.

There are additional complications in that even in double-blinded studies, the clinician's knowledge of the range of possible treatments may be transmitted to the patient and influence placebo efficacy,[25] and even variation in the personality of the investigator can produce variation in the strength of the placebo effect.[26]

It is not the intent of this paper to list comprehensively all of the anomalous outcomes of placebo investigation. Detailed compilations can be found elsewhere.[27,28] It is simply worth repeating that nothing in pharmacokinetic theory can account for the placebo effect.[3]

The similarity of the placebo problem and the control-group problem in the healing research reported here is obvious. In both, conventional thinking and hence explanation, seems to defy logic. After all, if no active agent is administered, how can there be an active effect? If no healing technique is applied, how can there be remissions?

With a conceptual shift toward resonance, placebos begin to take on a new light. Among the great mysteries of placebos is the fact that their effect is proportional to the strength of the experimental treatment.[29] Perhaps this is so because the placebo group is *not* independent of the experimental group, but is actually part of a larger bonded collective. Perhaps the question needs to be reformulated in terms of the conditions under which resonant bonds form and resonant bonds are broken. Perhaps as in the healing research, a treatment given to the experimental group results in an *actual* treatment being administered to the control (i.e., placebo) group. Within the resonance hypothesis a proportional effect becomes expected, even while the mechanism by which resonant groups are formed remains a mystery.

CONCLUSIONS AND SUGGESTIONS FOR FUTURE RESEARCH

A conceptual shift toward interpreting experimental results through the prism of resonant bond formation and destruction may be worthwhile in helping understand many anomalies found in previous research. While the long-term goal is the identification of the mechanism by which bonding may occur, at this preliminary stage, a great deal of work is needed to determine the conditions under which bonds are strengthened and weakened. Researchers are encouraged to reexamine their old data within the framework of resonance to determine whether these phenomena are as extensive as they now appear to be (e.g., placebos). This reexamination needs to broaden the question from the difference between experimental and control subjects to inquire more generally about the difference between experimental subjects and "what ought to have happened." This will involve not only a conceptual shift, but a methodological one as well. Experimental models which have well established baselines are a natural place to begin.

Based on the data presented here, several testable hypotheses can be advanced. Resonant bonds will be created and sustained by (1) more interaction among subjects; (2) more emotional engagement of the subjects in the research; and (3) greater emotional connection of the experimenter to the subjects. Researchers are encouraged to pay special attention to the role of consciousness in the production and destruction of resonant bonds, including the oft neglected subjective impressions of subjects *and* experimenter.

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