# Placebo Psychotherapy: Synonym or Oxymoron?

Irving Kirsch
*University of Plymouth*

Contrary to some recent claims, the placebo effect is real and in some cases very substantial. Placebo effects can be produced or enhanced by classical conditioning, but consistent with virtually all contemporary conditioning theories, these effects are generally mediated by expectancy. Expectancy can also produce placebo effects that are inconsistent with conditioning history. Although expectancy also plays an important role in psychotherapy outcome, the logic of placebo-controlled trials does not map well onto psychotherapy research. The idea of evaluating the efficacy of psychotherapy by controlling for nonspecific or placebo factors is based on a flawed analogy and should be abandoned. © 2005 Wiley Periodicals, Inc. J Clin Psychol 61: 791–803, 2005.

Keywords: placebo; placebo effect; psychotherapy; expectancy; conditioning

A placebo is a sham treatment that may be used clinically to placate a patient or experimentally to establish the efficacy of a drug or other medical procedure. The placebo effect is the effect produced by administering a placebo. In addition, active medications may produce placebo effects as well as drug effects, and these may be additive. In this case, the placebo effect is that portion of the treatment effect that was produced psychologically, rather than through physical means.

Typically, placebos are physically inert substances that are identical in appearance to an active drug. Occasionally, active substances are used as placebos. Active placebos have side effects that mimic those of the drug being investigated, but do not possess the physical properties hypothesized to produce the beneficial treatment effect. Active placebos are used to prevent patients from using the sensory cues provided by side effects to deduce the condition to which they have been randomized.

Placebo effects are not limited to drug treatments. Any medical procedure can have effects due to the physical properties of the treatment and effects due to its psychological

Correspondence concerning this article should be addressed to: Irving Kirsch, Faculty of Health and Social Work, University of Plymouth, Drake Circus, Plymouth, PL4 8AA, United Kingdom; e-mail: irving.kirsch@plymouth.ac.uk.

JOURNAL OF CLINICAL PSYCHOLOGY, Vol. 61(7), 791–803 (2005) 
Published online in Wiley InterScience (www.interscience.wiley.com). DOI: 10.1002/jclp.20126

properties. Just as the effects of the physical properties of a medication can be tested by comparing its effects to those of a sham medication, so too the physically produced effects of other medical procedures can be established by comparison with sham procedures (e.g., sham surgery). For example, real surgery and sham surgery have been compared in the treatment of angina (Cobb, Thomas, Dillard, Merendino, & Bruce, 1959; Dimond, Kittle, & Crockett, 1960) and osteoporosis of the knee (Moseley et al., 2002). In both cases, the effects appear to be due to the psychological properties of the treatment, rather than to the surgical procedures themselves.

When the terms *placebo* and *placebo effect* are confined to controls for pharmacological substances and other physical interventions, they are not at all difficult to define. However, insurmountable problems arise when attempts are made to extend the concept to psychotherapeutic procedures (see Kirsch, 1978). In this article, I argue that placebo effects are powerful, despite recent claims that they are not. Nevertheless, the idea of evaluating the efficacy of psychotherapy by controlling for nonspecific or placebo factors is based on a flawed analogy and should be abandoned.

## The Power of Placebo

The word *placebo* is Latin for "I shall please," and for many years placebos were thought to placate patients, but not to heal them. The double-blind clinical trial, which has become the gold standard for evaluating new medications, was promoted in the 1930s by Gold and his colleagues (Could that be why it is called the "gold standard"?), but it was not until the 1960s that the power of the placebo became widely recognized (Shapiro & Shapiro, 1997). More recently, the placebo effect has once again been questioned. Hróbjartsson and Gøtzsche (2001) reported a meta-analysis in which they purported to compare the placebo effect to no treatment. Their results indicated a small but significant placebo effect in studies reporting continuous outcome scores and a nonsignificant effect in studies reporting dichotomous outcomes. However, there is good reason to believe that this is a gross underestimate of the placebo effect.

Hróbjartsson and Gøtzsche (2001) analyzed clinical trials of treatments for a motley collection of disorders, including the common cold, alcohol abuse, smoking, poor oral hygiene, herpes simplex infection, infertility, mental retardation, marital discord, fecal soiling, Alzheimer's disease, carpal tunnel syndrome, and undiagnosed ailments. The placebos in these clinical trials were not limited to conventional pill placebos, but instead included relaxation (classified as a treatment in some of the studies and as a placebo in others); leisure reading; answering questions about hobbies, newspapers, magazines, favorite foods, and favorite sports teams; and talking about daily events, family activities, football, vacation activities, pets, hobbies, books, movies, and television shows. Not surprisingly, the most reliable finding in the Hróbjartsson and Gøtzsche (2001) meta-analysis was that there was substantial and significant heterogeneity in the outcomes produced by placebo. In other words, some placebos were significantly more effective than others, some disorders were more amenable to placebo treatment than others, or both. This, in itself, validates the existence of a placebo effect. One placebo cannot be more effective than another, unless placebos are capable of producing an effect.

Certainly, some conditions are not amenable to placebo treatment. The effects of placebo antihyperglycemics, for example, are negligible or nonexistent. Conversely, there are substantial data demonstrating the effectiveness of placebo treatment for some responses. Numerous studies not included in Hróbjartsson and Gøtzsche (2001) meta-analysis have shown that placebo analgesics, tranquilizers, stimulants, and alcohol produce effects beyond those observed in untreated control conditions (see citations in Kirsch, 1997).

The effect of inert placebo pills on depression is particularly strong. A meta-analysis of published studies indicates a change of 1.16 *SD* on measures of depression following administration of placebo antidepressants, compared to a change of 0.37 *SD* among untreated controls (Kirsch & Sapirstein, 1998). These data indicate a placebo effect size of 0.79 *SD*.[1] This is a powerful effect by any standard, but it may be an underestimate. Most clinical trials of antidepressant medication fail to show a significant difference between drug and placebo (Kirsch, Moore, Scoboria, & Nicholls, 2002). Almost all of the studies showing a significant difference have been published, whereas most of those failing to find a difference have been withheld from publication (Melander, Ahlqvist-Rastad, Meijer, & Beerman, 2003).

There is yet another reason for suspecting that the data reported by Kirsch and Sapirstein (1998) underestimate the placebo effect on depression. Included in the database they analyzed were four medications (amylobarbitone, lithium, liothyronine, and adinazolam) that are not antidepressants. The change produced by these active placebos (1.69 *SD*) was greater than that of inert placebos and as great as that observed in patients given selective serotonin reuptake inhibitors (SSRIs) (1.68 *SD*) and tricyclics (1.52 *SD*). This suggests that the response to antidepressant medication may be entirely a placebo response.

Recently, a methodology has been developed for assessing the placebo effect without the use of placebos (Benedetti et al., 2003). Participants give permission to receive a medication with or without foreknowledge of the onset of administration. Medication is subsequently administered intravenously, in some cases with the patient's knowledge and in other cases without any signal. Using this methodology, Benedetti and colleagues found that substantial proportions of the effects of morphine on pain, stimulation of the subthalamic nucleus in Parkinsonian patients, and β-blockers (propranolol) and muscarinic antagonists (atropine) on heart rate were due to the placebo effect. In addition, they reported that the effect of diazepam on postoperative anxiety was entirely a placebo effect. Hidden infusions of diazepam were totally ineffective in reducing postoperative anxiety.

These are not the only data indicating significant placebo effects. Just as inclusion of a placebo control is only one method of evaluating the effects of medical treatments, the inclusion of a no-treatment control group is only one method of evaluating the placebo effect. Medical treatment effects can be inferred when different doses of the same drug produce different effects or when a particular treatment is found to be significantly more effective than an alternative treatment. Similarly, placebo effects can be inferred when different placebos or apparent doses of the same placebo produce significantly different effects or when the effects of a placebo vary as a function of the information provided to the person to whom it is administered. Effects of this sort have been reported in a number of studies. For example:

- Asthmatic patients have been shown to exhibit bronchoconstriction after inhaling a placebo described as a bronchoconstrictor and brochodilation after inhaling a

[1]Not surprisingly, these data provoked considerable controversy and were challenged on a number of grounds, including restriction of the analyses to patients who had completed the trials, the limited number of clinical trials assessed, the methodological characteristics of those trials, and the use of meta-analytic statistical procedures (Klein, 1998). These criticisms provoked replication with a data set to which the objections did not apply, namely, the data submitted by pharmaceutical companies to the U.S. Food and Drug Administration (FDA) in the process of obtaining approval of antidepressant medications (Kirsch et al., 2002). The replication had the advantage of including unpublished as well as published studies and yielded the conclusion that Kirsch and Sapirstein (1998) had actually underestimated the placebo component of antidepressant medication.

placebo described as a bronchodilator (Luparello, Lyons, Bleeker, & McFadden, 1968; McFadden, Luparello, Lons, & Bleecker, 1969; Neild & Cameron, 1985; Spector, Luparello, Kopetzky, Souhrada, & Kinsman, 1976).

- Placebo morphine is considerably more effective than placebo Darvon, which, in turn, is more effective than placebo aspirin (Evans, 1974). In each case, the placebo is about half as effective as the pharmacologically active drug. Similarly, placebos produce more pain relief when given after a more potent drug than they do when given after a less potent drug (Kantor, Sunshine, Laska, Meisner, & Hopper, 1966). Thus, the effectiveness of a placebo pain reliever varies as a function of its believed effectiveness.
- Placebo and active analgesics are more effective when presented with a well-known brand name (Branthwaite & Cooper, 1981).
- Placebo injections are more effective than placebo pills (de Craen, Tijssen, de Gans, & Kleijnen, 2000).
- The color of a placebo can influence its effects (reviewed in de Craen, Roos, de Vries, & Kleijnen, 1996). When administered without information about whether they are stimulants or depressives, blue placebo pills produce depressant effects, whereas red placebos induce stimulant effects (Blackwell, Bloomfield, & Buncher, 1972). Patients report falling asleep more quickly after taking a blue capsule than after taking an orange capsule (Luchelli, Cattaneo, & Zattoni, 1978). Red placebos seem to be more effective pain relievers than white, blue, or green placebos (Huskisson, 1974; Nagao, Komia, Kuroanagi, Minaba, & Susa, 1968).
- Finally, the magnitude of the placebo response has been shown to vary as a function of the dose that the person is asked to consume (de Craen, Moerman, Heisterkamp, Tytgat, Tijssen, & Kleijnen, 1999; Kirsch & Weixel, 1988).

Taken together, these data provide ample documentation of the presence of a powerful placebo effect.

## Specifiying Nonspecifics: Explaining the Placebo Effect

There are placebo effects and in some cases they can be very powerful, but how are those effects produced? Currently, there are two prominent hypotheses: classical conditioning and response expectancy (see Stewart-Williams & Podd, 2004, for a comprehensive review). According to the conditioning hypothesis (Herrnstein, 1962), active treatments function as unconditional stimuli (USs) and the vehicles in which they are administered (i.e., pills, capsules, syringes) are conditional stimuli (CSs). Repeated administration of active treatments constitute conditioning trials, during which placebo effects mimicking the drug effects (the unconditional responses; URs) are acquired as conditional responses (CRs). According to the response expectancy hypothesis (Kirsch, 1985), placebo effects are produced by the self-confirming nature of response expectancies. Response expectancies are anticipations of one's own automatic responses to various stimuli. For example, a person may expect to feel more alert after drinking a cup of coffee and to feel less pain after taking pain medication. Unlike stimulus expectancies (i.e., expectancies about the external world), response expectancies are directly self-confirming in that they tend to produce the anticipated experiences. Of course, experience is also affected by many other factors. For example, caffeine can alter alertness and morphine can reduce pain independent of expectancy (e.g., Benedetti et al., 2003; Kirsch & Rosadino, 1993). Subjective experience depends on a combination of external stimulus factors and internal expectancies.

Conditioning and response expectancy hypotheses are often presented as opposing theories, but this is based on assumptions about classical conditioning that are now widely regarded as outmoded (see Kirsch, Lynn, Vigorito, & Miller, 2004; Rescorla, 1988):

> Contemporary mechanistic accounts of classical and operant conditioning typically involve the hypothesis that direct associations between the stimulus and outcome representations or the response and outcome representations are formed during conditioning. Obviously, the outcome representations activated by these associations constitute a low-level form of outcome expectancy. Thus, there is now virtually universal agreement that conditioning involves the production of expectancies. (Kirsch et al., 2004)

In addition, there are experimental studies demonstrating that conditioned placebo responses are mediated by expectancy. Voudouris, Peck, and Coleman (1985, 1989, 1990) reported a number of studies demonstrating that classical conditioning procedures can enhance placebo analgesia. The placebo effect was enhanced in these studies by surreptitiously lowering the intensity of a pain stimulus whenever part of the body treated with a placebo anesthetic was stimulated. The lowered intensity of the pain stimulus was the US and the placebo was the CS. This procedure increased the pain-reducing effect of the placebo when it was subsequently tested without lowering the intensity of the pain stimulus.

Although the Voudouris et al. (1985, 1989, 1990) studies convincingly demonstrate conditioned enhancement of placebo pain relief, they do not assess the degree to which this conditioning effect is mediated by expectancy. Presumably, the experience of less pain following treatment would lead participants to expect pain reduction when the treatment was re-administered. This mediational hypothesis was tested by Montgomery and Kirsch (1997). Recall that in the Voudouris et al. studies, the intensity of the pain stimulus was lowered surreptitiously. Participants did not know that the intensity was lowered, and they therefore attributed the reduction in pain to the effect of the supposed topical anesthetic. In addition to replicating this procedure, Montgomery and Kirsch added an "informed pairing" control condition, in which participants were told that the intensity of the stimulus was being lowered. As in the condition replicating the original studies, participants in the informed pairing condition were given trials in which reduced pain was paired with the application of a placebo. However, they were also given accurate information about how the reduction in pain was being produced. This verbal information completely reversed the effect of conditioning trials on the placebo response. In addition, regression analyses indicated that the effects of conditioning trials were mediated completely by participants' verbally rated expectancies.

Taken together, the Voudouris et al. (1989, 1990) and the Montgomery and Kirsch (1997) studies demonstrate two things. First, classical conditioning can enhance placebo pain reduction. Second, this conditioned enhancement of the placebo effect is mediated by response expectancy. There are other data, however, indicating that placebo effects can occur without or even in opposition to conditioning. For example, the CR to a stimulus that has been paired with morphine in laboratory animals is an increase in pain sensitivity (Siegel, 1983). Nevertheless, the human response to placebo morphine is pain reduction. Similarly, although tranquilizers like chlorpromazine reduce activity levels, the CR to stimuli with which they have been paired is to increase activity levels in laboratory animals Pihl and Altman (1971). Thus, the pain reducing effects of placebo morphine and the sedating effects of placebo tranquilizers cannot be conditioned responses. If anything, they occur despite the opposing effects of conditioning. These data are especially important because the effects of placebo analgesics and tranquilizers are particularly well established.

To summarize, the bulk of the data are consistent with the hypothesis that placebo effects in human beings are generally dependent on the activation of response expectancies. Classical conditioning is one source of these expectancies, but they can also be affected by other sources of information. Therefore, there are some cases in which the CR is inconsistent with the response expectancy; in some of those instances, the expectancy appears to override the conditioning effect (Montgomery & Kirsch, 1988; Siegel, 1983).

## Extending the Placebo Concept to Psychotherapy

Given the importance of placebo effects in medical interventions, it was only natural that researchers began to question the degree to which the effects of psychological interventions might be placebo effects. To answer that question, studies were designed to include placebo psychotherapies, the effects of which can be compared to the genuine treatment (e.g., Paul, 1966). At first glance, this strategy seems reasonable, but, in fact, it is very problematic. There are both practical and conceptual problems with attempts to extend the placebo concept from the medical setting to the psychotherapeutic setting. Practically, it cannot be done; conceptually, it makes no sense to try.

### *The Practical Problem*

In medical research, the purpose of using a placebo is to control for the psychologically produced effects of a particular treatment. To do this, it is important that the placebo have the same psychological properties as the treatment it is replacing. With pills and injections, this is easy. All that is needed is to omit the active ingredient. Thus, the placebo is the same size, shape, color, taste, and smell as the active medication. It is also provided with the same label and information.

With psychological treatments, constructing a matching placebo is impossible. By definition, a psychological treatment has no active physical properties. All of its ingredients are psychological. If a psychological treatment contained the same psychological properties as the real treatment (i.e., if the therapist used the same words and procedures), it would no longer be a placebo or a control condition of any other kind. Instead, it would be the treatment.

As a result, procedures used as placebos in psychotherapy research are usually very different from the psychotherapies to which they are being compared. Among the procedures used as placebos in psychotherapy research are the following: listening to stories, reading books, attending language classes, viewing films, participating in "bull" sessions, playing with puzzles, sitting quietly with a silent therapist, and discussing current events (see Prioleau, Murdock, & Brody, 1983, pp. 280–282). Indeed, simply being placed on a waiting list has been labeled a placebo (e.g., Sloane, Staples, Cristol, Yorkston, & Whipple, 1975).

### *The Conceptual Problem*

The function of a placebo in medical research is to control for the psychological effects of administering a treatment. This is vital, because without doing so, one cannot know whether the physical properties of the medication have any effect on the condition being treated. With psychotherapy, the situation is altogether different. We do not need placebo controls to determine whether the physical properties of the treatment have an effect. We know the answer to that question without doing any studies at all, and that answer is no. Most psychotherapies do not have physically active properties. Their substance is words,

but it is not the sound of the words having an effect. Rather, it is their meaning. Moerman and Jonas (2002) have defined the placebo effect as a patient's response to the meaning of a treatment. In this sense, most psychotherapy is a placebo by definition; it is an effective treatment because of its psychological properties rather than its physical properties.

There is a problem with identifying psychotherapy with the placebo effect. A placebo is something that is sham, fake, false, inert, and empty. Psychotherapy is none of these. In this sense, it is different from medical placebos, and it does not deserve the pejorative connotations associated with the term.

A common assumption is that some psychotherapies are indeed shams, whereas others are not. Placebo controlled psychotherapy outcome research is attempted precisely because one wants to know whether a particular psychotherapy is merely a placebo, that is, whether it—in contrast to other psychotherapies—is a sham. But what does it mean to say that a particular psychotherapy is a placebo? It cannot mean that the treatment is ineffective, because placebos can be effective. If this were not the case, there would be no need for placebo controls in any kind of outcome research, whether for pills, surgery, or psychotherapy. The general assumption seems to be that psychotherapy is a placebo if its effects are nonspecific. This is generally stated as if the meaning of the term *nonspecific* were clear. But it is not clear at all. Specifically, it is not clear how the effects of placebos are any less specific than any other psychologically produced effects.

### *The Dance of Specificity*

As recorded in the 2nd edition of the Oxford English Dictionary (1989), the term *specific* was first used in a medical context in the 17th century as a noun. Its use did not seem to correspond to our current use of the term. It was not used to differentiate effective from ineffective or real from sham substances. Instead, both could be described as specifics. This usage was common as late as the mid-19th century, when Graves (quoted in the OED) wrote that "all specifics lead to a false system of therapeutics." In other words, *specific* was simply a term used to denote something used as a remedy. Simply put, *a specific* is a drug. In this sense, a placebo is a nonspecific because (like psychotherapy) it is not a drug. This usage of the term *specific* is archaic, and our current use of the term as a treatment with particular or restricted effects may be nothing more than an historical accident.

What does it mean to say that the effects of a treatment are not specific? In this section, I examine a number of possible meanings of the term *specific* and evaluate their relation to the issue of placebo psychotherapy.

One thing it could mean is that the treatment does not just affect a particular condition, but instead affects a wide range of conditions. Placebos do indeed affect a wide range of conditions, as do aspirin, morphine, selective serotonin reuptake inhibitors, and psychotherapy. This, of course, is one of the strengths of these particular treatments, not one of their weaknesses. On the other hand, placebos are not *nonspecific*, in this sense of the term. There are many conditions that they do not effect, and they affect some conditions more than others.

Another potential meaning of the term *specific* is the effects are due to some specific characteristics of the treatment, that is to features of that treatment that are not generally shared with other treatments. But the effects of placebos do depend on their specific characteristics. They depend on their color, size, dose, mode of administration, label, and ability to produce side effects. What differentiates these effects from the so-called specific effects of active medications is that they are psychologically mediated. They are due to the meaning of these characteristics to the person (Moerman & Jonas, 2002), but this, of course, is true of psychotherapy as well.

There are some treatment characteristics that are common to most psychotherapies, and one might be tempted to refer to these as *placebo factors*. Common factors include the establishment of a therapeutic relationship, discussion of the presenting problem, cognitive restructuring, and the provision of warmth, empathy, positive regard, and response-contingent reinforcement, all of which might thus be labeled placebo factors. Conversely, the rationale provided to clients about how the treatment works varies from treatment to treatment. So this would not be a placebo factor. It is worthwhile to examine the degree to which the effects of various psychotherapies are due to common factors, and comparative effect sizes (e.g., Smith, Glass, & Miller, 1980) suggest that this may account for a substantial proportion of treatment effects. But what if all of the effects of psychotherapy were due to the common factors listed above? Should that lead us to conclude that psychotherapy is ineffective and abandon its practice, or should it lead us to find ways of maximizing these factors in treatment?

Finally, specific effects might be characterized as those that are produced by the theoretical mechanism specified in the theory underlying the treatment. Conversely, nonspecific effects would be those due to the mechanisms underlying the effects of medical placebos. This, of course, assumes that we know what factors produce treatment and placebo effects. The problem is that we generally do not. It may be true, for example, that the effects of behavior are due to classical conditioning, whereas placebo effects are produced by expectancy. Conversely, behavior therapy effects may be due to expectancy (e.g., Kirsch, Tennen, Wickless, Saccone, & Cody, 1983), and placebo effects may be due to conditioning (Stewart-Williams & Podd, 2004).

Among the factors that have been cited as mechanisms underlying the placebo effect are response expectancy, classical conditioning, endorphin release, hopefulness, and the therapeutic relationship. Let us assume for a moment, that one or another of these hypotheses is correct and let us apply it to psychotherapy. For instance, let us assume that the effects of a particular psychotherapy were found to be due to classical conditioning or to be mediated by endorphin release. Would that make the therapy any less legitimate? Is conditioning a sham? Are endorphins inert?

Similar questions can be asked about replacing hopelessness with hopefulness as a mechanism of therapeutic change, especially with respect to the treatment of depression. Hopelessness has been proposed as a cause of some forms of depression (Abramson, Metalsky, & Alloy, 1988). If this theory is correct, then the only treatments that can be said to be specific for the treatment of this form of depression are those that work by instilling a sense of hope. In other words, placebos (to the extent that they work by instilling hope) are specific for the treatment of depression, because lack of hope can be a specific cause of depression.

It is widely assumed that placebo effects are due to expectancy. Now I happen to be partial to that hypothesis (see Kirsch, 1985, 1990, 1999). Let us assume that it is correct. If the effects of a psychological treatment are mediated by expectancy, is that treatment a placebo? To answer affirmatively is to dismiss expectancy as somehow less legitimate a psychological factor than classical conditioning, the therapeutic relationship, abreaction, unconditional positive regard, or any of the other factors that have been proposed as explanations of the effects of psychotherapy.

Concluding that the effect of a drug is due to expectancy is equivalent to saying that the drug does not work. Hence, one is tempted to say that if the effect of a psychological treatment is due to expectancy, then the treatment does not work. But whatever the causes of the negative connotations, they are unwarranted. In fact, one can argue that dismissing expectancy is particularly egregious, because it is a particularly important and ubiquitous psychological factor. It is, after all, an essential component of classical and

operant learning; it is the only psychological factor routinely controlled in evaluations of new medical treatments. In fact, many scholars have noted that a primary purpose of the brain is to anticipate the future so that behavior can be adjusted accordingly. In other words, the brain functions as an expectancy machine (Craik, 1943; Dennett, 1991; Hyland, 1985; Jacob, 1982; Kirsch, 1990). What sense does it make to degrade a therapy because its effects are due to this central function of the brain?

## Control Conditions in Psychotherapy Outcome Research

I have argued that the placebo effect cannot, and should not, be controlled in psychotherapy outcome research. What, then, are the control conditions that should be included? In fact, they are not different from the control procedures that have been and are currently being used. My argument is not for a change in research practice, but rather for a change in interpretation.

A number of important questions can be addressed in psychotherapy outcome research. These include establishing the efficacy of a particular therapy, testing hypotheses about the reasons for its effectiveness, comparing it with alternate treatments, and determining whether there are particular groups of individuals for whom the treatment is indicated or contraindicated.

To determine the efficacy of a particular treatment, a no-treatment, wait-list, or natural history control group is needed. Finding a significant difference between treatment and no-treatment groups tells you that the treatment has had an effect and that improvement was not just due to spontaneous remission, the passage of time, or statistical regression. One should also want to establish the clinical importance of a treatment effect; how much difference does this treatment make in the quality of life of the client? Answering this question depends on using appropriate outcome measures, rather than any additional control groups.

Comparisons with so-called placebo control groups in psychotherapy research do not tell you whether a treatment works. Instead, they tell you something about how and why it works. These are also important questions, but they should not be confused with the question of efficacy. In the remainder of this section, I consider the interpretation of comparisons of a treatment with various types of control procedures.

Empirically derived controls (Kazdin & Wilcoxon, 1976) are specific procedures that are as credible and compelling as the investigational treatment, but are in other respects very different from it (see, for example, Kirsch et al., 1983). If the effects of these control procedures are found to be comparable to those of the investigational treatment, the hypothesis that the effects of both may be due to expectancies for improvement is supported. Conversely, if the investigational treatment is found to be significantly more effective than the equally credible control procedure, the logical conclusion is that some of the procedures that are specific to the investigational treatment have an effect that is not completely mediated by expectancy.

Attention control procedures are generally less compelling or credible than the investigational treatment (Borkovec & Nau, 1972). They are often described as placebo controls, but this label is misleading, because placebos in medical research are designed to be as credible as the treatment being investigated. In addition, they typically contain specific procedures that are different from the therapy being investigated and from other therapies in general. For example, Paul's (1966) seminal attention placebo included unique specific features (e.g., listening to audiotapes of white noise while supposedly under the influence of an anti-anxiety medication), whereas his less specific treatment, in which clients discussed their problem with a therapist, was deemed a comparison psychotherapy rather than a placebo.

To interpret the meaning of a difference between an investigational treatment and an attention control, one must first examine the specific procedural commonalities and differences between the two. Finding a difference in outcome may point to specific procedures that can enhance treatment outcome, but more information can be obtained by a more systematic variation of psychotherapy components. For this reason, partial treatment and treatment component controls may be more useful than attention controls.

Partial treatment conditions consist of an investigational treatment minus one or another component. Systematic desensitization, for example, includes relaxation training, the construction of a hierarchy of anxiety related scenes, and pairing of the imagined scenes with relaxation. Leaving out one component (e.g., relaxation) while keeping others constant tells one whether that particular ingredient is "active." However, it does not tell one the reason for the effectiveness of that component. For example, a particular component might enhance outcome because it increases the credibility of the treatment.

Treatment component control groups receive a particular component of the investigational treatment. For example, systematic desensitization might be compared to relaxation training. If it is found to be as effective as the full treatment, it can be hypothesized to be the active ingredient (rather than an active ingredient) of the treatment.

All of these control procedures are related to placebo controls as used in medical research, but none of them controls for the placebo effect. Neither do any of them provide information about whether the treatment is effective. Instead, they provide information on the psychological mechanisms underlying the effectiveness of a treatment. For that reason, a failure to find significant differences should not be interpreted as an indication that the treatment is not effective or that it is "only" a placebo.

## Synonym or Oxymoron?

So are the terms *placebo* and *psychotherapy* synonyms? Or is the phrase *placebo psychotherapy* an oxymoron? In a sense, both of these statements are true.

Placebos in medical research are interventions that have all of the psychological characteristics of the investigational drug, lacking only the chemically active ingredient. Because psychotherapy has no chemically active ingredient, it can be considered a placebo by definition. Thus, in the context of psychotherapy research, the two terms can be considered synonymous. Note that the psychological ingredients of both placebos may be active, in the sense of having an effect on the target condition. In fact, were they not active, there would be no need to use them as controls in medical research.

Conversely, placebo psychotherapy can be considered an oxymoron, because it is impossible to devise a meaningful placebo control for psychotherapy outcome research. Any procedure we design as a control must have psychological characteristics that are different from those of the investigational treatment; otherwise, it would be that treatment. More important, if the control procedure is effective in treating the disorder, then it is a bona fide treatment. It must have psychologically active ingredients or else it would not work. And it does not matter what those active ingredients are. They may be hope or faith or response expectancy. In principle, these are no different from any other psychological factor than can alleviate distress.

## References

Abramson, L.Y., Metalsky, G.I., & Alloy, L.B. (1988). The hopelessness theory of depression: Does the research test the theory? In L.Y. Abramson (Ed.), Social cognition and clinical psychology: A synthesis (pp. 33–65). New York: Guilford.

Benedetti, F., Maggi, G., Lopiano, L., Lanotte, M., Rainero, I., Vighetti, S., et al. (2003). Open

versus hidden medical treatments: The patient's knowledge about a therapy affects the therapy outcome. Prevention & Treatment, 6, Article 1. Retrieved June 23, 2003, from http://journals.apa.org/prevention/volume6/pre0060001a.html

Blackwell, B., Bloomfield, S.S., & Buncher, C.R. (1972). Demonstration to medical students of placebo responses and non-drug factors. The Lancet, 19, 1279–1282.

Borkovec, T.D., & Nau, S.D. (1972). Credibility of analogue therapy rationales. Journal of Behavior Therapy and Experimental Psychiatry, 3, 257–260.

Branthwaite, A., & Cooper, P. (1981). Analgesic effects of branding in treatment of headaches. British Medical Journal, 282, 1576–1578.

Cobb, L., Thomas, G.I., Dillard, D.H., Merendino, K.A., & Bruce, R.A. (1959). An evaluation of internal-mammary artery ligation by a double blind technique. New England Journal of Medicine, 260, 1115–1118.

Craik, K.J.W. (1943). The nature of explanation. Cambridge, UK: Cambridge University Press.

de Craen, A.J.M., Moerman, D.E., Heisterkamp, S.H., Tytgat, G.N.J., Tijssen, J.P.G., & Kleijnen, J. (1999). Placebo effect in the treatment of duodenal ulcer. British Journal of Clinical Pharmacology, 48, 853–860.

de Craen, A.J.M., Roos, P.J., de Vries, A.L., & Kleijnen, J. (1996). Effect of colour of drugs: Systematic review of perceived effect of drugs and their effectiveness. British Medical Journal, 313, 1624–1626.

de Craen, A.J.M., Tijssen, J.G.P., de Gans, J., & Kleijnen, J. (2000). Placebo effect in the acute treatment of migraine: Subcutaneous placebos are better than oral placebos. Journal of Neurology, 247, 183–188.

Dennett, D.C. (1991). Consciousness explained. Boston: Little, Brown, and Company.

Dimond, E.G., Kittle, C.F., & Crockett, J.E. (1960). Comparison of internal mammary ligation and sham operation for angina pectoris. American Journal of Cardiology, 5, 483–486.

Evans, F.J. (1974). The placebo response in pain reduction. Advances in Neurology, 4, 289–296.

Herrnstein, R. (1962). Placebo effect in the rat. Science, 138, 677–678.

Hróbjartsson, A., & Gøtzsche, P.C. (2001). An analysis of clinical trials comparing placebo with no treatment. New England Journal of Medicine, 344, 1594–1602.

Huskisson, E. (1974). Simple analgesics for arthritis. British Medical Journal, 4, 196–200.

Hyland, M.E. (1985). Do person variables exist in different ways? American Psychologist, 40, 1003–1010.

Jacob, F. (1982). The possible and the actual. Seattle, WA: University of Washington Press.

Kantor, T.G., Sunshine, A., Laska, E., Meisner, M., & Hopper, M. (1966). Oral analgesic studies: Pentazocien, hydorchloride, codeine, aspirin, and placebo and their influence on response to placebo. Clinical Pharmacology and Therapeutics, 7, 447–454.

Kaptchuk, T.J. (1998). Intentional ignorance: A history of blind assessment and placebo controls in medicine. Bulletin of the History of Medicine, 72, 389–433.

Kazdin, A.E., & Wilcoxon, L.A. (1976). Systematic desensitization and non-specific treatment effects: A methodological evaluation. Psychological Bulletin, 83, 729–758.

Kirsch, I. (1978). The placebo effect and the cognitive-behavioral revolution. Cognitive Therapy and Research, 2, 255–264.

Kirsch, I. (1985). Response expectancy as a determinant of experience and behavior. American Psychologist, 40, 1189–1202.

Kirsch, I. (1990). Changing expectations: A key to effective psychotherapy. Pacific Grove, CA: Brooks/Cole.

Kirsch, I. (1997). Specifying nonspecifics: Psychological mechanisms of placebo effects. In A. Harrington (Ed.), The placebo effect: An interdisciplinary exploration (pp. 166–186). Cambridge, MA: Harvard University Press.

Kirsch, I. (1999). How expectancies shape experience. Washington, DC: American Psychological Association.

Kirsch, I., Lynn, S.J., Vigorito, M., & Miller, R.R. (2004). The role of cognition in classical and operant conditioning. Journal of Clinical Psychology, 60(4), 369–392.

Kirsch, I., Moore, T.J., Scoboria, A., & Nicholls, S.S. (2002). The emperor's new drugs: An analysis of antidepressant medication data submitted to the FDA. Prevention and Treatment, 5, Article 23. Retrieved July 15, 2002, from http://www.journals.apa.org/prevention/volume5/pre0050023a.html

Kirsch, I., & Rosadino, M.J. (1993). Do double-blind studies with informed consent yield externally valid results? An empirical test. Psychopharmacology, 110, 437–442.

Kirsch, I., & Sapirstein, G. (1998). Listening to Prozac but hearing placebo: A meta-analysis of antidepressant medication. Prevention & Treatment, 1, Article 0002a. Retrieved June 26, 1998, from http://www.journals.apa.org/prevention/volume1/pre0010002a.html

Kirsch, I., Tennen, H., Wickless, C., Saccone, A.J., & Cody, S. (1983). The role of expectancy in fear reduction. Behavior Therapy, 14, 520–533.

Kirsch, I., & Weixel, L.J. (1988). Double-blind versus deceptive administration of a placebo. Behavioral Neuroscience, 102, 319–323.

Klein, D.F. (1998). Listening to meta-analysis but hearing bias. Prevention and Treatment, 1, Article 0006c. Retrieved June 26, 1998, from http://www.journals.apa.org/prevention/volume1/pre0010006c.html

Luchelli, P.E., Cattaneo, A.D., & Zattoni, J. (1978). European Journal of Clinical Pharmacology, 13, 153–155.

Luparello, T.J., Lyons, H.A., Bleeker, E.R., & McFadden, E.R. (1968). Influence of suggestion on airways reactivity in asthmatic subjects. Psychosomatic Medicine, 30, 819.

McFadden, E.R., Luparello, T., Lons, H.A., & Bleecker, E. (1969). The mechanism of suggestion in the induction of acute asthma attacks. Psychosomatic Medicine, 31, 134–143.

Melander, H., Ahlqvist-Rastad, J., Meijer, G., & Beermann, B. (2003). Evidence b(i)ased medicine—Selective reporting from studies sponsored by pharmaceutical industry: Review of studies in new drug applications. British Medical Journal, 326, 1171–1173.

Moerman, D.E., & Jonas, W.B. (2002). Deconstructing the placebo effect and finding the meaning response. Annals of Internal Medicine, 136, 471–476.

Montgomery, G.H., & Kirsch, I. (1997). Classical conditioning and the placebo effect. Pain, 72, 107–113.

Moseley, J.B., O'Malley, K., Petersen, N.J., Menke, T.J., Brody, B.A., Kuykendall, D.H., et al. (2002). A controlled trial of arthroscopic surgery for osteoarthritis of the knee. New England Journal of Medicine, 347, 81–88.

Nagao, Y., Komia, J., Kuroanagi, K., Minaba, Y., & Susa, A. (1968). Effect of the color of analgesics on their therapeutic results. Shikwa Gakuho, 68, 139–142.

Neild, J.E., & Cameron, I.R. (1985). Bronchoconstriction in response to suggestion: Its prevention by an inhaled anticholinergic agent. British Medical Journal, 290, 674.

Paul, G.L. (1966). Insight vs. desensitization in psychotherapy. Stanford, CA: Stanford University Press.

Pihl, R.O., & Altman, J. (1971). An experimental analysis of the placebo effect. Journal of Clinical Pharmacology, 11, 91–95.

Prioleau, L., Murdock, M., & Brody, N. (1983). An analysis of psychotherapy versus placebo studies. Behavioral and brain sciences, 6, 275–310.

Rescorla, R.A. (1988). Pavlovian conditioning: It's not what you think it is. American Psychologist, 43, 151–160.

Shapiro, A.K., & Shapiro, E. (1997). The placebo: Is it much ado about nothing? In A. Harrington (Ed.), The placebo effect: An interdisciplinary exploration (pp. 12–36). Cambridge, MA: Harvard University Press.

Siegel, S. (1983). Classical conditioning, drug tolerance, and drug dependence. In Y. Israel, F.B.

Glaser, H. Kalant, R.E. Popham, W. Schmidt, & R.G. Smart (Eds.), Research advances in alcohol and drug problems (Vol. 7, pp. 207–246). New York: Plenum Press.

Sloane, R.B., Staples, F.R., Cristol, A.H., Yorkston, N.J., & Whipple, K. (1975). Psychotherapy versus behavior therapy. Cambridge, MA: Harvard University Press.

Smith, M.L., Glass, G.V., & Miller, T.I. (1980). The benefits of psychotherapy. Baltimore: John Hopkins University Press.

Spector, S.L., Luparello, T.J., Kopetzky, M., Souhrada, J., & Kinsman, R.A. (1976). Response of asthmatics to methacholine and suggestion. American Review of Respiratory Disease, 113, 43–50.

Stewart-Williams, S., & Podd, J. (2004). The placebo effect: Dissolving the expectancy versus conditioning debate. Psychological Bulletin, 130(2), 324–340.

Voudouris, N.J., Peck, C.L., & Coleman, G. (1985). Conditioned placebo responses. Journal of Personality and Social Psychology, 48, 47–53.

Voudouris, N.J., Peck, C.L., & Coleman, G. (1989). Conditioned response models of placebo phenomena: Further support. Pain, 38, 109–116.

Voudouris, N.J., Peck, C.L., & Coleman, G. (1990). The role of conditioning and verbal expectancy in the placebo response. Pain, 43, 121–128.