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PSYCHIATRY'S THIRTY-FIVE-YEAR, NON-EMPIRICAL REACH FOR BIOLOGICAL EXPLANATIONS

W. Joseph Wyatt[1]
Marshall University

Donna M. Midkiff
River Park Hospital

ABSTRACT: This is our third article in a series that began with a special issue of *Behavior and Social Issues* in 2006. Here we briefly review our central points from the first two articles. First is that over the past thirty-five years, claims of biological causation of mental and behavioral disorders have gone well beyond the research data, for reasons that are largely related to psychiatry's lost esteem and protection of its "turf," as well as to the financial interests of the pharmaceutical industry. Our second position is that claims of psychotropic drugs' effectiveness have been overstated. We respond, as well, to the protestations of Professor Jerome C. Wakefield who defends biological psychiatry. We also provide an update on relevant events within the drug industry since our last article in this series.
KEYWORDS: FDA, behaviorism, biological causation, pharmaceutical industry, organized psychiatry, efficacy of psychotropic medications, identical twin studies, brain imaging studies, psychological paradigms

In this, our third paper in this exchange, we first reiterate what has taken place up to now. We and Stephen Wong (Wong, 2006; Wyatt & Midkiff, 2006) authored anchor papers in a special issue of *Behavior and Social Issues* (Mattaini, 2006). Our paper described the turn to biological explanations for mental and behavior disorders that has occurred in both the professional and popular cultures in the past thirty-five years. We also reviewed the non-empirical reasons for that turn. Those reasons included efforts by organized psychiatry (American Psychiatric Association) to recoup lost esteem and to rebuff "intruder" professions (clinical psychology, etc.), as well as the financial interests of the pharmaceutical industry. All of this has occurred with the evident support of the American Medical Association. We refer the reader to that earlier article for a complete presentation of those issues.

[1] Correspondence regarding this article may be sent to the first author at Department of Psychology, Marshall University, 1 John Marshall Dr., Huntington, WV, USA 25755 or Wyatt@Marshall.edu; or to the second author at Department of Psychology, River Park Hospital, PO Box 1875, Huntington, WV, 25819 USA or Psyd@comcast.net.

In reply to our article, six reviewers responded favorably, or at least benignly, elsewhere in that issue to the facts we had adduced. An additional reviewer objected to our theses. Jerome C. Wakefield (Wakefield, 2006) found our discussion to be less than compelling. He defended biological causation mainly by attacking behaviorism. We responded with our second article in this exchange in which we pointed out that Professor Wakefield had only tangentially addressed much of the general case we had made, and had failed to address many of the specific points we had raised, as well. Moreover, his critique of behaviorism was so limited in its focus that it was less than compelling (Wyatt & Midkiff, 2006b). That exchange concluded the special issue of *Behavior and Social Issues* and, we had thought, the discussion. However, Professor Wakefield contacted the editor and requested an opportunity to again convince the journal's readers that our presentation had failed, and that his efforts to dismantle behaviorism had succeeded.

As a result, now Professor Jerome C. Wakefield (this issue) has again struggled to defend an extreme version of biological causation of mental disorders, an extremism whose many weaknesses we have exposed in our first and second papers in this series (Wyatt & Midkiff, 2006a; Wyatt & Midkiff, 2006b). We now reply to Professor Wakefield's follow-up effort to both attack the behavioral model, and to defend a biological causation model. We judge his efforts to have again come up short, not a surprising outcome given that we have relied upon evidence, while Professor Wakefield has tended to rely upon verbal style, including a tendency to claim that our examples lacked completeness, our analogies were not analogous enough, or the data we presented were somehow off the mark. In the end, those were weak methods by which to attempt to undo our findings. We will provide the reader with examples of that style below.

Wakefield's defense of extreme biological causation (this issue) continues to miss its mark for several reasons. We feel that a fair reading of Wakefield's recent article shows that his essential tactic in defense of biological causation is to mount an attack on behaviorism, especially hospital token economy programs for schizophrenics and, more narrowly, the difficulty of those programs in achieving generalization of hospital treatment gains to the world outside the hospital. Such focus is interesting, but much too narrow in perspective to invalidate behaviorism, or any other model. Moreover, it is not a means by which anyone could logically refute our central theses—that the research said to support biological causation is relatively weak, and the claims of drug effectiveness are often overstated. Wakefield's approach continues to be unsatisfying, especially so when one considers that the above statement of our goals was difficult to miss. It had appeared in the second paragraph of our first paper in this series (Wyatt & Midkiff, 2006a) and in the first sentence of our second paper (Wyatt & Midkiff, 2006b).

Thus, Wakefield had begun by misrepresenting our goals—alleging that we were trying to validate behaviorism and that we were attempting to do that by revealing the shortcomings of biological causation. Having built that straw man, he now turns consistency on its head by attacking behaviorism, in his effort to defend the biological model (Wakefield, this issue).

When it comes to demonstration of a theory's validity, or lack of it, we believe that a fair reading reveals that Wakefield wants to have it both ways. He wishes to validate

biological causation by tearing down behaviorism, but chides that it would be improper to do the reverse—which was not our purpose anyway.

Thus, when one boils it down, Wakefield has first misrepresented our purposes, then employed a double standard to show what is wrong with a position we did not take. It should surprise no one then, to know that he accused *us* of employing straw man tactics. Moreover, Professor Wakefield has used a myriad of other tactics, statements and conclusions that, once one goes beyond his admirable verbal style, fall short as efforts to either pummel behaviorism or to defend biological causation. Some examples are informative.

DO DRUG ADVERTISEMENTS GO TOO FAR?

In responding to our claims that drug ads routinely go well beyond the data (Wyatt & Midkiff, 2006a), Wakefield had earlier written that it was medication that had "emptied the asylums" (Wakefield, 2006). Nothing more, just the medication. He had not considered numerous other factors that deserve as much, or more, credit for emptying the state hospitals. As we pointed out in our subsequent article (Wyatt & Midkiff, 2006b), other factors that had contributed to deinstitutionalization were the toughening of commitment laws; the advent of Social Security benefits which allowed for payment of patients' supported living arrangements in the community; the creation and growth of the community mental health movement; and legislative mandates that patients be deinstitutionalized. We added, as well, that there is a vast difference between "discharge" and "cure," as evidenced by the presence of homeless, schizoid individuals on our streets, and the near one hundred percent recidivism and re-hospitalization rates for the chronically mentally ill. Clearly, Wakefield's notion that medications alone had "emptied the asylums" had been simplistic, shortsighted and wrong.

Wakefield has now responded (this issue). Straining to devise a *post hoc* account for the variables he had earlier overlooked, and minus citation of supporting evidence, he responded as follows: "However, the other factors had their major impact because of the ability of medication to allow community placement for some previously unreleasable patients" (Wakefield, this issue, p. 175). Such an after-the-fact scramble to account for phenomena he earlier had overlooked is less than compelling. It is also erroneous. There is no reason we know of to move medications to the top of the heap of reasons that our asylums have been "emptied."

Here is another example of Wakefield's strenuous, but ultimately failed, effort to defend over-the-top subscription to both the biological causation model and to drugs as the treatment of choice. To return to one of our central theses—that drug ads routinely overstate medications' effectiveness—we provided a half-dozen verbatim examples of antidepressant ads that claimed medication would correct a serotonin imbalance, to treat depression. We contrasted those advertisements with the opinions of a number of neurobiological researchers who disputed that a serotonin-depression connection has even been proven to exist (Wyatt & Midkiff, 2006b).

The contrast between the advertisements and the experts validated our view. In reply, Wakefield sputtered that we had misrepresented his position as "pro-drug." (We hadn't said that, but it is probably a fair representation, given his drugs-emptied-the-asylums gaff.) He complained that we were guilty of "brazen distortion" of his position, etc. In fairness to Wakefield, he *had* said that the humoral approach to mental disorders has been oversold. Thus, the reader must continue to ask which is the waffling Wakefield's preferred position on psychotropic medications—his over-the-top claims that drugs alone can empty mental hospitals, or his rather convoluted agreement with our position that pharmaceutical company claims are incredible.

We feel that we have more than demonstrated that claims of drug effectiveness are routinely overstated. Not only do the advertisements frequently misstate the causes of mental and behavioral disorders, but drugs did not "empty the asylums," at least not without enormous help from community mental health agencies, Social Security funding of supported community living arrangements for discharged patients, laws that prevent patients from being locked up for undetermined lengths of time without probable cause hearings, and legislation that continues to force hospital officials to discharge patients to the communities or be faced with criminal charges.

BIOLOGICAL CAUSATION: PSYCHIATRIC GUILD AND PHARMACEUTICAL INDUSTRY OVERKILL?

On the first page of our initial paper we noted that there is little debate that a number of mental and behavioral disorders (Down's syndrome; autism; various dementias; disorders attributable to vitamin deficiencies, tumors or endocrine diseases; etc.) are biologically caused. Our second thesis held that, apart from such disorders as those, there is surprisingly little research evidence to support the commonly heard claim that our biology accounts for the majority of cases of common disorders such as unipolar depression, anxiety disorders, child conduct problems, ADHD and schizophrenia (Wyatt & Midkiff, 2006a).

To support our position we described a number of phenomena that we will list below with brief mention of Wakefield's (Wakefield, 2006) responses to them. See our earlier article for a more complete discussion.

The American Psychiatric Association's Collapse

In 2003 a federation of professionals and former patients known as MindFreedom had written to the American Psychiatric Association and asked it to provide any scientific evidence of biological causation of common disorders (depression, anxiety disorders, etc.). The psychiatric organization's medical director replied to MindFreedom by letter, referring to various textbooks and medical journals in which the relevant research was readily available. In the weeks that followed, MindFreedom's leaders reviewed those sources and wrote back to the APA's medical director, pointing out that those texts tended to say that the causes of disorders are unknown. At that point the American Psychiatric Association provided no further references or data, but instead issued a

position statement that asserted the APA's faith in biological causation (MindFreedom, 2003).

As we had noted in our first paper, the psychiatric guild's collapse was, to understate the case, revealing. Also revealing was Wakefield's failure to even mention the MindFreedom episode in his reply to our first paper. We noted that omission in our second paper. Now Wakefield (this issue, p. 174) deals with the disquieting MindFreedom-APA exchange as follows: "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." Professor Wakefield's response was as unfortunate as that of the American Psychiatric Association. When an organization that arguably is the most visible proponent of biological causation is kayoed in the second round, when a powerful guild is revealed to be without evidence for its claims, that is news. In our view it can be neither ignored nor dismissed as a public relations stunt, as Wakefield has cavalierly done.

Medication Ineffectiveness

Mounting evidence shows that a number of highly touted medications are less effective than advertised. In our initial paper (Wyatt & Midkiff, 2006a) we had pointed to major research showing that many anti-depressants are less effective than advertised. For example, Kirsch, Moore, Scoboria and Nicholls (2002) reviewed thirty-eight studies of anti-depressants such as Prozac, Zoloft, Paxil, Serzone, Celexa and Effexor whose results had been reported from1987-1999. On the 50-point Hamilton Depression scale the studies showed an average 10-point improvement in mood for patients who took the drugs, compared to an 8-point improvement for those who took a placebo. We pointed out that the 2-point advantage for the medications is unlikely to be meaningful in the real world, and even that slight advantage might not have occurred at all, were it not for the methodologically questionable placebo washout-group experimental designs. With an opportunity to comment in his first reply, Wakefield took a pass (Wakefield, 2006). At his second opportunity, he has done so again (Wakefield, this issue).

Psychiatry's Non-Empirical Turn to Biological Causation

In our initial article we traced a 35 year history in which psychiatry began and perpetuated an embrace of biological causation, not as a result of compelling research, but rather as a means to regain esteem and protect its turf from intruder professions (psychology, counseling, clinical social work, etc.). We also revealed an additional reason behind psychiatry's embrace of the biological causation model, and how that tactic was designed to deal with declining numbers of medical school graduates choosing psychiatry as a career—a drop from 11% to 5% from 1970-1980. That is, by presenting itself as more biological and, thus, less psychodynamic, psychiatry embarked upon a public relations effort designed to shore up the field's interest among young doctors (Wyatt & Midkiff, 2006a). With two opportunities to respond to this history, Wakefield (2006, and this issue) has twice chosen not to comment.

Advertising's Influence Upon Prescribing Practices

While Wakefield contends that the increasing use of drugs is due mainly to their effectiveness, we adduced evidence showing that the increased reliance on drugs to treat mental disorders may well be due to factors other than their effectiveness--drug company tactics, in particular. We showed how the doubling of drug company marketing jobs since 1995 has been paralleled by enormous increases in prescriptions of psychotropic medications. We showed how, over the same time span, the number of drug company jobs in research and development fell slightly.

We discussed a study from the *Journal of the American Medical Association* in which actors who had been trained to display depressive symptoms were six times more likely to be prescribed a specific drug (the anti-depressant Paxil) if they mentioned its name to a doctor and added that they had seen it advertised on TV (Kravitz, Epstein, Feldman, Franz, Azari, Wilks, Hinton & Franks, 2005). Given the energy with which Wakefield has chosen to defend drug treatment, it is unfortunate that he has chosen silence on that study, as well as on the doubling of drug industry marketing jobs and the parallel increases in prescriptions of psychotropic medications.

Further Aspects

In our initial paper (Wyatt & Midkiff, 2006) we had questioned the informational value of two lines of research generally thought to provide validation of biological causation. They are studies of identical twins reared apart, and studies of brain structure and functioning, as are typically done either on autopsy or on the living via PET scan or functional Magnetic Resonance Imaging (fMRI) technology. Although we will only briefly review that discussion here, we had concluded that these two lines of research are able to provide little evidence of biological causation of mental disorders. See our article that began this exchange for that entire discussion (Wyatt & Midkiff, 2006a).

To briefly reiterate, the twin studies consistently show above-baseline concordance for mental disorders, even when the monozygotic (identical) twins are reared apart. Although that fact is typically taken as powerful evidence of genetic causation, we pointed to what we feel is a fatal flaw in the studies. That flaw is the erroneous assumption that twins, once separated, are reared in quite differing environments, thus leaving genes to account for the twins' concordance. What has routinely been unaccounted for is that environments respond similarly to appearance (especially level of attractiveness, but also to factors such as height) and rate of development. Because identical twins possess identical physical appearance and will reach puberty at the same age, their environments will tend to treat them similarly in ways that have long been known to influence mental and behavioral status. We provided numerous citations, many from the social psychology literature, to support those facts. We went further, pointing out that both families and adoption agencies frequently *demand* that adoptees be placed in homes quite similar to their biological homes with regard to variables that are known to be related to mental status (i.e., religious affiliation, urban/rural placement, etc.). Our conclusion was not that this validates environmental causation, but that these

confounding variables leave studies of monozygotic twins (who were reared apart) of dubious usefulness as evidence of genetic causation.

Similarly, we pointed out that studies of brain structure and function are plagued by a number of factors. Chief among them is the difficulty of teasing out causation from correlation. Is an unusual structure or function in the central nervous system the cause or the result of a mental disorder, or did some third factor cause both? Nobody knows.

In reply to this discussion, Wakefield employed several tactics. He claimed that we had ignored most of the research, that we had provided no citations (odd, given that any reader of our initial paper in this exchange [Wyatt & Midkiff, 2006a] will see numerous citations in that section), that we had not attempted an extensive review of the literature (which had not been our purpose) and that we had created "*ad hoc* alternative hypotheses." He added that our points about the inadequacies of the twin and brain structure studies "have been around for a long time and have been extensively empirically studied…" and he concluded that our discussion had been "comfortably myopic" (Wakefield, this issue, p. 174).

Could Wakefield have been correct? Could the concerns we had raised about interpretative conundrums and confounding variables in twin studies and brain imaging studies be so open, well known and routinely acknowledged that nobody continues to be misled? To briefly explore that possibility we first looked at the chapters on depression and schizophrenia in two of the most widely adopted textbooks in the field of abnormal psychology. They were the fourth edition of David H. Barlow and V. Mark Durand's *Abnormal Psychology: An Integrative Approach* (2005) and the sixth edition of Ronald J. Comer's *Abnormal Psychology* (2007). Both textbooks provided discussion of identical twin studies. Neither mentioned how variables such as physical appearance, age of puberty, adoption practices, etc., elicit predictable environmental contributions to mental health. Neither discussed the fact that these environmental variables are confounded with genetic variables, a fact that leaves studies of twins reared apart of dubious usefulness. The Barlow and Durand text stated in part, "The best evidence that genes have something to do with mood disorders comes from *twin studies*..." (emphasis theirs, p. 225). If, as Wakefield believes, the genetic-environment confounds present in twin studies have long been adequately addressed in the literature, one would think that authors of leading textbooks in abnormal psychology would have noted it, but they have not.

Also regarding the twin studies, Wakefield now writes, "…Wyatt and Midkiff's two central points…have been around for a long time and have been extensively empirically studied…from classic articles by Scarr and Plomin et. al to Bouchard's analyses…none of which is considered in Wyatt and Midkiff's comfortably myopic discussion." Wakefield has not included those sources in his reference list, nor has he provided dates of publication in the text. Thus, myopic or not, we examined what we felt were representative publications by Plomin and Bouchard. A representative article by Bouchard and McGue (2003) contains a section on "Methodological Cautions" in twin studies. However, that section makes no mention of the variables about which we have raised concerns—physical appearance, age of puberty, adoption practices—variables well

known to influence the environment's reaction to children. (See the social psychology research we cited in our initial paper in this series, Wyatt & Midkiff, 2006a).

We next reviewed a major textbook that was co-edited by Robert Plomin titled *Behavioral Genetics in the Postgenomic Era* (Plomin, Defries, Craig & McGuffin, 2003), which was published by the American Psychological Association. This book is impressive in its depth and breadth, as well as in the credentials of its editors and chapter authors. We went to its index and found numerous sections indexed under the heading "twin studies." We reviewed those that dealt with affective disorders, anxiety, ADHD, personality traits, schizophrenia and one titled "reasons for using" (twin studies). In none of these were the confounding issues of physical appearance, age of puberty or adoption practices discussed. Thus, having reviewed representative works by two of the three scholars named by Professor Wakefield, it is clear that he is mistaken. The variables that confound twin studies tend not to be accounted for by leading scholars in the field.

We will add that the Comer text also presents the serotonin-depression link as valid, minus reference to experts such as we have cited (Wyatt & Midkiff, 2006b), and never mentioned the correlation-causation matter in its discussion of abnormal brain structure of schizophrenics. The same was true of the Barlow and Durand text.

Thus, while Wakefield has concluded that our concerns about studies of both identical twins and of the central nervous system have been adequately dealt with on a wholesale basis, a brief look at representative sources, including some suggested by Wakefield himself, reveals that he is mistaken. Our original concerns remain. Professor Wakefield, who described our position as "comfortably myopic," would do well to acknowledge that the environment-gene confounds we articulated tend to be unaccounted for across the landscape of widely accepted academic sources. Perhaps everybody is myopic, except professor Wakefield.

GENERALIZATION

In responding to our first article, and to that of Professor Wong, Wakefield undertook an effort to undo behaviorism by focusing on the frequent failure to maintain gains made in token economy programs by hospitalized schizophrenics, once they are discharged to the community. Professor Wong (Wong, 2006a, 2006b, & 2007) has answered that criticism. We will comment here only on Wakefield's scramble to regroup once we had pointed out the weaknesses of his position.

First, we noted (Wyatt & Midkiff, 2006b) that the failure to generalize is not evidence that a treatment is ineffective. Token economies change a great deal of schizophrenics' behavior in the hospital, as long as contingencies are maintained. If, upon discharge, the reinforcers for improved functioning no longer exist, it is little surprise that the improved behavior declines as well. Wakefield scoffed, "…Wyatt and Midkiff assert that this failure of generalizability to the community in fact shows that such treatment is successful!" We maintain that position, and offer that it is quite reasonable, for reasons that follow.

Consider that common among behavioral sciences researchers is the reversal design. In it, a treatment is undertaken, then stopped (or reversed). The subsequent return of problematic behavior provides evidence that the treatment brought about the improved behavior. We suspect that Wakefield has indeed at least a passing familiarity with reversal designs, but in this instance he opted for the expediency of disingenuousness, over the inconvenience of acknowledging that we had made our point.

Further, by analogy, we had suggested that many valid treatments are transitory: Exercise only strengthens the body as long as it is continued; a flu shot must be repeated annually to ward off that illness. Wakefield rejected those comparisons based upon his evident assumption that behavioral improvement somehow is not improvement at all, a position that on its face seems quite weak. Upon reading our analogous scenarios, Wakefield wrote that flu shots and exercise were not apt analogies because both "can be continued indefinitely without much trouble." We find his position to be anything but compelling. The reader will note that many treatments work only as long as they are continued—dialysis for renal failure, insulin for diabetes, blood pressure medication, on and on. But to employ Wakefield's reasoning, none of these treatments would be thought of as effective because they must be constantly repeated, or because patients sometimes discontinue them. In the end, on one side of this point stands Professor Wakefield, while on the other stands every tradition within the healing professions as to what constitutes valid treatment and patient improvement.

Does Drug Treatment Generalize?

Wakefield had touted "…the impressive results of modern psychotropic medications…the fact is that it was chlorpromazine, not token economies, that emptied the asylums…" (Wakefield, 2006). Was Wakefield unaware of the relatively high numbers of patients whose treatment with anti-psychotic medication fails to generalize? A *New England Journal of Medicine* study revealed the staggering percentages of patients who refuse to continue taking anti-psychotic medications. By 18 months, 64%-82% of patients taking Trilafon, Zyprexa, Seroquel, Risperdal or Geodon had refused to continue those medications (Leiberman, Stroup, et. al, 2005). We presented that fact (Wyatt & Midkiff, 2006b), but in reply Wakefield discounted the *NEJM* report because it "…concern(ed) high dropout rates rather than lack of generalization." That was similar to Wakefield's effort to discount the fact that many highly effective treatments are transitory. A trend had become evident—Wakefield, when confronted with inconvenient realities, tends to exclaim, in essence, "That's different!"

PSYCHOTIC BEHAVIOR MAINTAINED BY ITS CONSEQUENCES

In misrepresenting our position as an effort to prove behaviorism, Wakefield had dismissed the notion that environmental variables may be causal for schizophrenia. He had written that we possessed "…blind faith…in the existence of…unknown reinforcers that somehow manage to maintain seemingly painful and heavily punished behavior…" (Note his use of the term "maintain." We will comment on that terminology below.)

Wakefield went on to ask, "Is it really possible that the reinforcers (may maintain maladaptive behavior) despite the heavy toll in suffering and social stigma and disapproval that psychotic symptoms usually incur…?" (Wakefield, 2006, p. 209).

The answer, as we then pointed out, is yes. We gave numerous examples in which maladaptive, stigmatizing, even painful behaviors are nonetheless maintained by other reinforcers: Obesity may be maintained by fast food ads and the satisfying taste of food; smoking by the removal of nicotine withdrawal; intermittent explosive disorder by getting one's way; compulsive rituals by a reduction in anxiety; irrational fear by removal or avoidance of anxiety; psychotic behavior by withdrawal of demands, or receipt of kindly attention or even a doctor's leading question, "Are you hearing voices today?" (Wyatt & Midkiff, 2006b).

These are common examples, yet Wakefield seemed unaware, not mentioning such contingencies in his initial paper, then dismissing them once we had pointed them out in our second paper (Wyatt & Midkiff, 2006b). Wakefield now continues to rely upon the "That's different!" fallback position, saying that "…analogizing from one [mental disorder] to another is not a credible scientific argument" (Wakefield, this issue, p. 184). Perhaps professor Wakefield has considered few reinforcers beyond social approval, or perhaps he considers approval to trump all other potential reinforcers, all the time. If that is his view, it is inaccurate.

Also, in his first paper Wakefield expressed doubt that reinforcers could *maintain* stigmatizing behavior. We spoke to that by giving the numerous examples of maintenance that appear two paragraphs above. Wakefield now pouts that we had spoken *only* to maintenance, rather than to initiation of severely maladaptive behavior. For Wakefield to grouse that our response to the specific issue he had raised (maintenance of behavior) did not also speak to a related issue (initiation of behavior), was less than germane. We will add only that it is erroneous to assume, as Wakefield seems to do, that maintaining variables may not also be initiating variables. They may be, or may not be, depending upon the individual case. We will not belabor the point here.

IS THERE MORE TO BEHAVIORAL APPROACHES THAN THE HOSPITAL TOKEN ECONOMY?

The answer to the above question is, of course, yes. Wakefield's evident notion that difficulty in generalization of a single treatment for a single disorder (token economy for schizophrenics) somehow disproves or greatly undercuts behaviorism, is erroneous. Although he made at least brief mention of additional disorders and additional treatments, we believe that a fair reading of his articles demonstrates that our characterization is accurate.

In contrast, the philosophy of behaviorism, its extension to the laboratory known as the experimental analysis of behavior, and its panoply of uses to improve the human condition known as applied behavior analysis, are broad and growing disciplines. Here is a partial list of applied topics from the program booklet of the Association for Behavior Analysis, which is clearly the most behaviorally oriented professional organization in the

world: Abused children, academic difficulties, aggression, aggression with the chronically mentally ill, alcohol abuse, anxiety disorders, autism, brain injury rehabilitation, bulimia, failure to thrive, feelings, food refusal, inappropriate sexual behavior, language, obsessive compulsive disorder, pain management, pediatric sleep disturbance, perseverative speech, phobia, public speaking anxiety, self injurious behavior, stuttering, trichotillomania, and weight loss.

The evident breadth of problem behaviors being addressed by behavior analysts goes far beyond Wakefield's tunnel vision focus upon token economy for schizophrenics. Though Wakefield seems unaware, the above list is not news. It comes from the conference program booklet of ten years ago, 1997. The 2007 program is even more extensive. New applications of behavioral principles to areas such as corrections (prisons), workplace and roadway safety and more have arisen in recent years. Wakefield's focus on the problem of generalization among discharged schizophrenics fails in two ways—it does not disprove behaviorism, and it does not adequately address our contentions that both biological causation and claims of drug effectiveness are oversold.

UNIVERSALITY OF BOTH BIZARRE BEHAVIORS AND REINFORCERS

Wakefield holds that "bizarreness and cross-environmental persistence of the (schizophrenic) symptoms…and the frequent relatively sudden onset of symptoms…" provide evidence of biological causation. We agree that such an interpretation is possible, but so is an environmental account, mainly because primary reinforcers are also identical across environments and cultures, as are many secondary reinforcers. Moreover, the typical "sudden onset"of schizophrenia in late adolescence is at the time that environmental stressors involved in achieving independent functioning are greatest. As a result, it is quite plausible there might occur a collapse of functioning in those with poorly developed coping skills, or in those whose stressors accumulate far beyond the norm. In our view it is narrow-minded to assume that such a breakdown in functioning could not occur unless biologically causal variables were involved. One need only consider disorders of psychotic proportions that, by definition, are brought on environmentally, in order to understand the fallacy in Professor Wakefield's position. Post-traumatic stress disorder and brief reactive psychosis come to mind.

Is Schizophrenia Rightly Termed a Disease?

Related to the above, it is legitimate to ask whether schizophrenia is a single entity that can rightly be termed a biological disease. Wakefield treats it as such, despite the fact that the reliability of its diagnosis is well below 100 percent, and despite the fact that there is no biological test for it (no blood test, etc.), and despite the fact that it is defined solely in terms of what people do (hallucinate, exhibit context-irrelevant talk, wear several layers of clothing in hot weather, etc.). We modestly suggest that Professor Wakefield consider these facts.

Is Popularity of a View Evidence that it is Correct?

Wakefield had argued, weakly we feel, that popularity of the biological view tends to prove it (Wakefield, 2006). That was self-contradictory, given that he also stated that at one time the behavioral model was the most popular. We had replied that belief in UFOs is presently common, as once was belief in a flat earth (Wyatt & Midkiff, 2006b). Now Wakefield (this issue) has modified his "popularity" argument, adding that because schizophrenia is bizarre, persists across cultures, persists when the patient moves to a different environment and has a seemingly sudden onset, our UFO and flat earth scenarios are not apt. However, we ask Professor Wakefield whether belief in UFOs is not also bizarre, given that their existence has never been proved? We point out that belief in UFOs persists when a believer changes environments, and it is found cross-culturally. Also, for most UFO fanciers it probably begins in late adolescence. Thus, when Wakefield wrote, "There is nothing analogous in this situation (popularity of belief in biological causation of schizophrenia) to the belief in UFOs," it is difficult for us to imagine that he could have been more wrong. Additionally, we humbly and respectfully suggest that it is time that Professor Wakefield retire his "that's different" reaction to disquieting data.

Low Blows Are Not Evidence

When pressed, Wakefield resorts to what some may consider low blows. Regarding our position that environments may account for schizophrenic behavior, near the end of his current paper he characterizes our position as "…disturbingly similar to that of many in the general public who don't want to admit that mental illnesses really exist in a child, and who blame the symptoms on character defects instead." He adds that our position is "quite similar to that of John Hinckley's father who withheld financial support of Hinckley just prior to his son's assassination attempt on Ronald Reagan" (Wakefield, this issue). In contrast, we prefer to keep our train on the rails. As well, we remind the reader that Wakefield thinks it is *we* who have produced weak analogies.

ON TERMS

Wakefield has demonstrated an affinity for the term "pseudoscience" in describing behaviorism. One gets the feeling that Professor Wakefield is convinced that if he repeats that term frequently enough, it will be true. He used it in the titles of each of his papers in this exchange, and at least a half-dozen times in the body of his paper in this issue.

In contrast, we documented with data psychiatry's flight from science, such as by tracing the history of that movement, by relying on psychiatry's failure to adduce scientific evidence when it was challenged to do so, by demonstrating significant weaknesses in drug studies and by articulating the hand-in-glove relationship between the pharmaceutical industry and psychiatry.

Although it was not our intention to prove the validity of behaviorism, or to demonstrate its applications to be useful in the real world, we appreciate Wakefield's

strenuous efforts to disprove it, mainly by hitching his wagon to the narrow generalization issue.

Wakefield's tendency to claim, in essence, that each point we raised was irrelevant, or a poor analogy, or in some other way did not suit him, is transparent enough to the reasonably sophisticated reader. Thus, there is little need to say more here on that topic, with two exceptions.

First, perhaps Wakefield's essential misunderstandings are evident in, or at least are exemplified by, his misuse of the term behaviorism itself. His titles ask, "Is behaviorism becoming a pseudoscience?" As questions go, that is a non sequitur. Behaviorism makes no pretense to be a science. Rather, behaviorism is a philosophy that underlies sciences known as the experimental analysis of behavior and applied behavior analysis (Skinner, 1974). Because it is a philosophy, behaviorism isn't properly thought of as a science, or as a science wannabe.

Finally, Professor Wakefield (Wakefield, this issue) made no effort to account for a strange conclusion he had earlier reached relative to our first article in this exchange. He had pointed the finger of accusation at us because, as he put it, we had, "cite(d) politics and other unscientific considerations in defending our position…" (Wakefield, 2006, p. 219). However, that had been our point exactly: Non-scientific factors (lost esteem for the profession of psychiatry, "intrusion" by non-psychiatrists, profit motives of the pharmaceutical industry) have been at the heart of psychiatry's, and the culture's, turn to biological explanations over the past 35 years. If Wakefield is accusing us of doing exactly what we set out to do, we shall not complain.

When it all falls away, our view is that Wakefield has produced a strenuous effort that failed. He neither undid behaviorism (even though proving it true was not our purpose) nor successfully defended the current overreaching biological causation model.

We have felt, and continue to feel, a sense of validation for our central theses—that biological causation of most common disorders such as unipolar depression, anxiety disorders, child conduct problems, ADHD and schizophrenia is weak to non-existent, and that claims of psychotropic drugs' effectiveness are routinely overstated.

AN UPDATE

We offer an update on several of the kinds of non-scientific factors that reveal the ongoing overreach of the biological causation and biological (drug) treatment zeitgeists in the United States today. Individually, these phenomena might be relatively meaningless. Taken together they add to the growing perception that all is not well.

On April 26, 2007, the *New England Journal of Medicine* published an article titled, "Paying for drug approvals—who's using whom?" (Avorn, 2007). In it, the author pointed out that, "Since 1992, the United States has relied heavily on the pharmaceutical industry to pay the salaries of Food and Drug Administration (FDA) scientists who review new drug applications." In the early 1990s, an era in which efforts were constantly underway to reduce the size of government, it seemed sensible to develop a plan under which drug companies would pay the salaries of FDA employees who reviewed the

companies' submissions. These user fees, the *NEJM* article stated, now account for 40 percent of the budget of the FDA division that reviews new drug applications. This has contributed to the notion that the FDA views the drug makers as its primary clients, a perspective reiterated by FDA whistleblower David Graham in testimony to Congress in February, 2007.

Drug industry spending on lobbying continues at astonishing rates. The Center for Public Integrity released its annual report on drug industry lobbying on April 1, 2007. In 8.5 years, from 1998 to June 30, 2006, the top twenty (in sales) drug makers spent $733 million on lobbying and another $71 million in campaign contributions. However, in just one of those years the same companies' sales were 200 times greater than that total, with balance sheets showing $195 billion in sales in 2005 alone (Ismail & Bengtsson, 2007).

In October, 2006, former FDA chief Lester Crawford pled guilty to charges of conflict of interest and false reporting. Crawford had kept secret his stocks in food, beverage and medical device companies he regulated while head of the FDA (Ex-FDA Chief, 2007).

In August, 2006, the editor of *Neuropsychopharmacology*, Charles B. Nemeroff, resigned because he had published a paper without disclosing that 8 of its 9 authors—including himself—had financial ties to Cyperonics, Inc., maker of the vagus nerve stimulation (VNS) device, which the article had favorably reviewed. The device has been used to treat seizures, and approval had been sought for its use in treatment of depression. Permission ultimately was denied by the FDA (Pincock, 2006).

In November, 2006, the Associated Press reported that increasing numbers of medical schools are requiring classes in which "Medical students in several states are taught to challenge (drug) sales pitches and avoid being dazzled by them" (Caruso, 2006).

In December, 2006, the *New York Times* reported that hundreds of internal documents and e-mails among top Eli Lilly Company managers revealed how they coached sales representatives to understate their anti-psychotic drug Zyprexa's links to obesity, high blood pressure and diabetes (Berenson, 2006).

In February, 2007, the FDA directed manufacturers of all drugs for which it had earlier given approval for treatment of ADHD to develop patient Medication Guides to warn patients about the drugs' possible links to psychotic behavior and cardiovascular risks, including the possibility of sudden death (Patient Medication Guides, 2007).

In May, 2007, the FDA proposed changes to the warning labels of antidepressants to include adults aged 18-24 among the populations with heightened risks of suicidal thoughts and behavior during the first month or two of treatment. The language had previously applied only to children and adolescents (U. S. drug agency, 2007).

On June 23, 2007, the American Medical Association's annual policy meeting began in Chicago. It included a report that recommended that video game addiction be officially classified as a psychiatric disorder "to raise awareness and enable sufferers to get insurance coverage for treatment." Clearly, some individuals play video games compulsively, even to the extent that they fail to accomplish important life tasks, and otherwise suffer stigmatizing, punishing consequences. However, rather than medicalize

a maladaptive habit, a more parsimonious explanation is that enjoyment of the games, coupled with avoidance of less enjoyable activities that playing them brings about, likely are at the heart of the problem.

Where Are the Data?

The above events are not presented as scientific evidence. They are, however, indicative of the kinds of phenomena that one would predict to occur within a culture in which the drug industry's influence has gone too far.

It is our view that the pharmaceutical industry, along with the FDA that is supposed to oversee it, as well as the medical and psychiatric professions as they relate to the drug industry, are in need of reform. A recent article in the *New England Journal of Medicine* (Campbell, Gruen, Mountford, Miller, Cleary & Blumenthal, 2007) showed that, in a survey of more than three thousand physicians, 94 percent of respondents had some form of relationship with the drug industry. Thirty-five percent of respondents reported receiving reimbursement for costs associated with professional meetings, and more than one quarter received payments for consulting, giving lectures or enrolling patients in drug trials. Eighty-three percent reported receiving food in the workplace.

Meanwhile, non-physician mental health practioners continue to "intrude" upon what was once medicine's exclusive turf. For example, psychologists consistently have gained ground in terms of insurance reimbursement, hospital privileges and prescription privileges, while psychiatry has resisted the competing professions at every step. It is in this atmosphere that the twin interests of psychiatry and the drug industry have come together in the past thirty-five years with the result that patients are being shortchanged. Those seeking treatment are cavalierly told that their depression is due to a chemical imbalance, in the absence of evidence to that effect, or they are told that the conduct problems of their children are due to genetic or other biological problems, absent any evidence to that effect.

Some, such as Professor Wakefield, likely are not open to the possibility that a wrong turn has been taken. Nor will they agree that that this wrong turn has been undergirded at each step by oversubscription to the biological causation model of mental and behavioral disorders. However, more open-minded individuals, upon hearing yet another claim that one's biology is the cause of depression, anxiety, a "video game addiction" or another behavioral or mental disorder, understand that it is always appropriate to ask, "Where are the data?"

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