

IS BEHAVIORISM BECOMING A PSEUDOSCIENCE? REPLIES TO DRs. WYATT, MIDKIFF AND WONG

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ABSTRACT: Wyatt and Midkiff (2006a) and Wong (2006a) argued that the eclipse of token economy treatment for schizophrenia was due not to scientific judgments but to the biological politics of the mental health field. I argued that the treatment's fate was due to its own limitations, particularly the failure of effects to generalize adequately to natural environments given deinstitutionalization (Wakefield, 2006). Wyatt and Midkiff (2006b) and Wong (2006b) vigorously disputed my claim. In this reply, I analyze their responses regarding generalization, and their arguments for behavioral etiology. I conclude that we all agree that such treatments were not shown to adequately generalize, providing a scientific reason for the treatment's fate. I also find their etiological arguments unsound. Even-handed attention to evidence, recognition of behaviorism's limits and strengths, and an integrative approach are essential if behaviorism is not to veer toward pseudoscience.

KEYWORDS: behaviorism, behavioral treatment, schizophrenia, token economy, etiology of schizophrenia, mental disorder, history of psychology, history of psychiatry, philosophy of science, harmful dysfunction, biological causation, pharmaceutical industry, psychotropic medications

In an earlier issue of this journal, I published a commentary (Wakefield, 2006) on Dr. Wong's (2006a) and Drs. Wyatt and Midkiff's (2006a) target articles in which they critiqued the turn toward biological approaches and away from behavioral approaches to severe mental disorder. My commentary elicited vigorous, quite harsh responses (Wong, 2006b; Wyatt & Midkiff, 2006b). The Editor having generously allowed me to reply, I hope to advance the discussion by clarifying the arguments behind the heated rhetoric.

First, some general comments might help orient the discussion. In their target articles, both Wyatt and Midkiff and Wong attempt to resuscitate behavioral intervention for severe mental disorders by attacking biological psychiatry and blaming behaviorism's waning fortunes on the politics of the mental health field rather than on any scientific findings about behavioral intervention: "The decline of behavioral approaches in the treatment of psychoses is a minor mystery in the history of science, and I have suggested that an understanding of this seeming dead end requires that one look beyond the activities of behavioral researchers to larger ideological, political, and economic movements..." (Wong, 2006a, p. 169). And the conclusion, as expressed by Wyatt and

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Midkiff (2006a), was that “it is time for a paradigm shift, away from extreme biological causation and toward an environmental causation model...” (p. 147). In other words, the fault lies not in behaviorism itself, but in its political stars.

I pointed out that the authors’ claim that the waning of behavioral intervention was due to politics, not scientific considerations, needed to be assessed as a causal hypothesis, and this entailed assessing the scientific pros and cons of behavioral intervention: “Such a claim must be supported not merely by showing that biological psychiatry has weaknesses—so does every theory!—but by showing also that behavioral approaches do not have equal or greater weaknesses” (Wakefield, 2006, p. 203). I also noted that political processes always accompany scientific change; the question is whether there was an underlying scientific logic that allowed those forces to hold sway, and whether political processes *replaced* or merely *accompanied* a reasonably legitimate scientific judgment. For example, the switch at many psychiatric institutions from predominantly psychoanalytic to predominantly biological research often involved intense politics, but was grounded in legitimate scientific considerations.

The potential pseudoscientific status of the authors’ “politics” claim should be clear. When Freudians blamed rejection of the oedipal theory on the defensiveness of opposing theoreticians, that was dismissed as pseudoscientific avoidance of evidence. Blaming behaviorism’s rejection on politics could be seen similarly, unless accompanied by a careful look at whether evidential weaknesses in behaviorism might be responsible.

Moreover, the authors’ claim that biologically dominated politics is responsible for behavioral treatment’s sidelining is *prima facie* implausible. Where evidence is strong for behavioral therapeutic effectiveness, it has become a standard part of the therapeutic armamentarium, as is the case in phobias where desensitization, avoidance, exposure, and other behavioral concepts remain basic to clinical thinking. Harshburger (2006) also notes the recent rapid increase in behavior-based treatment for children with diagnoses of autism and other developmental disabilities due to recent studies suggesting effectiveness. The explanations for such successes seems to lie in the persuasiveness of the scientific evidence. The target articles consider no such behavioral success stories in assessing the “politics” claim; the word “phobia” does not appear in either article. Moreover, other non-biological approaches to various disorders, such as dialectical behaviorism for borderline personality disorder and cognitive and interpersonal therapy for depression, have thrived when they appeared to have demonstrated effectiveness. The evidence-unresponsive political biologicalism postulated by the target articles to dominate the field would surely have expunged those non-biological approaches, as well.

In addition, while theory and treatment are not necessarily tied in a one-to-one correspondence, surely the fate of evidentially weaker behavioral interventions was to some extent tied to the scientific evidence casting doubt on the broader behavioral account of human behavior. After being the dominant paradigm in American psychology for some decades, behaviorism was overtaken by a variety of research results that yielded anomalies revealing its limitations as an overall account of psychological functioning. These evidential failures led to the “cognitive revolution” and to a resurgence of biological approaches. These anomalies included evidence of complex planful cognitive

behavior that is teleologically ordered even in rats let alone humans, the reality and importance of unconscious semantic processing and conscious imagery, and startlingly strong evidence of biological preparedness to learn and to maintain certain kinds of learned behavior in ways not in accord with classic learning theory. The net result was that, rather than being a dominant default view, behavioral accounts had to be demonstrated to be useful in each domain to which they were applied. In contrast to the target authors' staunch defense of core beliefs, many behavioral theorists accepted the evidence and looked to cognitive-behavioral theory and other forms of integration. Salzinger's (2006) commentary, for example, offers an appealing integrative approach that recognizes the research evidence, eschews the "them vs. us" mentality of the target articles, and embraces biological etiologies as forming the necessary context in which behavioral intervention must prove itself useful.

So, I undertook in my commentary to do what Wyatt and Midkiff and Wong failed to do, namely, to examine whether token economies might have been sidelined based on their own scientific merits rather than politics. I used my commentary to focus both more narrowly and more broadly than the target articles. My focus was narrower in exclusively addressing token economy treatment of schizophrenia. In my view, each disorder must be considered individually regarding the issues in dispute. For example, behavioral methods are integral to treatment of phobias, Rhett's disorder is now known to be of biological origin, and depression is yet a different case due to confusion of normal sadness and depressive disorder (Horwitz & Wakefield, 2007). Schizophrenia seemed most relevant to the articles' claims, and was the subject of many comments with which I disagreed. To address the issues raised by the articles within the compass of a commentary—even a lengthy one—seemed attainable if the questions were framed specifically in terms of the rejection of token economies for schizophrenia.

My commentary's focus was also broader than the target articles' because I looked beyond their unsupported claims and considered whether the defensive maneuver of blaming politics for behaviorism's woes pseudoscientifically ignored evidence to preserve behaviorism's ideological core. Regarding the broadness, Wong complains that my commentary "focused on papers *other than the ones* published in this issue of BSI" (p. 239; emphasis in original), as if looking to a broader literature to assess the soundness of an argument is a violation of scholarly rules of engagement. I was trying to be fair, so I took it upon myself, since the target authors did not do so, to examine the literature on token economy treatment of schizophrenia. What emerged was repeated mentions of trouble with generalization. I wanted to consider the strongest argument for their position rather than a straw man, and Paul and Lentz's (1977) study is sometimes cited as the best the token-economy field has to offer, so I paid particular attention to that study.

Finally, it is important to keep in mind what I actually said versus how the authors portray what I said. I amply recognized the virtues of behavioral interventions as part of an integrative approach and within the constraints of what research has demonstrated. My conclusion began as follows: "Behaviorist research on treatment of schizophrenia can boast legitimate achievements that are worth defending. Clearly, irrespective of the generalization issue, when ward behavior itself is what is desired to change, token control

is very useful indeed” (p. 219). Earlier in my article, I note that Paul and Lentz’s (1977) study “did indeed support the substantial effectiveness of a token economy as part of a psychosocial training intervention in shaping the behavior of hospitalized psychotic patients” (p. 213). As to the biological literature, I agreed there are weaknesses and especially overselling of drug treatments even as I tried to correct what I saw as the authors’ overstatements. I said that the authors’ critiques are “partly true” although there are “offsetting strengths and achievements” (p. 202), and I characterized the biological psychiatric record as “suffering from many deficiencies,” though “not as weak as they claim” (p. 203). None of this answers the question whether research on token economies indicated potential weaknesses that warranted focusing attention on alternatives.

WYATT AND MIDKIFF’S CRITIQUE OF BIOLOGICAL RESEARCH AND TREATMENT

Keeping in mind what I actually said is especially important when reading Wyatt and Midkiff’s (2006b) response. Contrary to their absurd straw-man characterization of me as avidly pro-drug, the position I actually took in my commentary recognized the complex realities and severe limitations of both drug therapy and behavioral intervention for schizophrenia. This nuanced approach earned me the insult of being “slippery” like a “greased pig” (p. 224), reminiscent of the way conservative ideologues label any opponent who recognizes some merit on both sides of an issue as a “waffler.”

To mention one salient example of Wyatt and Midkiff’s brazen distortion of my position, they devote a substantial part of their response to contrasting a list of negative judgments by experts about the serotonin imbalance theory of depression with a list of examples of the drug companies’ shameless use of the imbalance theory in ads to appeal to consumers. They say the expert opinions are “in contrast to Wakefield’s assertions” (p. 225) without citation. Now, here is what I said in my commentary: “Turning to the “unbalanced neurotransmitter” theory of mental disorder, I agree with Wyatt and Midkiff (and I think there is broad agreement among biological psychiatrists) that this “humoral” approach has been oversold, and perhaps for the worst of reasons” (pp. 204-205). Wyatt and Midkiff’s lists actually *support* the position I explicitly stated in my commentary.

In their response, Wyatt and Midkiff focus on defending what they consider their central theses, which they identify as follows: (1) “The research said to support biological causation of mental disorders is relatively weak”; (2) “The claims of drug effectiveness are often overstated” (p. 222; see also pp. 224-225). Wyatt and Midkiff incorrectly say I called these two anti-biological contentions a “myth” and took the “polar opposite” position. In fact, as I have explained, these anti-biological theses were not my primary concern. What I labeled a “myth” was Wyatt and Midkiff’s contention that *biological science eclipsed behavioral treatment of schizophrenia for sheerly political, unscientific reasons*. This pivotal claim leads them to urge a return to the behavioral paradigm.

While I will not revisit Wyatt and Midkiff’s critique of biological research in any detail here or offer citations to the literature—it is a vast topic that would require an extensive literature review that they did not attempt—I offer some brief general comments on their critique before focusing on the behaviorist account. All research has

limitations, so conclusions are most compelling when they derive from diverse sources of data that converge in the same direction, each with its own set of offsetting limitations. This is the case with the research supporting biological causation of schizophrenia, which ranges from standard twin and brain imaging studies to a vast array of ancillary findings of biological correlates from paternal age and head trauma to season of birth. Wyatt and Midkiff ignore most of this research, and argue against the rest by creating ad hoc alternative hypotheses in each case to show that the results could be explained by behavioral principles, but without offering any independent evidence that their speculations are the correct explanations or addressing the convergence of so many areas of research. Even if the twin studies, their primary focus, were without merit, that would cast doubt on genetic causation but not biological causation that is environmental, as in traumatic, toxic-substance, and viral-infection theories of schizophrenia.

In any event, Wyatt and Midkiff's two central points about twin studies, that environments assigned to separated twins may be biased toward similarity and that the greater physical similarity of identical twins may lead to similar environmental responses to them and thus to similar pathologies due not to genetics but to similar environmental reinforcement, have been around for a long time and have been extensively empirically studied (e.g., see the literature on the "equal environments assumption"), from classic articles by Scarr and Plomin et al. to Bouchard's analyses of similarity of environments (see also the recent exchange between Sulloway and Kramer in *New York Review of Books*), none of which is considered in Wyatt and Midkiff's comfortably myopic discussion. The results have supported the biological conclusions and cast doubt on the alternative hypotheses; the proposed causes, independently measured, do have some small impact, but not nearly enough to cast doubt on the primary conclusions regarding genetic factors. Nor do Wyatt and Midkiff consider known factors that may lead to underestimation of heritability in twin studies that compare dizygotic and monozygotic rates of concordance, such as assortative mating for pathology. As to studies of brain pathology, biological researchers readily acknowledge that brain differences may be due to the disorder or to its treatment rather than a biological cause of the disorder, and seek to distinguish these hypotheses. Biological research has the virtue of being a progressive paradigm that offers novel hypotheses, acknowledges evidential weaknesses and addresses them with further studies, and gives up hypotheses when disconfirmed. The behavioral theory of schizophrenia offers no comparably progressive paradigm.

Wyatt and Midkiff state: "Wakefield failed to mention the American Psychiatric Association's collapse when it was challenged (Mind Freedom, 2003) to produce any scientifically valid evidence to support biological causation" (p. 203). How the APA responds to a public relations stunt—and their response left much to be desired—is not a legitimate way to evaluate the scientific research evidence. In any event, although the circumstantial evidence for biological causation is strong, no one knows the causes of schizophrenia. Even the NIMH schizophrenia public information website states: "Because the causes of schizophrenia are still unknown, current treatments focus on eliminating the symptoms of the disease." As we shall see, Wyatt and Midkiff's response

IS BEHAVIORISM BECOMING A PSEUDOSCIENCE?

to my challenge to them reveals that they have not a shred of scientific evidence for their behaviorist view of etiology, so whatever the APA said offers no comfort to behaviorism.

Wyatt and Midkiff admit that “there is truth” in my point that the emptying of the asylums was due to chlorpromazine, not token economies, but note other reasons for the rapid emptying of asylums (e.g., mandated deinstitutionalization, social security disability payments used for outpatient support, etc.). However, the other factors had their major impact because of the ability of medication to allow community placement for some previously unreleasable patients. Wyatt and Midkiff also note that release does not imply cure, and point to the enormous problem of the homeless mentally ill. That deinstitutionalization was botched, and that many deinstitutionalized patients were anything but cured and did not receive the promised community mental health support services (with collusion by professionals), is a painful daily reality on our cities’ streets. However, there is no comfort for behaviorism in this, and Wyatt and Midkiff’s own view (see below) implies that behavioral treatment should not be expected to do any better with released patients.

I now turn to Wyatt and Midkiff’s and Wong’s responses to, first, my critique of token-economy treatment, and second, behavioral etiological theory of schizophrenia.

WYATT AND MIDKIFF ON THE FAILURE OF GENERALIZATION OF TOKEN-ECONOMY EFFECTS

My central claim was that token economies were eclipsed because research failed to demonstrate adequate generalization to natural environments to make them a viable treatment given deinstitutionalization. Indeed, behavioral researchers—not armchair critics or outsiders like me, but those steeped in and committed to the behavioral approach to severe mental illness—have been saying for decades that generalization is the key test of success and that the results have been disappointing. For example:

The generalization of treatment effects to stimulus conditions in which token reinforcement is not given might be expected to be the *raison d’être* of token economies. An examination of the literature leads to a different conclusion. There are numerous reports of token programs showing behavior change only while contingent token reinforcement is being delivered. Generally, removal of token reinforcement results in decrements in desirable responses and a return to baseline or near-baseline levels of performance. Such a state of affairs led Zimmerman, Zimmerman, and Russell (unpublished) to conclude that token economies are prosthetic rather than therapeutic. (Kazdin & Bootzin, 1972, p. 359)

The implicit assumption underlying any treatment is that the program will help the patient to function in the natural environment. The extent to which the effects of token reinforcement procedures transfer to settings outside the hospital has not been sufficiently addressed. (Stahl and Leitenberg, 1976, p. 235)

Behavioral programming has been found to be successful with a large number of problems; however, treatment effects often do not generalize to nontreatment settings or maintain across time. (Fuoco and Tyson, 1986, p. 253)

However, as is also common with other studies, patients from the token economy who moved on to the predischARGE unit showed significant deterioration in social withdrawal. (Baker, 1988, pp. 636-637)

Although skills training is a validated psychosocial treatment for schizophrenia, generalization of the skills to everyday life has not been optimal. (Glynn et al., 2002, p. 829)

For over 3 decades, studies measured generalization of acquired skills, although no intervention was used to facilitate it. Consequently, the results have been mostly discouraging for transferring skills to participants' environments. (Kopelowicz, Liberman, and Zarate, 2006, p. S15)

Now, here is how Wyatt and Midkiff (2006b) address my "generalization" claim:

We agree that getting improvement within the hospital to generalize to the community remains a work in progress for behavioral scientists. But that fact does not undo the validity of behavioral treatment. The lack of generalization is caused by discontinuation of the behavioral techniques, once the patient is discharged to the community. That fact demonstrates that treatment is effective, not the reverse as Wakefield implies. (p. 227)

So, first, they *agree* with me that the research never established generalizability of token-economy interventions for schizophrenia. That settles the issue by establishing a scientific, non-political reason for the eclipse of such treatments despite the uncertainties and weaknesses of drug therapy. Second, in a classic "glass half full" move, Wyatt and Midkiff assert that this failure of generalizability to the community in fact shows that such treatment is successful! This confuses success of a theoretical prediction that reinforcers will influence symptoms with success as a therapy in real life conditions, which in this case includes return to natural environments as a major concern.

In an effort to downplay of the importance of generalizability, Wyatt and Midkiff devote a paragraph to reminding us that many of the best things in life are transient:

Would Wakefield argue that exercise does not strengthen the body, given that its benefits are lost when one stops exercising? Would Professor Wakefield advise us to forego the annual flu shot simply because its benefits do not last forever? The point here is that the transitory nature of improvement is not evidence that the treatment has failed, as Professor Wakefield evidently believes. (p. 227)

The effects of exercise and flu shots wear off after awhile, yet both can be heartily recommended because the current benefit is the main point, and for future benefit both can be continued indefinitely without much trouble. One would not exercise now and

then stop if the goal is to be fit for a race three years from now, and one would not get a flu shot now in order to avoid the flu three years from now in an environment in which flu shots would not be available. The reason generalization is so important for token economies is that, although the current benefit in reducing schizophrenic symptoms in a controlled environment is important, a crucial additional goal is change that is maintained after discharge into the community environment, but there is no easy way to continue systematic reinforcement schedules for schizophrenic symptoms in natural environments. If token economy effects do not generalize, then they are simply not very useful in an age of deinstitutionalization except as social control devices for institutions. Indeed, Wyatt and Midkiff's thesis that the natural environment reinforces schizophrenic symptoms (see below) violates Ayllon and Azrin's (1968) Relevance of Behavior Rule that one should teach only behaviors that will continue to be reinforced after training and thus will fall under the control of naturally occurring reinforcers in the person's natural environment.

Citing some recent disappointing drug studies, Wyatt and Midkiff attempt to defend behavioral intervention against the "generalization" objection by pointing to problems with the generalization of drugs: "Nor did Professor Wakefield account for another troubling fact—the absence of generalization of drug treatment." The results they focus on in fact concern high dropout rates rather than lack of generalization per se. The meaning of this study's disturbing result is still being pondered. But the real point is that Wyatt and Midkiff get it exactly right: Such results, if upheld, cast doubt on the usefulness of these particular drugs, thus, by parity of reasoning, lack of demonstrable generalization is good scientific reason to question the usefulness of behavioral treatment.

WONG ON GENERALIZATION OF TOKEN ECONOMY EFFECTS

Wong (2006b) addresses at length my central point that "behavioral interventions for psychotic disorders...have failed to demonstrate generalized effects beyond the treatment setting, usually the hospital ward" (p. 240). Dr. Wong claims that "this criticism contains several fallacies," and he presents four counterarguments.

The first counterargument is that behaviorists were the first to develop the notion of generalization: "First, it overlooks that operant research provided the first systematic analyses of 'generalization' as a behavioral phenomena, identifying its properties and variables affecting it" (p. 240). This is a non sequitur. All the references Dr. Wong cites in support of this point are to articles about the concept and process of generalization itself, not to research showing generalization of token-economy treatment effects to natural environments, thus irrelevant to the present dispute. I did not argue that behaviorists did not originate the concept of generalization; I argued that they failed to show that token economies adequately generalize. "Generalization" here is just behavior-speak for a commonsense goal that, no matter how effective in a controlled environment, treatment effects must endure when the patient leaves the controlled environment.

Wong's second counterargument is that I am ignorant of the literature that demonstrates generalization:

Second, Dr. Wakefield portrays generalization as an obstacle or barrier which behavioral interventions have not been able to penetrate. This fallacy may reflect Dr. Wakefield's own narrow reading of the behavioral literature. The importance of generalized behavior change was recognized early during the formulation of the field of applied behavior analysis (Baer, Wolf, & Risley, 1968) and this awareness guided the development of a technology for promoting generalization (e.g., Dunlap, 1993; Horner, Dunlap, & Koegel, 1988; Stokes & Baer, 1977; Stokes & Osnes, 1989). There is nothing magical about programming generalization of positive behavior change for clients in general or for psychiatric patients in particular. (p. 240)

As Wong's citations illustrate, the behavioral literature contains many fine discussions of generalization and many lists of techniques for encouraging generalization. Generalization has sometimes been successfully demonstrated in other domains. But, these points are all non sequiturs. I never disputed any of them. None of the references Wong cites demonstrate that effects of token economies for schizophrenia generalize to natural environments.

As to Dr. Wong's assertion that there is no magic to generalization, it is by no means the cakewalk he suggests. Even in domains much simpler than schizophrenia with much more opportunity for environmental control, such as (to take an example at random) teaching children to eat healthful snacks at home by generalization from training at school, substantial effort and continued intervention in the natural environment are often necessary to achieve limited generalization success ("Results indicated that children's healthy snack choices increased in the preschool training setting, generalization to home was achieved only when procedures to program it were implemented, and that the best results were found when the generalization procedures were tailored to the individual child" [Stark, Collins, Osnes, & Stokes, 1986, p. 367]). Research may yet reveal that schizophrenic symptoms can be enduringly altered across environments with the right mix of techniques. But simply asserting on one's own authority (and contrary to many other authorities) that there is no magic to producing generalization is not science.

Wong chides me as follows: "If Dr. Wakefield had bothered to dig a bit deeper he would have seen that even I have done a couple of studies promoting generalization of desired skills across settings and social situations" (p. 240). Always ready to dig deeper, even at the risk of digging my own scholarly grave, I accepted Wong's challenge to examine his two articles, hoping thereby to reassure myself that indeed "there is nothing magical" about generalization.

Wong's research is impressive and a real contribution, but I do not believe it proves his point. Of the two mentioned articles, one uses a game to teach verbal skills to mentally retarded adolescent male patients with behavioral problems, thus is not strictly relevant to this discussion. One would think that verbal skills would be quite useful and once learned would be reinforced across environments, and thus generalization should be expected. Wong et al. (1996) in fact found the following: "Game training produced large and consistent gains in all target responses in a unit activity room; however, no generalization was recorded in covert assessments taken by staff at other times of the day or in other living areas of the unit" (p. 1). So, Wong extended the reinforcement program

from the activity room where training took place to the rest of the unit where generalization had failed to occur: "To improve performance in daily interactions, an in-situ training procedure consisting of intermittent verbal prompts and reinforcement was programmed into Ss' daily encounters with the staff. This procedure significantly increased use of skills in all Ss, with most of these gains maintaining in 2 Ss who were available for 3-mo follow-up" (p. 1). The maintenance at follow-up is encouraging. However, the target environment is still professionally controlled. In community settings, one does not generally have similar power to systematically manipulate reinforcers.

The other study cited by Wong explicitly concerns generalization of social skills taught to schizophrenic inpatients using token rewards and so is directly relevant to the dispute here. The results parallel those in the study just described. Interestingly, Wong there recognizes that generalization to natural settings is a crucial, understudied, and undemonstrated criterion for success with this population:

Programs that do not train directly in the client's living environment must induce carryover of trained skills to natural settings in order to produce clinically significant gains. While the importance of evaluating social skills generalization in natural settings is widely recognized, there is scant research on this critical outcome....Other experiments that assessed generalization to more naturalistic situations used measures in which it was apparent that an assessment was being performed or had other limitations that obscured their findings....Thus, previous studies teaching conversational skills to schizophrenic clients have not thoroughly assessed stimulus generalization or have used measures that may have been reactive. Most research on social skills training with schizophrenics suffers from these same shortcomings. (Wong et al., 1993, p. 287)

Wong et al. (1993) describe the results of the study's initial stage as follows:

The present study taught conversational skills to schizophrenic clients with conventional SST procedures in a therapy room and conducted unobtrusive assessments of generalization in the hospital unit dayhall and courtyard with nursing staff and total strangers. These assessments revealed variable and often minimal spontaneous generalization across the various stimulus conditions. (p. 288)

Having failed to achieve substantial spontaneous generalization from the training room to the unit, Wong et al. (1993) pursued generalization as follows: "In an attempt to produce desired behavior change in a clinically relevant setting, a series of increasingly intensive procedures, including homework assignments and in vivo training, was applied in the unit dayhall..." (p. 288). Two such interventions failed to produce the desired effect. Only the third and most intensive level of intervention, including intermittent reinforcement of multiple ward interactions in vivo, produced desired changes on the ward. This level of intervention depends heavily on the control possible in the unit environment. As in this study, continued success may depend on the intensity of continued intervention. For example, Lauriello et al. (1999) recount how, in a study of social skills training using weekly training sessions that yielded benefits in relapse rates,

“The researchers decided to provide a biweekly treatment in the final 3 months to prepare for termination. The superiority of the SST plus drug lasted only as long as the weekly intervention was in place and could not be sustained in a biweekly fashion” (p. 1412).

Wong’s careful studies reveal the effort needed to extend quite adaptive behaviors even to controlled environments immediately outside the training room. This raises the question of the source of the symptoms. It is sometimes argued that unusual histories of reinforcement (rather than biological processes) led to symptoms and that symptoms are highly stigmatized, so once symptoms are extinguished in a controlled environment, the patient will likely remain symptom free in the natural environment. As Kazdin and Bootzin (1972) observed, this idea traditionally shaped the selection of behavioral targets:

Perhaps the most frequently used procedure [to promote generalization] is to follow Ayllon and Azrin's (1968, pp. 49-56) Relevance of Behavior Rule which states: "Teach only those behaviors that will continue to be reinforced after training." Thus, behaviors should be selected that can come under the control of naturally occurring reinforcers in the person's environment. Target behaviors that have typically been selected in token economies (self-care behaviors, work skills, academic behaviors) do meet this criterion. These are behaviors that will continue to be reinforced (e.g., by social approval) after training. (p. 361)

Psychotic symptoms do not generally remain extinguished if the patient returns to a natural environment. There seem to be two possible explanations. Either natural environments in fact do reinforce such symptoms, contrary to common sense and sociological studies of stigma (this is Wyatt and Midkiff’s explanation); or some internal biological process is continuing to generate such symptoms, and this process returns to dominance once reinforcement ceases. Yet, natural environments do not seem to reinforce delusional, withdrawn, hallucinatory, and other schizophrenic behavior, so if someone requires continual overt reinforcers in addition to the normal incentives of the social system that are set up to support normal behavior, just as if they require continued medication, that suggests that some underlying disease is likely. In any event, there is nothing in Wong’s fascinating studies that addresses let alone resolves the question of generalization to natural environments.

Wong’s third argument is the following:

Third, Dr. Wakefield’s criticism of behavioral studies for their lack of generalization is extremely one-sided. If behavioral treatments are being compared with psychotropic drugs, why is the issue of generalization being brought up at all? Psychotropic drugs are not assumed to produce generalized effects (effects that carry over to times and places in which the drugs are not in the patient’s body), such effects are virtually never assessed, and their absence is not reported. Generalization is a form of extended behavior change that behavioral researchers assess because they seek to give their clients functional capabilities that will make them *independent* of therapy and professional services. This is an outcome that few psychiatric and drug researchers pursue, especially the latter since it would limit product profitability. Apparently, Dr.

IS BEHAVIORISM BECOMING A PSEUDOSCIENCE?

Wakefield believes that he is being even-handed when he evaluates behavioral research by a higher standard than he applies to drug research. (p. 240)

Allow me first to say that I think it is inappropriate to suggest that those who do research on drug treatment would generally prefer to make money than to cure the illnesses that they study. Such demonizing should have no place in scholarly discussions.

Wong accuses me of unfairness in criticizing behavioral intervention's lack of generalizability but not the equal lack of generalizability of drug treatment. It is true that current psychotropic medications for schizophrenia do not cure but at best control symptoms, so in this regard the treatments are on a par. But there are reasons why lack of generalization is a special concern for behavioral treatments. Token economies require not just a minimal action like taking a pill or occasional professional monitoring but ongoing administering of reinforcers requiring environmental control difficult to achieve in a natural environment. Thus, in an age of deinstitutionalization, generalization is crucial for behavioral intervention in a way it is not for medication. As noted, behavioral researchers themselves have consistently recognized this reality. So, it is misleading for Dr. Wong to suggest that generalizability is merely an add-on benefit that researchers seek in order to confer independence on patients; rather, it is central to treatment success.

The issue of patient independence raised by Wong deserves comment. Baker (1988) pondered the challenge of independence in community living when progress depends on ongoing environmental control:

One problem with establishing a token economy as part of a more general rehabilitation programme preparing patients for discharge is that the methods it employs represent something of a goal clash with life in the community. The giving of tokens for self-care, socialability, etc., is certainly culturally abnormal....Additionally, while a major goal of psychiatric rehabilitation may be to develop a patient's independence, the token economy uses methods which could be thought of as overly controlling....[A] token economy unit in the community has the advantage in terms of reducing the problem of generalization, although when considering the norms of communities in the UK one wonders whether token economy could be congruent with life in the community. (p. 637)

Baker's point applies to the US as well: even if token economy methods can be continued, they do not represent a normalization of life relative to community standards.

Wong's fourth counterargument is as follows:

Fourth, many of Dr. Wakefield's criticisms were aimed at 30-year-old demonstration projects....In the normal course of technological progress, these demonstration projects would have been replicated, refined, tailored for different subpopulations and settings, and upgraded to maximize stimulus generalization, response generalization, and maintenance. This never happened....The deinstitutionalization movement transferred patients out of the state hospitals and back to their home communities, where community-based treatment programs revolved around drugs and drug management. Meanwhile, inadequate funding hobbled the development...[of] programs suitable for open settings. (p. 241)

This passage resolves the generalization issue in my favor. Wong acknowledges that scientific evidence for generalization of token-economy treatment results to community settings “never happened.” He offers several explanations, including lack of focus of early studies on generalization, deinstitutionalization, and lack of funding. For whatever reason, he and I are in agreement that such scientific evidence is lacking.

Wong speculates about what might have happened if deinstitutionalization and loss of funding hadn’t occurred. Behaviorists, he says, would eventually have done the research and succeeded in achieving generalization from hospital treatment to community behavior. I trust that no one will confuse this speculation with scientific evidence.

Wong also says: “These preliminary demonstrations were needed because of the historic and prevailing view that these disorders were the manifestation of diseases and not modifiable by restructuring stimuli in the client’s immediate environment (a view defended by Dr. Wakefield, based partly on the age and popularity of these beliefs)” (p. 241). This is a partial mischaracterization in that I agree that operant interventions can influence symptoms as I stated: “The token economy literature, by showing that even bizarre psychotic behaviors are influenced by learning to some extent, did offer a corrective to ideological claims of rigid biological determination of psychotic symptoms” (p. 206). This is entirely consistent with these disorders likely being due to underlying diseases.

WYATT AND MIDKIFF ON ETIOLOGY

I offered a commonsense argument for the implausibility of a reinforcement account of schizophrenogenesis based on such facts as the bizarreness and harmfulness of the symptoms and their persistence across environments as radically different as loving family, mental institution, and indigent homelessness—reasons that biological causes have been assumed since antiquity. Wyatt and Midkiff derided the commonsense argument as akin to saying that UFOs must exist because many people say they do. But the point—also going back to antiquity—is not obeisance to authority but a sense of humility: when most people believe something to which everyone has access to the evidence, they can certainly be wrong, but if one disputes the belief, one should at least offer evidence and explain why the people have commonly been misled. In the case of UFOs, we understand why the evidence that convinces UFO believers is misleading and can be due to a variety of other causes, we have confirmed alternative explanations for many claimed sightings, and we have background theories that suggest the *prima facie* implausibility of the belief that alien spaceships are visiting earth in the described manner. In the case of schizophrenic symptoms, the evidence that has convinced people over millennia of a biological cause cannot so easily be discounted. Such evidence includes, for example, not only the bizarreness and cross-environment persistence of the symptoms but also cross-cultural occurrence of similar symptoms at about the same rate, and the frequent relatively sudden onset of symptoms in young adulthood in a way that does not seem linked to environmental context, as well as myriad other strands of evidence. There is nothing analogous in this situation to the belief in UFOs.

IS BEHAVIORISM BECOMING A PSEUDOSCIENCE?

To their credit, Wyatt and Midkiff do take my challenge that belief in behavioral causation is based not on science but on blind faith in the existence of scientifically unestablished reinforcers seriously, and attempt to elaborate a behavioral theory of schizophrenia that might explain why medical opinion has been misled for 2500 years:

Hallucinations and other bizarre behaviors may well be maintained by withdrawal of demands, or reinforced by kindly attention, or triggered by the doctor's suggestion, "Are you hearing voices today?" These are common examples, yet Wakefield evidently has never heard of them. (p. 228)

Challenged for evidence that psychotic symptoms are caused by reinforcement, the best Wyatt and Midkiff can do is to offer a sheer speculation, without any support, that these symptoms "may well be maintained" by certain reinforcers such as reduction of social demands. This confirms the "blind faith" charge, insofar as scientific standards of evidence go. Note that their stated hypothesis addresses only maintenance, not etiology, and evades the question whether the reinforcers are powerful contributors or just minor players (perhaps representing secondary gain) in a basically biological etiology.

Regarding the hypothesized reinforcers themselves—withdrawal of social demands, kindly attention, and the doctor's suggestion—there is an ad hoc, "just so story" quality to these speculations. It is of course almost always possible to come up with possible reinforcers for almost anything; the worse an individual's physical or mental condition, after all, the more the individual is relieved from social role responsibilities in accordance with the "sick role," and the more "kindly attention" and doctors' inquiries the individual is likely to garner. These reinforcers are explanatorily neither highly potent (they are omnipresent without psychotic symptoms occurring) nor highly specific (i.e., they do not explain the specific form the symptoms take). Without empirical evidence, they serve as all-purpose behavioral pseudo-explanations.

To the "commonsense" objection to behavioral etiology that symptoms persist resiliently across radically differing environments, Wyatt and Midkiff respond as follows:

However, Wakefield's speculation is easily undone when one considers that primary reinforcers (and many secondary reinforcers) are the same across settings. If a patient's best method of getting his parents, neighbors or treating professionals to cease their demands (for example, demands that he engage in useful activity) is to create a psychotic disturbance, it is likely to be reinforced across settings. The exception of a well-designed token economy in which such behavior is not reinforced tends to prove the rule. (p. 229)

First, note the claim, essential to Wyatt and Midkiff's position, that psychotic symptoms, with all the negative and intrusive consequences they entail, are still for some individuals the "best method" for getting parents, neighbors or treating professionals to cease their demands for useful activity (if there were a better method, presumably other reinforcers would dominate). There is of course no evidence that this wildly implausible hypothesis applies to any sizable number of schizophrenic individuals. Wyatt and Midkiff are quick to note that brain changes in the disordered predicted by biological approaches

may represent the effects of the disorder or its treatment rather than the cause, but fail to consider whether social withdrawal may represent not the reinforcer/cause but the non-functional effect of independently biologically generated psychotic symptoms that cause the desire for withdrawal and the lack of ability to meet social demands.

In any event, it seems clear that psychotic symptoms endure in environments in which there are no kindly attenders or suggestive doctors and where withdrawal from social demand is not an issue because there are no such demands (e.g., homeless mentally ill, chronically hospitalized “warehoused” mentally ill). Empirical evidence can always overturn such apparent truths, but there exists no such empirical evidence either for the “best method to reduce social demands” claim or the “cross-environment reinforcement” claim; they both amount to ad hoc hypotheses to preserve the behavioral view.

A further commonsense objection was that the symptoms of schizophrenia are too bizarre, debilitating, and stigmatized to be functionally caused behaviors in natural environments. Wyatt and Midkiff attempt to take the paradox out of their position by a series of analogies that remind us that reinforcement can yield stigmatized behaviors:

We are daily confronted with examples in which maladaptive, stigma-producing, painful behaviors are maintained by other consequences. For example, the stigma that accompanies obesity is intruded upon and overwhelmed by the immediacy of the good taste of food. Community disapproval comes with smoking, but the behavior is maintained by negative reinforcement as one’s withdrawal symptoms are removed by lighting up. The social rejection accruing to a tantruming adult (intermittent explosive disorder) is not the only consequence. Such fits of temper are reinforced when the tantrum thrower gets his way. An Obsessive-Compulsive ritual is maintained by the reduction in anxiety that results when it is performed, even though it may greatly disrupt one’s life. An irrational fear of heights may cause the sufferer to race from the third floor to the ground level, with the result that the action is negatively reinforced by the resulting feeling of relief, notwithstanding the disruption to the client’s lifestyle. (p. 228)

However, this argument-by-analogy has many problems. Mental disorders have quite different etiologies, so simply analogizing from one to another is not a credible scientific argument. Moreover, why not give equal or greater weight to analogies to symptomatically more similar conditions, such as general paresis, Rhett’s disorder, or Alzheimer’s disease, where biological causes of symptoms—symptoms that also allow people to evade social demands, unlike the symptoms of Wyatt and Midkiff’s analogized conditions—have been established? Nonetheless, it is important to consider to what degree these analogies do render the behavioral position on schizophrenia more plausible.

The problem is that, even if one accepts behavioral accounts of these other conditions for the sake of argument, the analogies do not adequately address the specific sources of doubt regarding behavioral etiology and maintenance of schizophrenic symptoms, for several reasons. (1) The “analogous” conditions are nowhere near as stigmatized, and on average have nowhere near the destructive impact on the individual’s life, as schizophrenic symptoms; for example, none warrant complete withdrawal from social roles. (2) The “analogous” reinforcers (taste of food, avoidance of withdrawal

symptoms, reduction of anxiety, gaining dominance) are intense biologically anchored phenomena that are understandable as a motive for maladaptive behavior, whereas the proposed reinforcers of schizophrenic symptoms appear relatively weak by comparison. (3) Consequently, there does not seem to be a reasonable proportionality between the proposed reinforcers and the extraordinary costs of schizophrenic symptoms. (4) One can see that eating may for some people be the best method to obtain taste, smoking the best method to avoid withdrawal symptoms, fleeing a phobic stimulus the best method to reduce anxiety, exploding with anger the best method to get a resistant other to do what one wants, and so on, but there seem to be many other, less costly ways than schizophrenic symptoms to get others to stop demanding socially useful behavior. Other than someone who is creating an ad hoc behavioral explanation of schizophrenia, who would judge it plausible that psychotic symptoms, with the many endless demands of treatment, hospitalization, and so on that they have traditionally entailed, are explainable as the best method for the individual to obtain relief from social demands? Finally: (5) There are direct means-ends explanatory links, not only apparent to common sense but often stated in first-person reports, between the desire for the stated reinforcers and the “analogous” stigmatized behaviors (e.g., between eating and obtaining the taste of food, anger and getting one’s way, smoking and avoiding withdrawal symptoms, etc.), whereas no such direct or commonly self-reported explanatory link exists between the desire to reduce social demands and the psychotic symptoms and their proposed reinforcers; patients do not generally report that the symptoms were their means of avoiding social demands.

None of this proves that Wyatt and Midkiff are wrong. It does show that their analogies are too weak to be credible. Given that these strained analogies are the only “evidence” they present, there is no cogent reason for believing their account.

There has been ample opportunity to test the theory that schizophrenia is caused by contingent reinforcement for avoiding social demands. Doing careful studies of reinforcers in natural environments and their relationship to the occurrence of psychotic symptoms is challenging but not inherently more so than other kinds of causal studies. Yet, despite decades of dominance by behaviorism in American psychology in the last century, the behavioral theory has never been scientifically supported by a body of research. Nonetheless, Wyatt and Midkiff assert that this is what causes schizophrenia. Claiming to know the cause of schizophrenia in such circumstances is pseudoscientific.

I note in passing that, from my clinical perspective, Wyatt and Midkiff’s theory is disturbingly similar to that of many in the general public who don’t want to admit that mental illness really exists in a child, and who blame the symptoms on character defects instead. They assume, for example, that the child’s withdrawal and lack of social performance is motivated by the desire to avoid responsibility, and their goal becomes to deprive the child of “reinforcement” for his or her behavior, sometimes leading to harsh deprivation. Family disputes often occur about whether to interpret a child’s symptoms as avoidance of social demands or as mental illness. To take a famous example, John Hinckley’s father insisted on keeping him from returning home or giving him financial support because, after he failed to achieve social role success, the father thought that

Hinckley's symptoms should not be rewarded; this occurred just prior to his assassination attempt on President Reagan. Dressed up in scientific language, Wyatt and Midkiff's theory is quite similar, that these individuals are not ill but evading social demands.

WONG ON ETIOLOGY

Dr. Wong also offers some arguments on the causation question:

Dr. Wakefield scoffs at my supposition that environmental conditions, such as deprivation, poor socialization, and aversive stimulation, might play an important role in the etiology of mental disorders....Why Dr. Wakefield, a social work professor, should object so strongly to this idea is curious, since...there is recent evidence to support it...some provided by a social work researcher. (p. 239)

I did not scoff at the idea that environmental conditions can influence the development of schizophrenia. This is part of the well-supported stress-diathesis model postulating genetic-environmental interaction in the etiology of schizophrenia, a view so widely accepted that NIMH's schizophrenia information website states: "Interactions between genes and the environment are thought to be necessary for schizophrenia to develop." Note that, irrespective of the causes of schizophrenia, I believe in an array of environmental interventions of the kind social workers are often best prepared to render.

However, Wong tries to twist the well-supported environmental influence on schizophrenogenesis into support for a specifically behavioral theory in which reinforcement for symptomatic behavior is the primary causal agent. That leap just does not follow from the data cited by Dr. Wong, any more than, say, the psychoanalytic environmental notion of a schizophrenogenic mother follows. For example, Hudson (2005), the social work researcher to whom Wong refers, concludes that it is most likely *economic stress*—and presumably the resultant deprivation and psychological distress -- that is the explanatory causal factor linking low SES with higher rates of schizophrenia: "Of the various social causation hypotheses tested, the idea that the impact of SES on mental illness is mediated by economic stress received the strongest support" (p. 17). "Environmental causation" need not imply "contingent reinforcement"!

Dr. Wong asks:

And, what is so groundless and illogical about the following argument?: 1) If reinforcement contingencies can create and increase psychotic behavior (as in the study by Allyon, Haughton, & Hughes, 1965); and, 2) if reinforcement contingencies can decrease or eliminate psychotic behavior; then, 3) is it not reasonable to assume that reinforcement contingencies might have contributed to the emergence of psychotic behavior in the first place? (pp. 239-240)

To take Dr. Wong's question seriously, there are at least two things wrong with this argument. First, the argument is manifestly invalid because there are many conditions that have symptoms that behavioral interventions can influence but are known to be physical disorders in which the symptoms do not initially emerge due to reinforcement (e.g., behavior of the genetically mentally retarded or brain damaged). NIMH recently

IS BEHAVIORISM BECOMING A PSEUDOSCIENCE?

called for grant proposals exploring “behavioral interventions that improve cognitive function” in those with Parkinson’s disease; such rehabilitation or disease management for many biological disorders can include behavioral components. So, the fact that behavioral intervention can influence psychotic symptoms does not make it “reasonable to assume that reinforcement contingencies might have contributed to the emergence of psychotic behavior in the first place,” any more than the fact that headaches can be cured by aspirin implies that headaches are caused by an aspirin deficiency.

The second thing wrong is that the argument is used to conclude that behavioral rather than biological causes underlie schizophrenia, yet it applies equally to biological causes. We might imagine a biologically oriented opponent asking: “What is so illogical about the following argument?: 1) If biological processes (e.g., drugs) can create and increase psychotic behavior; and (2) if biological processes can decrease or eliminate psychotic behavior; then, 3) is it not reasonable to assume that biological processes contributed to the emergence of psychotic behavior in the first place?” This argument is as fallacious as Dr. Wong’s; drugs can obviously influence conditioned responses.

CONCLUSION

This interchange, despite all the heat, has arrived at a scientific consensus about which we are all in agreement. Both replies, like the broader literature, acknowledge that, as I claimed, the generalizability of the effects of token economy interventions for schizophrenia from controlled environments to uncontrolled natural settings has proven problematic and never been scientifically demonstrated (Wong: “That didn’t happen”; Wyatt and Midkiff: “a work in progress”). This provides a major scientific reason to explain why behavioral treatments were sidelined in an age of deinstitutionalization, and a motive for attempting to develop other approaches that can be efficiently continued in natural environments, such as drug treatments. I conclude that behaviorism’s fate lies not in its political stars but in itself. When behaviorists actually do scientifically demonstrate effectiveness and offer persuasive theoretical accounts, as in the study of phobias, politics does not stop them from making major and influential contributions to treatment.

Nor, if the responses are any guide, is there any serious scientific evidence after all these years for a behavioral theory of the etiology of schizophrenia, even though such a theory is eminently testable. As the philosopher Frank Cioffi has argued, the mark of a pseudoscience is not, as positivists originally believed, that it is untestable, but rather that it is testable but its adherents do not put it to potentially disconfirming tests. By this and many other benchmark philosophical criteria, behaviorism of the brand defended by the target authors is not a progressive science and, given the “blame it on politics” approach evidenced here, is indeed in danger of becoming a pseudoscience.

REFERENCES

- Ayllon, T., & Azrin, N. H. (1968). *The token economy: A motivational system for therapy and rehabilitation*. New York: Appleton-Century-Crofts.
- Baker R. (1988). Token economy in a hospital rehabilitation system. *Journal of Advanced Nursing*, 13, 631-639.

- Dunlap, G. (1993). Promoting generalization: Current status and functional considerations. In R. Van Houten & S. Axelrod (Eds.), *Behavior analysis and treatment* (pp. 269-296). New York: Plenum Press.
- Fuoco, F. J., & Tyson, W. M. (1986). Behavior therapy in residential programs for psychiatric patients. In F. J. Fuoco & W. P. Christian (Eds.), *Behavior analysis and therapy in residential programs* (pp. 231-259). New York: Van Nostrand Reinhold.
- Glynn, S. M., Marder, S. R., Liberman, R. P., Blair, K., Wirshing, D. A., Ross, D., & Mintz, J. (2002). Supplementing clinic-based skills training with manual-based community support sessions: Effects on social adjustment of patients with schizophrenia. *American Journal of Psychiatry*, 159, 829-837.
- Horner, R. H., Dunlap, G., & Koegel, R. L. (1988). *Generalization and maintenance: Life-style changes in applied settings*. Baltimore, MD: Paul H. Brookes Publishing Co.
- Horwitz, A. V., & Wakefield, J. C. (2007). *The loss of sadness: How psychiatry turned normal sorrow into depressive disorder*. New York: Oxford University Press.
- Hudson, C. G. (2005). Socioeconomic status and mental illness: Tests of the social causation and selection hypothesis. *American Journal of Orthopsychiatry*, 75, 3-18.
- Kazdin, A. E., & Bootzin, R. R. (1972). The token economy: An evaluative review. *Journal Of Applied Behavior Analysis*, 5, 343-372.
- Kopelowicz, A., Liberman, R. P., & Zarate, R. (2006). Recent advances in social skills training for schizophrenia. *Schizophrenia Bulletin*, 32 (S1), pp. S12-S23.
- Lauriello, J., Bustillo, J., & Keith, S. J. (1999). A critical review of research on psychosocial treatment of schizophrenia. *Biological Psychiatry*, 46(10), 1409-1417.
- Paul, G. L., & Lentz, R. J. (1977). *Psychosocial treatment of chronic mental patients: Milieu versus social-learning programs*. Cambridge, MA: Harvard University Press.
- Stark, L. J., Collins, F. L. Jr., Osnes, P. G., & Stokes, T. F. (1986). Using reinforcement and cueing to increase healthy snack food choices in preschoolers. *Journal of Applied Behavior Analysis*, 19, 367-379.
- Stahl, J. R., & Leitenberg, H. (1976). Behavioral treatment of the chronic mental hospital patient. In H. Leitenberg (Ed.), *Handbook of behavior modification and behavior therapy* (pp. 211-241). Englewood Cliffs, New Jersey: Prentice-Hall.
- Stokes, T. F., & Baer, D. M. (1977). An implicit technology of generalization. *Journal of Applied Behavior Analysis*, 10, 349-367.
- Stokes, T. F., & Osnes, P. G. (1989). An operant pursuit of generalization. *Behavior Therapy*, 20, 337-355.
- Wakefield, J.C. (2006). Is behaviorism becoming a pseudo-science?: Power versus scientific rationality in the eclipse of token economies by biological psychiatry in the treatment of schizophrenia. *Behavior and Social Issues*, 15, 202-221.
- Wong, S. E. (2006a). Behavior analysis of psychotic disorders: Scientific dead end or casualty of the mental health political economy? *Behavior and Social Issues*, 15, 152-177.
- Wong, S. E. (2006b). Response to the commentaries. *Behavior and Social Issues*, 15, 232-243.
- Wong, S. E., Martinez-Diaz, J. A., Massel, H. K., Edelstein, B. A., Wiegand, W., Bowen, L., & Liberman, R. P. (1993). Conversational skills training with schizophrenic inpatients: A study of generalization across settings and conversants. *Behavior Therapy*, 24, 285-304.
- Wong, S. E., Morgan, C., Crowley, R., & Baker, J. N. (1996). Using a table game to teach social skills to adolescent psychiatric inpatients: Do the skills generalize? *Child and Family Behavior Therapy*, 18, 1-17.

IS BEHAVIORISM BECOMING A PSEUDOSCIENCE?

- Wyatt, W. J., & Midkiff, D. (2006a). Biological psychiatry: A practice in search of a science. *Behavior and Social Issues, 15*, 132-151.
- Wyatt, W. J., & Midkiff, D. (2006b). Six-to-one gets the job done: Comments on the reviews. *Behavior and Social Issues, 15*, 222-231.