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RESPONSES TO COMMENTARIES

SIX-TO-ONE GETS THE JOB DONE: COMMENTS ON THE REVIEWS

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We are pleased to note that six of the seven responses to our article were marked by approval, and/or thoughtful contemplation, regarding our central theses—that the research said to support biological causation of mental disorders is relatively weak, and that the claims of drug effectiveness are often overstated.

Richard F. Rakos (2006) put forth an interesting hypothesis—a belief in agency (free will) may be genetically determined because it has survival value. A sense of autonomy may function as a powerful primary reinforcer. As Rakos put it, this belief seems unshaken by logic or evidence, an observation with which it is difficult to disagree.

On reflection, however, one must account for the fact that humans are quite often eager to abandon agency, such as when they engage in inappropriate, illegal, unethical or otherwise maladaptive behavior. On such occasions humans are often quick to blame external factors (“The dog ate my homework”), rather than themselves. Perhaps what is born into us is a gene for adoption of any position that is self-serving.

Rakos has thoughtfully analyzed the human tendency to attribute free will in doses that correspond to functional level. The developmentally disabled are seen as more appropriate targets for behavioral techniques because they are seen as possessing less free will than those who function at a higher level.

Rakos takes note of the powerful influence of the pharmaceutical industry and asks what is to become of behavior analysis in a society that is moving closer to *Brave New World* than to *Walden Two*. He suggests that the nonscientific manner in which syndromes are selected for inclusion in the *DSM* is not widely known, and adds that now may be a good time for behavior analysts to work toward a new system of diagnostic classification based upon functionally-defined problems. We agree, but remain somewhat pessimistic due to the enormous power of the psychiatry and pharmaceutical industries that likely will strongly resist such a move. At any rate Rakos recognizes that we have presented, “...compelling reasons for behavior analysts to incorporate political activity into our role definition.”

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Kurt Salzinger (2006) eloquently describes the state hospital of years gone by. We both have worked full-time in a state hospital—the first author as far back as the 1970s. Indeed, many changes have taken place. Like Rakos, Salzinger puts forth an interesting hypothesis—in this case about a possible inborn disposition of schizophrenics to respond to stimuli preponderantly in the immediate environment, to the exclusion of stimuli that are more removed. We see no reason why that could not be the case, and the hypothesis awaits confirmation. We tend to disagree, however, with Professor Salzinger’s assertion that evidence for the biological model is much more compelling today than it has been in the past. He cites the genome project and the, “...possibility of real breakthroughs...” Certainly the possibility is there, but up to now the breakthroughs appear to be less the products of the scientific enterprise than the culmination of the economic interests of the medical and pharmaceutical industries.

It is suggested by Salzinger that we should view the biological model as a friend that will make our procedures more powerful. Upon reading that we were put in mind of Skinner’s frequently stated desire to see behavior analysis become a part of the science of biology—a thoroughly natural science. In that context we agree wholeheartedly with Salzinger.

Christopher G. Mitchell (2006) felt challenged to review the acceptance of biological causation that he had acquired over the years. Thus, we have succeeded in one of our goals. We have created a dialogue. Interestingly, in contrast to Professor Rakos who considers that the biological model imbues the individual with a heightened sense of autonomy, Dr. Mitchell holds the opposite view, that the biological model disempowers patients in their efforts to change. We lean toward Mitchell’s position, but recognize the intriguing and thoughtful discussion made by Rakos, as well.

Mitchell focused at some length on the adverse side effects of psychotropic medications. Indeed, these are significant, and at times underplayed in drug company advertisements. We agree with Mitchell’s conclusion that vast sums of money and battles for territorial dominance leave our patients with the most to lose.

Judy Blumenthal (2006) nicely reviews the historic efforts to understand behavior, including supernatural “explanations.” This is refreshing. We find it a healthy exercise when, upon learning of some new causal hypothesis, to ask whether it is substantially different from the witchcraft, zombies or multiple personalities to which Blumenthal pointed. Too often the answer is no, although slick drug company advertisements, as well as organized psychiatry’s position statements, frequently possess the language and trappings of science and, as a result, have accrued greater face validity than have explanations which invoke witches and zombies.

As Blumenthal cogently points out, drug companies are adept at using knowledge of behavior to change it. The public is indeed bombarded by advertising about the effects of medications, so much so that at times the people may be convinced that, for example, school children are incapable of functioning properly without drugs.

We are encouraged to read Blumenthal’s endorsement of the notion that behavior analysts must pass on their knowledge to the lay public. It is unfortunate that, with exceptions of treatment of the developmentally disabled and a few other disorders, the

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public knows little about the field. Finally, Blumenthal presents several studies that have recently been, or easily may be, featured in the media--a note of much needed optimism for behavior analysts.

An interesting point on the usefulness of genetic explanations is provided by Meredith Hanson (2006) who points out that much of what is considered “biological” in social science research is actually a social construction. An example is the construct of race/ethnicity which Hanson describes as “a cultural construct masking as biology.” It may well be, as he points out, that an “environmental fit” best explains much behavior earlier thought to be genetically determined. For example, the significance of skin color tends to recede as a consequence of social interaction, Hanson notes. In rats, social isolation creates heightened stress responses and probability of tumors. The thesis recalls the brain’s plasticity and that experience changes neuroanatomy.

Hanson also points out the limitations of medications. He cites a recent study showing that large numbers of patients simply quit taking medications, which suggests that without accompanying psychosocial interventions, medication alone only weakly serves to make life better for the majority of patients.

Dwight Harshbarger (2006) describes the parade of drug commercials that confront anyone who views the nightly news (or almost any other hour of television, we might add). Additionally, he notes the methodological inadequacies that render relatively meaningless the findings of both twin studies and studies of brain structure and function. Unlike the final reviewer (see below), Harshbarger understands that suspect research practices, unwarranted underlying assumptions and overreaching interpretations (all of which we described) have often gone unchallenged, and subsequently are lost, plowed under in “the flood of dollars poured into drug marketing.”

Regarding that, he remarks on the failure of the scientific community to take a hard look at the economic interests that bend scientific findings for private gain. We are heartened to find that, like Harshbarger, most of our reviewers found some degree of validity in our central theses. On a hopeful note, Harshbarger advises us that the “behavioral blues” is not a permanent condition.

THE DISSENTER

In deciding how to respond to Professor Jerome C. Wakefield’s (2006) review we were put in mind of efforts to latch onto a greased pig. The creature’s slippery condition, combined with its tendency to dart hither and yon, render it nearly impossible to know which part to grab first. However, we will address, in turn, a number of issues that were raised by Professor Wakefield. Also we will point out instances in which his lengthy review conveniently turned a blind eye and a deaf ear to our challenges to biological causation theory.

Were one to read Wakefield’s review without having read our article, one might well conclude that we had failed to provide evidence for our central contentions—that the burgeoning acceptance (in the professional and popular cultures) of biological causation of mental disorders goes well beyond the data, and that claims of drug effectiveness are at

times overstated. Having considered his efforts to undo our positions on these two issues, we conclude that Professor Wakefield's Herculean efforts have come up quite short.

For example, in contrast to Wakefield's assertions, consider what a number of experts have said in reaction to just one frequently heard pharmaceutical advertising claim—that a serotonin imbalance causes, or contributes to, depression (Lacasse & Leo, 2005):

- “Although it is often stated with great confidence that depressed people have a serotonin or norepinephrine deficiency, the evidence actually contradicts these claims.” Elliot Valenstein (1998), Professor Emeritus of Neuroscience.
- “A serotonin deficiency for depression has not been found.” Joseph Glenmullen (2000), clinical instructor of psychiatry at Harvard Medical School.
- “Some have argued that depression may be due to a deficiency of NE (norepinephrine) or 5-HT (serotonin)...However, this is akin to saying that because a rash on one's arm improves with the use of a steroid cream, the rash must be due to a steroid deficiency.” Psychiatrists Pedro Delgado and Francisco Moreno (2000), in the *Journal of Clinical Psychiatry*.
- “I spent the first several years of my career doing full-time research on brain serotonin metabolism, but I never saw any convincing evidence that any psychiatric disorder, including depression, results from a deficiency of brain serotonin.” David Burns (2003) winner of the A. E. Bennett Award given by the Society for Biological Psychiatry for his research on serotonin metabolism.
- “Indeed, no abnormality of serotonin in depression has ever been demonstrated.” Psychiatrist and former Secretary for the British Association for Psychopharmacology, David Healy (2004).
- “We have hunted for big simple neurochemical solutions for psychiatric disorders and have not found them.” Psychiatrist Kenneth Kendler (2005), coeditor-in-chief of *Psychological Medicine*.

Compare the above experts' statements to drug company advertising for antidepressants:

- “Celexa helps to restore the brain's chemical imbalance...” (Forest Pharmaceuticals, 2005)
- “LEXAPRO appears to work by increasing the available supply of serotonin...In people with depression and anxiety, there is an imbalance of serotonin...” (Forest Pharmaceuticals, 2005).
- “When you're clinically depressed...the level of serotonin...may drop...(T)o help bring serotonin levels closer to normal, the medicine doctors now prescribe most often is Prozac.” (Eli Lilly, 1998)
- “Paxil...works to correct the chemical imbalance believed to cause (generalized anxiety).” (GlaxoSmithKline, 2001)
- “...depression may be related to an imbalance of natural chemicals...Zoloft works to correct this...” (Pfizer, 2004)

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The gap between the researchers' conclusions and drug company advertisements is awesome. We stick to our contention that drug firms' claims of biological causation have routinely gone well beyond the data. At the polar opposite, Professor Wakefield has written, "...I think by and large (Wyatt and Midkiff's) contention is a myth..."

Of equal or greater interest is Wakefield's gaping failure to address sections of our article that tend to disconfirm his pro-biological causation position. The most revealing example? In his review, the length of which approximated that of our paper, 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.

Wakefield also failed to address a number of other inconvenient facts. We cited research (Kirsch, Moore & Scoboria, 2002) showing that many anti-depressant medications are less effective than one might suppose. With an opportunity to comment, Wakefield took a pass. Similarly we described psychiatry's ongoing efforts to rebuff intruder professions, and how the major arrow in psychiatry's quiver became the profession's turn to biological causation. Again, Wakefield chose to leave that thorny matter alone.

We documented the influence of drug company marketing upon doctors' prescribing practices (Kravitz, Epstein, Feldman, Franz, Azari, Wilks, Hinton & Franks, 2005), but again Professor Wakefield seemed more interested in defending his point of view than in accounting for the irritating facts. Likewise, we provided evidence for the enormous increase in drug company marketing jobs since 1995 when direct-to-consumer advertising became legal, and the parallel increase in prescribing of psychotropic medications (Antonuccio, Danton & McClanahan, 2003). Again, rather than addressing the facts, Wakefield chose silence.

We described methodological and interpretative problems that essentially render the ubiquitously cited studies of identical twins (reared apart) of little value in establishing genetic contributions to mental disorders. Evidently Wakefield agrees with us because he counts those studies as, "...of limited value in the overall scheme of things..." Then, to bolster his position, he cites an in-press article (Keller & Miller) that similarly fails to account for the issues we raised. What Wakefield missed completely is that the evidence he prefers (e.g., family studies not involving identical twins) suffers from the environmental-genetic confounds that themselves gave rise to studies of monozygotic twins reared apart.

Wakefield was dismissive of the data we presented to show that identical twins, even if raised apart, will be treated rather similarly (in ways shown to contribute to mental health) because of their identical physical attractiveness and identical rates of developmental maturation. Likewise we presented evidence to show that both adoption agencies and families frequently make special efforts to place adoptive children in homes quite similar to their biological homes. These are variables that render the environments of separated monozygotic twins quite similar. Despite our citation of a number of studies, in a paroxysm of denial Wakefield wrote that we had offered, "...no evidence for (our)

speculation...” that identical twins grow up in rather similar environments even though they are reared apart.

There are other interesting aspects to Wakefield’s review. First is his focus upon schizophrenia. Although we indeed mentioned schizophrenia, it wasn’t our focus, as we addressed disorders more generally. However, upon reading his review one would get the impression that we focused exclusively upon a single disorder.

Aside from the above aspects of Wakefield’s review, his special focus was upon the (lack of) generalization of effectiveness of behavioral treatment, especially in studies of schizophrenics. 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.

There are other problems with Professor Wakefield’s efforts to defend both biological causation and biological treatment by pointing to the issue of generalization of behavioral treatment. 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.

Beyond that is Wakefield’s evident blind faith in psychotropic drugs, and the fissure in his understanding of the causes of the reduction in mental hospital populations over the past forty years. Wakefield reports with animation on “...the impressive results of modern psychotropic medications...the fact is that it was chlorpromazine, not token economies, that emptied the asylums...” There is truth to this. But there are two problems with his analysis. First, “empty asylums” does not equal patients cured. Witness the numbers of homeless former state hospital patients who wander the streets muttering to themselves, or who now shuffle through the halls of group homes, little better in their functioning than when in the “asylum.” Those poor souls are hardly good advertisements for effectiveness of psychotropic medications.

Second, Wakefield seems unaware of the numerous factors other than medications that have contributed to the vast reduction in numbers of hospitalized patients. Could Wakefield be unaware that in the past forty years state after state has enacted tough commitment laws that mandate probable cause hearings and routine reviews of patient progress? Has he not learned of the availability of Social Security disability benefits which enable many former “asylum” patients to qualify for placement in group homes and supported living apartments? Does he not know of the rise of the community mental health movement of the past forty years, and the fact that patients now have much greater access to outpatient treatment? Could Wakefield not know of legislation nationwide that has *mandated* deinstitutionalization of most patients? We have both worked full-time in a large state hospital and have observed the impact of these factors up close. Wakefield did not trouble himself to account for them, even though they have contributed as much, or

more, than medications, when it comes to the reduction in numbers of hospitalized patients.

Nor did Professor Wakefield account for another troubling fact—the absence of generalization of drug treatment. An enormous number of former hospital patients simply quit taking their medications. Usually they do so because of uncomfortable side effects. A recent study published in the *New England Journal of Medicine* revealed the percentage of patients who had quit taking their anti-psychotic medication by eighteen months. The startling findings are: Trilafon—75% stopped taking the medication; Zyprexa—64%; Seroquel—82%; Risperdal—74%; and Geodon—79% (Leiberman, Stroup, McEvoy, Swartz, Rosenheck, Perkins, Keefe, Davis, Davis, Lebowitz, Severe & Hsiao, 2005). In responding to our article, Professor Wakefield hitched his thesis to problems in generalization of behavioral treatment. In fairness, he might have considered the same issue with drug treatment, but he did not.

Clearly, Wakefield's tangential attack on behavioral treatment does not qualify as an undoing of our critique of the biological causation model. However, his response does reveal Wakefield's failure to understand essential behavioral principles. He is not alone. Misrepresentations and misunderstandings of behavioral principles, techniques and philosophy are common (Wyatt, Lamal, Newman & Hobbie, 1997). For example, he accuses us of possessing, "...blind faith...in the existence of ...unknown reinforcers that somehow manage to maintain seemingly painful and heavily punished behavior..." Similarly, elsewhere he asks, "Is it really possible that the reinforcers (are able to maintain maladaptive behavior) despite the heavy toll in suffering and social stigma and disapproval that psychotic symptoms usually incur...? The simple answer to Wakefield's wide-eyed question is yes. In fact, 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. 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.

Several times in his review Wakefield accuses us of possessing blind faith in our position. But when we critiqued the "unbalanced neurotransmitter" theory of depression, Wakefield responded weakly saying, "...that does not mean that schizophrenia and major depression do not involve other biological problems." We agree that such is possible.

However, Wakefield then provides no evidence or other indication about what those hypothetical other phenomena might be. Additionally he describes, "...the impressive results of modern psychotropic medications..." but seems unimpressed with the growing number of studies suggesting that much, though not all, of the effects of medications are placebo related. For example, when data from the published and unpublished trials of SSRI anti-depressants were pooled, the placebo was found to have accounted for about 80% as much improvement as the anti-depressant (Kirsch, Moore & Scoboria, 2002).

It gets worse as Wakefield attempts to prove his case with statements such as, "the resurgence of the biological view is based on traditional prima facie inferences about biologically designed normal human capacities across expectable environments, plus persuasive contextual evidence (i.e., the kind we commonsensically use when judging that blindness or paralysis is a disorder even when we know nothing about physiology of the eye or musculature), plus a failure of all suggested alternative theories...to reach a minimal threshold of plausibility. In particular, the relative independence of psychotic symptoms from environmental change and intervention suggest(sic) an internal cause..."

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.

Professor Wakefield's reliance upon blind faith is evident elsewhere as he maintains, "...most observers through history have been persuaded of the biological position." That is not evidence. Does Professor Wakefield believe in UFOs? Most Americans are convinced of their existence. Likewise, at one time most observers were convinced that the earth was flat. Popularity of a point of view is not evidence of its validity, as Wakefield would have us believe.

Wakefield accuses us of, "...abandoning parity of reasoning..." We reject that assessment. We have adduced a body of evidence to support our central theses that were stated at the beginning of our article: The research in support of biological causation is weaker than one would expect given its increased acceptance over the past three decades, and there are reasons to think that the claims of drug effectiveness are overstated, especially by the pharmaceutical industry.

Wakefield's preference for pejorative language ("pseudoscientific," "verbal gymnastics") to describe our efforts seem weak and leaden methods by which to critique. His style and absent substance remind one of the political conservative's misguided efforts to deny the existence of global warming.

His concluding section was revealing in another way. It contained his charge that we, "...cite politics and other unscientific considerations in defending (our) position..." But that was our point exactly. Politics, psychiatry's war against "intruder professions," and the financial interests of the drug industry—all are non-science factors that have contributed to the resurgence of the biological causation model over the past several

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decades. We have elucidated the inconvenient facts, and in response Professor Wakefield seems to cry, “No fair.”

REFERENCES

- Antonuccio, D. O., Danton, W. G. & McClanahan, T. M. (2003). Psychology in the prescription era: Building a firewall between marketing and science. *American Psychologist*, 58, 1028-1043. <http://dx.doi.org/10.1037/0003-066X.58.12.1028>
- Blumenthal, J. (2006). The treatment of behavior: Drugs or behavior analysis. *Behavior and Social Issues*, 15, 196-198. <http://dx.doi.org/10.5210/bsi.v15i2.364>
- Delgado, P. & Moreno, F. (2000). Role of norepinephrine in depression. *Journal of Clinical Psychiatry*, 61, 5-11.
- Hanson, M. (2006). Commentary on Wong, Wyatt and Midkiff. *Behavior and Social Issues*, 15, 178-180. <http://dx.doi.org/10.5210/bsi.v15i2.371>
- Harshbarger, D. (2006). The evening news and the behavioral blues: Comments on Joseph Wyatt and Donna Midkiff's *Biological Psychiatry: A Practice in Search of a Science*, and Stephen Wong's *Behavior Analysis of Psychotic Disorders: Scientific Dead End or Casualty of the Mental Health Political Economy?* *Behavior and Social Issues*, 15, 199-201. <http://dx.doi.org/10.5210/bsi.v15i2.392>
- Eli Lilly (1998 January). Prozac advertisement. *People Magazine*, p. 40.
- Forest Pharmaceuticals (2005). Frequently asked questions. New York: Forest Pharmaceuticals. Retrieved December 1, 2005 from <http://www.celexa.com/elexa/faq.aspx>.
- Forest Pharmaceuticals (2005). How Lexapro (escitalopram) works. New York: Forest Pharmaceuticals. Retrieved December 1, 2005 from http://www.lexapro.com/english/about_lexapro/how_works.aspx.
- GlaxoSmithKline (2001, October). Paxil advertisement. *Newsweek*, p. 61.
- Glenmullen, J. (2001). Prozac Backlash: Overcoming the dangers of Prozac, Zoloft, Paxil and other antidepressants with safe, effective alternatives. New York: Simon and Schuster.
- Healy, D. (2004). Let them eat Prozac: The unhealthy relationship between the pharmaceutical companies and depression. New York: New York University.
- Kirsch, I., Moore, T. J. & Scoboria, A. (2002). The emperor's new drugs: An analysis of antidepressant medication data submitted to the U. S. Food and Drug Administration. *Prevention & Treatment*, 5, Article 23. Retrieved July 26, 2004, from <http://journals.apa.org/prevention/volum5/toc-jul15-02.html>.
- Kramer, P. (2002, July 7). Fighting the darkness in the mind. *The New York Times*, Section 4, p. 8.

- Kravitz, R. L., Epstein, R. M., Feldman, M.D., Franz, C. E., Azari, R., Wilks, M. S., Hinton, L. & Franks, P. (2005). Influence of patients' requests for direct-to-consumer advertised antidepressants. *Journal of the American Medical Association*, 293, 1995-2002. <http://dx.doi.org/10.1001/jama.293.16.1995>
- Lacasse, J. R. & Gomory, T. (2003). Is graduate social work education promoting a critical approach to mental health practice? *Journal of Social Work Education*, 39, 383-408.
- Leiberman, J. A., Stroup, T. S., McEvoy, J., Swartz, M., Rosenheck, R. A., Perkins, D. O., Keefe, R. S. E., Davis, S. M., Davis, C. E., Lebowitz, B. D., Severe, J. & Hsiao, J. K. (2005). Effectiveness of antipsychotic drugs in patients with chronic schizophrenia. *New England Journal of Medicine*, 353, 1209-1223.
- Mitchell, C. G. (2006). Response to Wong, Wyatt and Midkiff. *Behavior and Social Issues*, 15, 181-184. <http://dx.doi.org/10.5210/bsi.v15i2.368>
- Pfizer (2004 March). Zoloft advertisement. Burbank (California): NBC.
- Rakos, R. F. (2006). Applied behavior analysis: Niche therapy par excellence. *Behavior and Social Issues*, 15, 185-191. <http://dx.doi.org/10.5210/bsi.v15i2.367>
- Salzinger, K. (2006). Behavior analysis in the real world. *Behavior and Social Issues*, 15, 192-195. <http://dx.doi.org/10.5210/bsi.v15i2.366>
- Valenstein, E. S. (1998). *Blaming the brain: The truth about drugs and mental health*. New York: Free Press.
- 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. <http://dx.doi.org/10.5210/bsi.v15i2.363>
- Wyatt, W. J., Lamal, P. A., Newman, B. & Hobbie, S. A. (1997). Treatment of behavior analysis in five leading introductory psychology textbooks. Monograph published by BALANCE, a Special Interest Group of the Association for Behavior Analysis International.