COMMENTARY

Integrating Psychotherapy and Pharmacotherapy: Myths and the Missing Link

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ABSTRACT. A critical review of three articles reveals flawed empirical evidence underpinning the case for integrating pharmacotherapy and psychotherapy. Medical model dominance favors biology in a diathesis/stress framework, creating myths of valid diagnosis, underlying biological causes, and targeted pharmacological treatments. Meanwhile, a for-profit pharmaceutical industry influences clinical trials, constructing an illusory justification for medical intervention and bolstering the integration hypothesis. The apparent logic of integration threatens to diminish the crucial, empirically supported role of clients in psychotherapy outcome. The authors call for the inclusion of client feedback in intervention choices, based on accurate, unbiased information, and a continued critique of pharmacotherapy. doi:10.1300/J085v17n03_05

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In the past decade, psychotherapy has moved ever more closer toward a medical base. Increasingly, journals, textbooks, workshops, and curricula stress the biological substrata of human distress. Hardly a day goes by without the reported discovery of some new gene or underlying neurological/chemical mechanism responsible for this or that mental disorder, accompanied by the ever-present promises of breakthrough treatments just on the horizon. Explaining the human psyche in terms of neurons and brain chemistry promises to give psychotherapy a long-sought scientific foundation. Biopsychology offers a product that can be seen and touched–image scans and blood tests replace the Rorschach, and treatment no longer resides in the nebulous realm of meaning and interpretation.

Many therapists are gravitating toward this future. To precisely pinpoint an emotion in a computerized picture, or to confidently explain the common problems of depression and anxiety as chemical imbalances, offers a satisfying certainty, for long the sole property of the medical practitioner. At the same time, membership in the medical community perhaps promises a status and financial privilege previously unavailable to psychotherapists.

Other therapists, however, are troubled. They see this direction as diminishing what originally attracted them to the profession. Replacing the uniquely interpersonal phenomena of the therapy relationship with the medical model–diagnosis and pharmacological treatment–seems a less than ideal trade-off. They question the value of membership in a medical community that denotes the qualities of hope and personal connection as mere holding environments for the pharmaceutical cure, or illness-management devices to insure compliance with medication regimens.

This issue of the *Journal of Family Psychotherapy* explores this territory. As a family journal, it asks the question: Can family psychotherapy and pharmacotherapy reside productively under the same roof? What advantages accrue by integrating the two seemingly divergent perspectives to assist those in distress? Does integration offer the best of both worlds?

In contrast to the previous three articles, this commentary challenges the notion that integrating pharmacotherapy and psychotherapy represents a logical or informed direction for our field. We argue that the

push to establish integration as "best practice" contradicts what is known about the effectiveness of pharmacological intervention as well as how people change in psychotherapy.[1] Drawing on critical theory, we also suggest that medical dominance precludes a level-playing field, and that integration further disguises the power dynamics that privilege a medical perspective and diminish the role of therapy.

The three articles reviewed in this issue (Goodman, Vail, West, New, & Siever; McLean, Miller, McLean, Chadkiewicz, & Whittal; Preston) give us ample opportunity to make this case. We examine each in light of its basic assumptions, science, and implications. We argue that that the three articles represent typical presentations of myths in professional press that justify broad, medically based practice parameters. Finally, we recommend the inclusion of the missing link in any proposed integration effort–the client.

THE MYTH OF DIAGNOSIS

The medical model can be summarized by the following: diagnosis + prescriptive treatment = symptom amelioration (Duncan, Miller, & Sparks, 2004; Wampold, 2001). All of the articles reviewed start with diagnosis, the ground floor of this equation. For example, McLean et al. claim in their opening sentence that "anxiety disorders are the most prevalent of all mental disorders . . ." (p. 3). Goodman et al. focus on personality disorders, and Preston deals with depression. From the outset, the validity and utility of DSM diagnoses are unquestioned and function invisibly behind all that follows. But how cavalier can we be about this taken-for-granted assumption?

There are several important ways the medical model equation and its starting point, diagnosis, are ill-suited templates for therapy. From a medical standpoint, the first step in determining what needs to be done is to determine what is wrong. The way to determine what is wrong is to have a clear picture of health. Medicine is able to define those conditions that can be considered optimal or disease-free. For example, physicians know the normal range for glucose levels in blood. They are therefore able to discern deviations and can confidently diagnose diabetes. In mental health, the concept of normalcy is significantly more problematic. Ideas of normal behavior are shaped by social and cultural norms, including arrangements of power, hierarchy, inclusion, and exclusion. Human behavior exhibits a significant range of variation, made even more complex by social systems that either condone or con-

demn difference. Mental health works the equation in reverse–we define deviation, but have considerably more difficulty defining normalcy (Watzlawick, 1976).

Goodman et al. quote the DSM-IV's definition of personality disorder as "an enduring pattern of inner experience and behavior that deviates markedly from the expectation of the individual's culture, is pervasive and inflexible, has an onset in adolescence or early adulthood, is stable over time and leads to distress or impairment." Such definitions beg numerous, unanswered questions–what are the cultural expectations, who defines these, and what role does identification, treatment, and stigmatization play in the perceived stability and inflexibility of the client's behaviors and distress? These questions are not trivial, but address the core politics of any system of classification based on social norms. For example, Goodman et al.'s presumably inadvertent use of a female pronoun in their discussion of "difficulties in emotional regulation" for borderline personality disorder (BPD) exposes the darker side of how diagnosis reinforces gender (and other) stereotypes and maintains the status quo (p. 19).

Second, diagnosis in mental health lacks reliability and validity, cornerstones of any respectable measurement system. In a recent interview, Robert Spitzer, the architect of the DSM, candidly observed: "To say that we've solved the reliability problem is just not true. . . . It's been improved. But if you're in a situation with a general clinician it's certainly not very good. There's still a real problem, and it's not clear how to solve the problem" (Spiegel, 2005, p. 63). The last major study of the DSM, using highly trained clinicians at multiple sites under the supervision of some of the most experienced diagnostic specialists in the world (Williams et al., 1992), found reliability coefficients not much different from studies in the 1950s and 1960s. In fact, Kirk and Kutchins (1992) noted that some reliability coefficients in this study were worse than earlier attempts. When trained clinicians in highly controlled settings cannot agree even on general categories of diagnosis, how much credence can we give to the specific diagnoses ordinary clinicians in busy practices routinely ascribe to their clients?

In addition to questionable reliability, psychiatric diagnosis lacks an even more critical dimension: validity. Here, we ask, does a DSM diagnosis actually represent some defined entity in the real world? Kendell and Zablansky (2003, p. 7), writing in the *American Journal of Psychiatry*, conclude that "At present there is little evidence that most contemporary psychiatric diagnoses are valid, because they are still defined by syndromes that have not been demonstrated to have natural bound-

aries." The authors make the point that psychiatric diagnoses fail the most basic definition of validity–they lack empirical standards to distinguish the hypothesized pathological states from normal human variation to the problems of life. The result is a set of murky over-inclusive criteria for an ever-growing list of disorders (Duncan et al., 2004).[2]

Despite these flaws, most professionals in the field take for granted that the DSM is both reliable and valid and identifies the most salient aspects of a person for resolving complaints. McClean et al., Preston, and Goodman et al. do nothing to disabuse readers of this error. Instead, they speak about diagnostic categories as though they are unquestioned realities. Definitional quotes from the DSM grant legitimacy. We suggest that this reification takes place in virtually every corner of standard mental health practice and in most training curricula. Constant, uncritical repetition in scientific journals and lay press backed by unchallenged science produces an illusion of sound validity, engendering a confidence that far overreaches the DSM's deeply flawed infrastructure.

Third, DSM diagnosis seeks to mimic the objectivity of disease diagnosis. Here the analogy breaks down once again. To presume that disorders can be discerned through a recipe-like set of questions discounts the very rudiments of social science. Humans speak and respond to each other in relationship. This process includes ongoing accommodation between communicators and complex turns of conversation structured by context, social power, and participant's goals and interests. The mental health diagnostic interview and diagnostic assignment are social scripts, imbued with particular roles, rules, and alignments of power deeply engrained in Western culture.

Even more importantly, attributing problems in living or the ranges of human inner experience to individual disorders radically dismisses the essence of what it means to be human. Humans are first and foremost members of social communities, and their behaviors and states of mind are fundamentally connected to and influenced by these contexts. Psychiatric diagnoses represent pathologies that presumably transcend time, place, and culture. For example, rather than viewing the fearfulness of an inner-city child as the product of a specific set of environmental conditions, a diagnostic system may assign noncontext-bound descriptors such as phobia, anxiety, or paranoia. McLean et al. describe the clinical presentation of the "anxious" child. Given a hypothetical scenario of approaching a playground of laughing kids, "The anxious children were more likely to interpret the situation as threatening (e.g., 'the children are laughing at me')" (p. 22). Treatment in this case involves correcting the child's cognitions, challenging the child's self-

talk, and problem solving. The pervasiveness and noncontextual nature of diagnosis locates the problem inside the child and overlooks other possible explanations such as whether the child has been harassed, has been taught to be wary by a parent or sibling, is isolated from support, or is attempting to engage the interviewer in a particular way.

The articles reviewed claim to embrace a contextual, systemic perspective by including the family in treatment. For example, Goodman et al. describe how couple and family therapies facilitate better communication in families where a member is diagnosed with BPD. They further note that family approaches help educate significant others about the "illness," so they can more easily support the "patient" and navigate the interpersonal difficulties the disorder inevitably entails. Following a similar perspective, McLean et al. suggest that family members of those diagnosed with anxiety disorders can be taught not to participate in the diagnosed individual's repetitive, checking behaviors. And, although Preston mentions the success of marital therapy for depression, acknowledging the role of unhappy marriage in depressive symptoms, his review of marital/family therapy for depression does not clarify whether such interventions address the relational basis of the problem or simply help families cope better with the disorder's effects. As a consequence, the take-home message from all three articles is that including the couple/family does not mean challenging the central concept of individual illness, but simply serves as an add-on palliative to treatment of the diagnosed patient.

Finally, diagnosis tells us little about a person that is relevant to the process of change. Diagnosis in mental health is not correlated with outcome or length of stay, and cannot tell clinicians or clients the best approach to resolving a problem (Brown, Dries, & Nace, 1999; Wampold, 2001). The importation of medical diagnosis into psychotherapy positions clients as passive holders of disease to be fixed by the skilled interventions of the clinician. This positioning of clients is particularly unhelpful and flies in the face of what is known about the importance of client factors in psychotherapy. The bulk of outcome research in the past 45 years confirms the critical role clients play in their own change (Asay & Lambert, 1999; Tallman & Bohart, 1999). Research makes abundantly clear that clients are the heroic actors in the drama called therapy (Duncan et al., 2004).

Diagnosis paints a flat and colorless picture that highlights weakness, stigmatizes, and renders invisible vital capabilities and life resources that can be brought to bear in resolving problems. As an example, GAD (generalized anxiety disorder)-diagnosed persons must be convinced of the

inadvisability of worry and uncertainty, regardless of the client's unique circumstances (McLean et al.). While many therapists undoubtedly would want to explore why a person might be worried and anxious, or desire greater certainty, McLean et al. do not mention this line of query as part of standard intervention, once a GAD diagnosis is made. This myopia for individual deficit that diagnosis engenders runs the risk of blinding clinicians to the real and present worries and dangers clients may face in their lives and the validation they might deserve for responding in a reasonable way to these threats. While it is possible that people may overreact to nonthreatening events or imaginings, overreaction may also have a sound basis for the individual given a particular life circumstance or history. At the same time, the determination of overreaction, again, brings into play the position and power of the diagnosing clinician. He or she, by virtue of economic and social advantage, may be ill-informed of the actual struggles that inhabit a client's world. When diagnosis does not dominate the picture, clinicians have greater permission to search with clients for explanations other than illness to the problems in their lives, and to engage in an active pursuit of a broader array of options for alleviating the distress. Rather than constructing a patient in need of correction, we have the possibility of constructing resourceful, active agents deciding how they wish to reorganize their lives and relationships.

In summary, the myth of diagnosis is not benign. Many of the proposed interventions in the three articles may have utility, especially when the client resonates with them (see Duncan & Miller, 2000, for a discussion of the client's theory of change). However, we argue that these interventions when used in conjunction with diagnosis–of questionable reliability, validity, and predictability–regardless of context poses a significant risk of demoralization, diminishment of options, and decreased capacity for clients to harness their best attributes.

THE MYTH OF BIOLOGICAL CAUSALITY

The three articles reviewed suggest that both biology and environment play a role in the expression and treatment of the disorders under investigation. McLean et al. discuss the "relationship between psychological and biological aspects of anxiety" (p. 5). Citing Pauls et al., 1995, they posit that anxiety disorders seem to be genetically mediated, the result of interaction between environmental stimuli and a heightened capacity for "fight or flight" arousal. Goodman et al. follow a similar line of reasoning.

They propose that "abnormalities in cognitive control, affective instability, impulsivity/aggression and anxiety, are biologically mediated . . ." (Abstract). They also highlight the association between personality dimensions and "a variety of neurotransmitter systems" and suggest that "biological susceptibility related to genetic factors (possibly in the neurotransmitter systems, but also likely in a variety of other brain chemicals) are significant correlates of what they call personality disorders (pp. 2-3). Preston states that a "large body of neuroscience research has strongly implicated that dysregulation of certain central neurotransmitters *may* be associated with particular psychiatric symptoms" (p. 9). While he cites a study indicating that most individuals who are deficient in serotonin do not become depressed, he asserts that those who do likely have "underlying genetic or other vulnerability factors" (p. 9).

In each article, a diathesis/stress model underpins the case for combined pharmacotherapy and psychotherapy. There are significant problems with this proposition. First, the association of biological markers with specific states of distress is not a confirmation of biological causality. Valenstein (1998) provides a useful metaphor for this common mistake. He states that interpreting causality when a biological marker is identified with a particular state of mind is like saying, rain is caused by the presence of umbrellas. For example, Goodman et al. begin with the biological *aspects* of BPD and move to the biological *basis* of schizotypal personality disorder (SPD), citing functional images of differences in the gyrus regions during a learning task between controls and SPD-diagnosed persons. Goodman et al. also speculate that affective instability and anxiety all contain likely biological substrates.

Differences in brain activity or processing, of course, say nothing about causality; it is not a biological explanation of "why" but rather a biological "description" of what's happening in the brain of so-called SPD people–that may or may not have any practical relevance. A dual occurrence of events, even *if* found reliably (which is not the case with biological markers of mental disorders), cannot determine the direction of causality. Clearly, the authors of the three articles talk about association, influence, and mediation. Nevertheless, because the brain is commonly thought of as the seat of thinking and emotion, "it is much harder to resist the temptation of elevating its status to that of cause" (Valenstein, p. 130).

Second, deducing causality based on a given treatment's success in alleviating symptoms is indefensible. For example, Preston implies that data from studies showing drug efficacy support the notion of a biological substrate for depression. The logic is that if drugs acting on the

serotonergic system produce the desired effect (the reduction of depression symptoms), then this system must be implicated in the problem. The fact that some people respond to antidepressants with reduction in symptoms explains nothing about causality; at least half of study participants do not respond to the drug in question, and nearly as many people who respond to the drug improve by taking placebos in clinical trials of antidepressants (see the following sections). Following this logic would lead to the conclusion that there are two underlying mechanisms: a serotonin and sugar deficiency! This reasoning is strictly inferential and cannot substitute for specific knowledge of an underlying disease process. Consequently, responses to medication in clinical trials do not support a biological explanation for depressive, anxious, or disordered personality symptoms.

Finally, throughout the pages of serotonergic system deficits, brain activity reduction, paralimbic structures, and sensory processing abnormalities (Goodman et al., this issue), along with Preston's "compelling case for the use of antidepressants," founded on the hyperactive H-P-A axis "seen in many cases of major depression" (p. 7), we have a reinforcement of a biological explanation for human distress based on impressive sounding but very skimpy evidence. What these pages do not say is that *despite 50 years of Herculean efforts, the invention of electron microscopy, the advent of radiolabeling techniques, the revolution of molecular biology, and the merger of computers with neuroimaging machines, no reliable biological marker has ever emerged as the definitive cause of any psychiatric "disease."* What many fail to appreciate is that biochemical imbalances and other so-called functional mind diseases remain the only territory in medicine where diagnoses are permitted without a single confirmatory test of underlying pathology (Duncan et al., 2004).

THE MYTH OF SMART PILLS

When biology is assumed to underlie psychology, medication inevitably follows. For example, Preston's lengthy discussion of neurochemical evidence for depression logically leads to the Major Depressive Disorder Treatment Algorithm, a step-by-step blueprint for prescribing substances that affect brain chemical levels. "Antidepressant medications can reduce cortisol levels and reactivate the production of BDNF, which can lead not only to clinical improvement, but also to the birth of new nerve cells in the hippocampus" (Preston, p. 8). Whether one

challenges the causal link between biology and symptoms or not, the lure of medication's promising effects makes pharmacotherapy, either alone or in combination with psychotherapy, first line treatment for psychiatric problems.

We then need to ask, just how effective are pharmacological treatments? Looking at recent scientific studies, Moncrieff (2001) concluded that despite their increasing use, antidepressants give no indications that they have lessened the burden of depressive experiences. Her observation was partially fueled by an inspection of prevalence rates over a 40-year-period. That analysis showed that rates of depression had not changed since the 1950s. The most recent National Comorbidity Study Replication (Kessler et al., 2005) confirms Moncrieff's assessment that rising antidepressant rates have made hardly a dent in depression prevalence. Antidepressants, Moncrieff reasoned, are overrated. Others have come to the same conclusion, citing that the difference in outcome between antidepressants and placebos is much smaller than the public has generally been led to believe.

Kirsch and Sapirstein (1998) make a persuasive case that side effects and the power of the placebo may account for the lion's share of effects attributed to antidepressants. Their meta-analytic review of 19 studies involving 2,318 people showed that 75% of the response to antidepressants was duplicated by placebo. They speculated that even the remaining 25% of the positive antidepressant effect might turn out to be attributable to the unblinding power of side effects. The review also echoed a point made by others (Fisher & Greenberg, 1997; Moncrieff, Wessely, & Hardy, 1998). Namely, that by using *active* placebos (those that mimic the side effects of the real drug), studies might show the advantage for antidepressants to be quite small or possibly even nonexistent.

The controversy about the benefits of antidepressants heated up even more when Kirsch, Moore, Scoboria, and Nichols (2002) analyzed the efficacy data submitted to the U.S. Food and Drug Administration (FDA) for the six most widely prescribed antidepressants approved between 1987 and 1999. Approximately 82% of the response to medication was duplicated by placebo control groups. Moreover, the drug/placebo difference was only 1.8 points on the clinician-rated Hamilton Depression Rating Scale (HAM-D)! FDA memoranda intimated that the clinical significance of such a small difference was questionable. Hollon, DeRubeis, Shelton, and Weiss (2002) noted that until recently the small drug/placebo response difference had been a "dirty little secret" known only to researchers who conduct clinical trials, FDA reviewers,

and a small group of critics who inspected the published data. Finally, punctuating the fact that the difference between antidepressants effects and placebo are negligible, the Kirsch et al analysis of FDA studies revealed that most of the drug studies funded by the pharmaceutical industry (57%) failed to show a drug/placebo difference. From this perspective, antidepressants, because of their side effects, might best be considered as a last resort and not as a matter of course to address biological underpinnings as suggested by the reviewed articles.

If antidepressants have attained near mythical, but empirically unjustified status, neuroleptics are the grand myth of psychiatry. Here, medication is not a choice; it is a requirement–those diagnosed with severe psychiatric disorders can expect continuous medication to manage a lifelong struggle with mental illness. How many times is deteriorating behavior in a diagnosed person attributed to "He's off his meds." While critiquing antipsychotics is beyond our task here, a series of studies thoroughly discredit the medication-necessity myth. First, the World Health Organization's replicated studies of varying outcomes for schizophrenia in developed versus nondeveloped countries found that what is called schizophrenia tends to dissipate within five years *when medication is either never used or when clients wean themselves off medication*; outcome for people diagnosed with schizophrenia was *better* in nondeveloped countries, where most individuals did not take medication (see de Girolamo, 1996; Jablensky, 1992). Those diagnosed with schizophrenia fared far better in poorer countries, spending less time in hospitals with lower rates of relapse and more likely to be employed and socially connected (Vedantam, 2005). Saraceno, director of the department of mental health and substance abuse at WHO's headquarters in Geneva stated, "Good mental health service doesn't require big technologies but human technologies. Sometimes, you get better human technologies in the streets of Rio than in the center of Rome" (p. AO1). Next, consider a study by Harding, Zubin, and Strauss (1987) that tracked 269 clients admitted to Vermont hospitals with a diagnosis of schizophrenia 32 years after their first admission. They found that about two-thirds of these former backward patients showed no signs at all of schizophrenia and had long since stopped their medications. These facts may appear shocking to many, which speaks to how myth structures our worldview and the actions taken in light of that view.

All the articles reviewed are replete with citations bolstering the case for medication intervention. In this respect, they do not differ from the opening paragraphs of most articles and clinical trials published in psychiatric journals, appearing to present a robust body of literature

supporting the prescription of psychiatric drugs. However, critical analysis uncovers several flaws, including compromised methodology and conflicts of interest, which call into question the cited studies' conclusions. Key flaws include use of inactive placebos, effectively compromising study blinds; use of short time intervals for endpoint analyses, allowing inadequate time to truly measure differences beyond the standard 12 weeks; and reliance on clinician-rated rather than self-report measures, a strategy invariably more favorable to the drug under investigation (Fisher & Greenberg, 1997). In addition, many studies use placebo run-ins, eliminating placebo responders before randomization and unevenly skewing group membership.

While these flaws weaken even seemingly rigorous studies, other cited studies lack even the basic design that would allow a confident conclusion to be drawn. Small sample sizes, nonrandom assignment, or lack of controls should always be mentioned, and those studies that are limited in this way should not be identified as providing support for a medication. For example, Goodman et al. review the literature on the serotonergic system and its proposed association with mood regulation in those diagnosed BPD. They claim studies have "led directly to advances in psychopharmacologic treatment of BPD, namely the use of selective serotonin reuptake inhibitors (SSRIs)" (p. 5). They cite New et al.'s (2004) study that indicates an association between clinical improvement in impulsive aggression and treatment with SSRIs. The New et al. study includes 22 nondepressed impulsive aggressive participants meeting DSM-IV criteria for BPD who were randomized to 20 mg/day of fluoxetine (Prozac) or placebo for 12 weeks with weekly evaluations. Thirteen participants completed the study, 10 receiving fluoxetine and 3, placebo. Clinical improvement was determined by change in mean Overt Aggression Scale (OAS-M) scores (aggression, irritability, and suicidality). The OAS-M is an eight-item clinician-rated scale. The HAM-D, also clinician-rated, was administered weekly with the OAS-M, but no results were reported, begging the question of why and leaving the suspicion of deterioration of depressive symptoms. The OAS-M indicated a significant difference on 2 of its 3 measures (of the 10 in the medication group vs. 3 in the placebo group who remained), with no change on suicidality. The authors, in the "Medication effect on symptoms" section, claim that "non-depressed impulsive aggressive patients with personality disorders show a decrease in aggression and in irritability on 12 weeks of fluoxetine." They then state that since only three subjects completed the placebo arm, this effect "can only be taken as a

suggestion of a drug effect on symptoms" (New et al., 2004, p. 457). Finally, this study was funded by Eli Lilly.

After careful analysis, we find that the New et al. study, cited by Goodman et al., used nonactive placebos, a one-week placebo run-in, a clinician-rated measure of limited psychometric properties, small sample size, limited time frame, incurred a 40% dropout rate, and was funded by the investigational drug's manufacturer. We conclude that this study offers questionable support for "advances in psychopharmacologic treatment of BPD."

It doesn't take too much additional inquiry to follow the trail of how a case gets built in the psychiatric trial literature for later broad claims that drugs are effective. Few, however, have the time to research these claims and the actual soundness of the evidence that backs them up. We looked, for example, in the New et al. study (2004) and found their literature review asserted that a randomized placebo-controlled trial confirmed that SSRIs led to significant improvement in impulsive aggression in those diagnosed with BPD (see Coccaro & Kavoussi, 1997). Examining this trial, we found that it contained a two-week single blind placebo lead-in, did not use active placebo, and showed *no* differences on all client self-report measures. At 12 weeks, out of 40 beginning participants, 50% of the medication group dropped out, leaving 10 participants taking the actual drug. The authors hypothesized that participant dropout was due to the instability characteristic of those diagnosed BPD, and the lack of difference on self-report was symptomatic of poor self-awareness-again, they claim, typical of the study population. Finally, the study was funded by Lilly, makers of Prozac, the drug under investigation. While these kinds of trials may be of some value in pointing a direction for better designs and more research, we decry their uncritical co-optation as evidence for efficacy that, over time, forms the foundation for practice guidelines and broad, unsubstantiated claims in professional as well as lay press.

To examine the problem of conflict of interest in drug study claims, consider Preston's exploration of partial response and nonresponse to antidepressants. Earlier, Preston begins his review of the evidence for psychopharmacology for depression by correctly mentioning that treatment guidelines are suspect, given that most pharmacology research is funded by drug companies. Unfortunately, Preston does not apply this insight to his review of the Texas Medication Algorithm Project (TMAP), the Sequenced Treatment Alternatives for Relieving Depression (STAR-D), and UCLA's Targeted Treatment for Depression Program (TTD). Preston claims that these large-scale studies are largely

untainted by drug company money. Because they are funded by the Texas Department of Mental Health, National Institute of Mental Health (NIMH), and a university, respectively, these studies "may more accurately reflect realistic outcome data, non-influenced by the profit motives of drug companies" (p. 9).

The Texas Department of State Health Services sought to develop, implement, and evaluate "an algorithm-driven treatment philosophy" for major adult psychiatric disorders (TMAP, 2005). Phase 1 of this project created algorithms derived from "scientific evidence and expert clinical consensus" (TMAP, 2005), and later phases sought to implement the algorithms in clinical practice. A medline search reveals just how entwined the Texas Medication Algorithm Project (TMAP) is with pharmaceutical companies. The authors' ties to industry can be found on the first page of a major clinical trial publication (Trivedi et al., 2004) and are worth quoting below:

> Dr Trivedi is a grantee and/or speaker for Abbott Laboratories, Organon Inc (Akzo), Bayer, Bristol-Myers Squibb Company, Eli Lilly and Company, GlaxoSmithKline, Janssen Pharmaceutica Products, Johnson & Johnson, National Institutes of Mental Health, Mead Johnson and Company, Parke-Davis, Pfizer Inc, Solvay, Wyeth, NARSAD, and Forest Laboratories Inc. Dr Rush is a grantee, consultant, and/or speaker for the Robert Wood Johnson Foundation, National Institutes of Mental Health, and Stanley Medical Research Institute, Bristol-Myers Squibb Company, Cyberonics Inc, Eli Lilly and Company, Forest Laboratories Inc, GlaxoSmithKline, Organon Inc (Akzo), and Wyeth. Dr Crismon is a grantee, consultant or advisor, and/or speaker for AstraZeneca, Bristol-Meyers Squibb Company, Eli Lilly and Company, Forest Laboratories Inc, and Janssen Pharmaceutica Products, Pharmacia Pharmaceuticals, McNeil Specialty and Consumer Products, and Pfizer Inc. Dr Suppes is a grantee, consultant, and/or speaker or advisor for Abbott Laboratories, AstraZeneca, Bristol-Meyers Squibb Company, GlaxoSmithKline, Janssen Pharmaceutica Products, National Institutes of Mental Health, Novartis, Robert Wood Johnson Pharmaceutical Research Institute, Stanley Medical Research Institute, Johnson & Johnson Pharmaceutical Research & Development, Pfizer Inc, Pharmaceutical Research Institute, Ortho McNeil Pharmaceutical Inc, UCB Pharma, and Novartis. Dr Miller is a grantee, consultant, and/or speaker for AstraZeneca, Abbott

Laboratories, Bristol-Myers Squibb Company, Eli Lilly and Company, Janssen Pharmaceutica Products, and Pfizer Inc.

Drug company investment in TMAP paid off huge dividends: Pfizer invested $232,000 while gaining $233 million in drug sales; Janssen contributed $224,000 for a $272-million return; and Lilly reaped the most profit by supporting TMAP with $109,000 while receiving $328 million in return sales (Wilson, 2004). The TMAP might be better named the PMAP, or Pharmaceutical Medication Algorithm Project.

Second, Preston appears not to be aware of the four-year investigation by the *Los Angeles Times* into widespread, well-hidden associations between the NIH (umbrella organization of the NIMH) scientists and the biomedical industry (see Willman, 2003). Instead of NIH employees working under the pure and objective auspices of the government, Willman's expos, revealed the NIH to be "one of the most secretive agencies in the federal government when it comes to financial disclosures," documenting extensive, NIH-sanctioned nondisclosure of outside income and confidentiality agreements with corporate employers (p. 2). In looking at the NIH's STAR*D (see, Fava et al., 2003), it appears to be an extension of the TMAP–where the algorithm for the treatment of depression is now tested for implementation on the national stage. We find many of the principal authors from the TMAP in STAR*D, but with an expansion out of Texas and the University of Texas Southwestern Medical Center (the National Coordinating Center for the study) to 13 other hospitals or medical centers with 30-56 primary and specialty care practice sites across the United States (www.star-d.org).

Finally, to claim, as Preston does, that university research is "clean" reflects a lack of understanding about how industry money gets spent. The majority of drug company trials are conducted by university research institutions. Industry money constitutes a significant portion of the dollars that flow into academic research, supporting researchers and general operations (Angell, 2000; Antonuccio, Danton, & McClanahan, 2003). Preston mentions UCLA's Targeted Treatment for Depression Program in this category and cites a presentation at a professional conference, a source that doesn't lend itself to the scrutiny of a published reference. A search on UCLA's home page finds their Neuropsychiatric Institute and various research projects and publications, completed and in process. Nowhere could we find either the Targeted Treatment for Depression Program or the name R. J. Metzner. Nor could we locate Metzner or the TDD by searching the UCLA site. This seemed curious

given Preston's citation on page 8 for "UCLA's Targeted Treatment for Depression Program (Metzner, 2000)." Following up on this, we performed a Google search and found one page (http://www.demomysite.com/psych20/Home/) that contained the words "targeted treatment of depression" and "TDD." This page was also the only page where Richard J. Metzner's name could be located via a Google search. The page's Web site is called PsychiatrySource, The Objective Psychiatry Resource, and claims to be "The only psychiatry resource dedicated to delivering a time-saving, valuable service through unbiased, unfiltered information straight from the source." Readers also learn that the Web site is provided "as an educational resource by AstraZeneca." Readers need not worry, however, as "Information on this site is provided by third parties and not edited by AstraZeneca in any way." On this page, Metzner, listed as affiliated with the University of California-Los Angeles, is quoted in the article entitled, "New approach to treating depression targets specific symptoms":

> A new approach to the management of depression that utilizes the different mechanisms of action of antidepressants is emerging, according to Dr. Metzner. The approach, known as the targeted treatment of depression (TTD), involves a revision of conventional treatment [1]. "This approach [is designed to] improve therapeutic efficacy by utilizing antidepressants with different mechanisms of action for treating different depressive subtypes," he said. He and co-investigators at UCLA have developed TTD.

Footnotes at the bottom of the page indicate none authored by "co-investigator" Metzner.

By means of this original source inquiry, a cornerstone of critical analysis, we now begin to raise more pointed questions. What is the TDD? What is Metzner's role at UCLA and at the American Psychiatry Association 2000 Conference, and who employs him? Until these questions are adequately answered and until source publications can be accessed and analyzed, Preston's case for "smart," targeted pharmacotherapy based on these sources is compromised.

In summary, when one scratches below the surface of drug efficacy studies, smart drugs don't look so smart. The superior effectiveness of many drugs is largely myth, based on an empirical house of cards and driven by corporations that have no limits in their ability to spread their influence. Antonuccio et al. (2003) detail the vast reach of the pharmaceutical industry–from Internet, print, and broadcast media, direct-

to-consumer advertising, "grassroots" consumer-advocacy organizations, and professional guilds to medical schools, prescribing physicians, and research–even into the boardrooms of FDA. They conclude, "It is difficult to think of any arena involving information about medications that does not have significant industry financial or marketing influences" (p. 1030). Saturation of popular and professional media accompanied by the inability to make informed evaluations of research reinforces medical discourse. In the process, the credibility of nonmedical options, particularly stand-alone (not combined with medication) psychotherapy to affect desired results recedes into the background.

THE MYTH OF PSYCHOTHERAPY/ PHARMACOTHERAPY INTEGRATION

General wisdom has it that medications and psychotherapy are more effective combined than either separately. This view clearly calls for a collaborative approach between the medical prescriber and therapist. Goodman et al. cite the Clinical Practice Guidelines for BPD of the American Psychiatric Association, recommending a combination of psychotherapy with symptom-targeted pharmacotherapy. While acknowledging the limited empirical evidence for more delineated and comprehensive guidelines for personality disorders, their hope is that advances in understanding the neurobiologic components of these problems and the development of more effective pharmacologic interventions, along with further validation of psychotherapy, will produce more numerous and effective interventions. McLean et al. see the complex biological, psychological, and social interactions of anxiety as requiring an interdisciplinary effort. And Preston, stating that studies demonstrate that combined psychotherapy and pharmacotherapy produce better outcomes, calls for an integrated and multidisciplinary approach.

Why does such a sensible-sounding option concern us? First, the assumption that both psychotherapy and pharmacotherapy produce better outcomes has weak empirical support. Reviews prior to 1997 demonstrated no advantage for combining approaches (e.g., Fisher & Greenberg, 1989, 1997; Robinson, Berman, & Neimeyer, 1990; Wexler & Cicchetti, 1992). The combined theory attained its current taken-for-granted status after the publications of Thase et al. (1997) and Keller et al. (2000) studies. The first, a "mega-analysis," examined 595 persons enrolled in six treatment protocols conducted over a 10-year

period. This study found that, for persons suffering from mild-to-moderate depression, no advantage was gained by adding an antidepressant. However, combining the two did appear to offer some benefit for the minority of those suffering with severe, recurrent depressions (Thase et al., 1997). In Keller et al.'s chronic depression trial, three-quarters of the combined group compared to one-half of both the medication and psychotherapy alone groups showed a positive response at 12 weeks. The authors stated that the results demonstrated that the combination treatment was more efficacious than either treatment alone. Critics of the study raised questions about the lack of a placebo control group, the 12-week duration, and the use of only a single clinician-rated outcome measure. In addition, the authors of this study were so heavily tied to pharmaceuticals, the journal stated: "It would have used too much space to disclose them fully in the Journal" (p. 1462).

Recently, the combination hypothesis has made in-roads in the treatment of adolescent depression. The Treatment of Adolescents Depression Study (TADS Team, 2004) claimed that combined psychotherapy and fluoxetine was better than either alone for depressed adolescents. Press announcements were breathless–finally, an unequivocal solution to adolescent depression. A closer look at this study reveals all the flaws mentioned previously, including the fact that two of the four study arms were not blind. The TADS investigators acknowledged that, because of inequities in conditions and lack of blinding, the "'active ingredient' in improvement cannot be specified" (p. 818). In summary, actual research evidence, even with the most generous interpretations, indicate that for most experiences of depression, combination treatments do not provide added benefits and may unnecessarily subject clients to unpleasant side effects and unnecessary costs. Psychotherapy *alone*, in other words, should be considered first.

Second, medications continue to pose risks related to adverse events. These risks are often downplayed in clinical trial presentations, but can be discerned through careful scrutiny. For example, in the Shelton et al. 1999 study cited by Preston, which examined the efficacy of augmentation of fluoxetine with olanzapine (Zyprexa) (a Lilly-funded study, augmenting one of its drugs with another of its products) for treatment-resistant depression, the authors claim in the abstract that there were no significant adverse drug reactions.[3] In the results section, the authors list mean weight differences, while not pointing out here that these differences were significant among groups. Not until the discussion section do the authors indicate that weight gain for both the olanzapine and combined (olanzapine and fluoxetine) groups was a

treatment-emergent event. Weight gain is listed as 6.07 kg for the olanzapine group and 6.67 kg for the combined group over the eight-week double-blind period. Excess weight is increasingly recognized as a risk factor for a host of medical problems, not to mention its impact on individuals' social ease and self-esteem. Still, the authors say, olanzapine, either alone or in combination, was "well tolerated," an oft-used phrase that effectively disguises real adverse drug effects.

Finally, we suggest that a combined approach often places therapists in confined and scripted roles in the treatment process, hampering their ability to flexibly connect with their clients. We value collaboration with other professionals, and often find it useful when clients are fully in accord with this type of assistance. However, our experience has been that the role of therapy is often relegated to one that supports the medical intervention. Therapists, as Preston suggests, are in prime positions to monitor treatment compliance, drug response, and adverse effects. The "collaborating" therapist is often seen as the *in vivo* arm of the busy physician, smoothing over the rough edges as the medication takes effect, educating clients or family members about the disorder, and making sure that medication compliance is a treatment goal. We have experienced invaluable collaborations with prescribing physicians where there is true partnership, particularly where all insist on active client participation in the team. However, more often, if we support a client's position to discontinue medication or challenge a diagnosis, our own expertise, even ethics, can be brought into question.

In summary, critical theory offers a useful framework for understanding why integration is problematic at the least, and assimilative at worst. When one discourse is so dominant and so pervasive, as we believe medical/biology is in our culture as well as our field, then an opposing point of view is always second. Even when presented as "both/and," "integration," "collaboration," or that appealing term, "biopsychosocial," clinical practice parameters tend to be based on the dominant biological paradigm. Biopsychosocial becomes Biopsychosocial, with interpersonal and social aspects bringing up the rear. We appreciate the emphasis on psychosocial approaches discussed in the articles reviewed here. However, our concern continues to be that, within the context of integration, they may simply provide further fuel for the promotion of a medical paradigm in psychotherapy, including research dollars and practice initiatives despite increased risks and minimal additive benefits.

THE MISSING LINK: THE CLIENT

Conspicuously absent from discussion in the three articles was the owner of the dysregulated neurotransmitters, aberrant brain activity, genetic predisposition, or diagnosis and the object of intervention and integration–namely, the client. We do not claim that never the twain, therapy and medication, shall meet. We simply have a different reference point for how such a combination might occur. Instead of algorithms or manualized treatments, our reference point is the client, including his or her view of the problem, preferred future, and ideas for change. Rather than diagnosis and prescriptive treatment, we place the client center stage, not because of some humanistic impulse (though that is certainly a motivation), but because empirical research has repeatedly attested to the client's dominant role in change (Asay & Lambert, 1999; Bohart & Tallman, 1999; Duncan et al., 2004; Hubble, Duncan, & Miller, 1999). According to almost five decades of research, outcomes in psychotherapy are not due to specific ingredients such as a technique or a pill, but to factors that all bona fide treatments have in common–the engagement of the client through a strong therapeutic relationship.

The three articles reviewed virtually ignore that most of the variance in change in psychotherapy is accounted for by the so-called extratherapeutic factors–those variables associated with the client, including unexplained (and error) variance. These variables are incidental to the treatment model and idiosyncratic to the specific clientfactors that are part of the client and his or her environment that aid in recovery regardless of participation in therapy (Lambert, 1992). What clients bring to the process–their strengths, attributes, struggles, motivations, and social supports–accounts for 40% of the variance (Lambert, 1992); clients are the engine of change (Bohart & Tallman, 1999). Wampold's (2001) meta-analytic perspective assigns an 87% contribution to these client factors and unexplained variance.

In the absence of compelling evidence for any of the specific client variables to predict outcome or account for the unexplained variance, this most potent source of variance remains largely uncharted. This suggests that the largest source of variance cannot be generalized because the factors differ with each client. These unpredictable differences can only emerge one client at a time, one alliance at a time, one therapist at a time, and one treatment at a time.

If therapy is to use this knowledge, then it cannot hamstring itself with the medical model. The medicalized milieu of present-day practice increasingly defines service as the appropriate application of empirically supported treatments. However, the client is not a diagnosis, the therapist, not a technician, and therapy not a simple prescription. Instead, therapy is a dialogical interaction fueled by relationship and idiosyncratic client factors; it is predictable only in that it hinges on the correct fitting together of client (including family, community and culture), therapist, and the unpredictable path that evolves between them. Instead of evidence-based treatment, we argue for practice-based evidence, where evidence is systematically and routinely gathered from clients throughout therapy to inform the role we play and the interventions we use (Duncan et al., 2004) Valuing such client-based feedback has shown to be a robust enhancer of outcome, improving effectiveness up to 65% in clinical settings (Miller et al., in press; Whipple et al., 2003).

Having said that, we would never stand in the way of a client considering medication if they believed their problems were of biological origin and thought drugs might be helpful. It is up to therapists to privilege clients' wishes in the therapy conversation, including their trains of thought, their brainstorming, and their talk. When clients put medication on the table, then therapists can naturally help them explore it as an option. When clients believe medication will help, feel more hopeful at the possibility of trying it, and are "in the driver's seat" in making an informed choice (including information about side effects, length of treatment, and possibilities of relapse), then medication can be beneficial. Following the client's theory of change maximizes client participation, strengthens the therapeutic bond, and enhances therapeutic outcomes.

The integration of psychotherapy and psychopharmacology likely began as a goodwill effort to help clients overcome distress. Given the empirical evidence, we have come to believe that seeking integration based on a diathesis/stress paradigm is a mistake. Biology, largely the emphasis in this equation, cannot explain human distress. As Valenstein (1998) points out, analyzing human behavior by focusing on neurotransmitters and brain activity is like understanding books by the composition of their ink, paper, and binding. Instead, we view human behavior as more appropriately understood by exploring how people make sense of their lives and are connected to their social communities. We believe that integration must be grounded on clients' initiative and resiliency-in particular, clients' perspectives of the fit and benefit of the services they receive. This kind of integration can only happen by

bringing in the missing link, the client's voice, and tailoring any combination of approaches in accord with direct client feedback at the individual client level, *one client at a time*.

RESISTING MYTH

Through the lens of the three articles reviewed, we view the future. Psychotherapy, including family therapy, promises advancing science, buttressed by expanding imaging and pharmacological technologies. In time, the precise markers of mental illnesses will be delineated, not only by discreet expressions of emotion and behavior, but by tell-tale patterns of brain activity. Next, the right drug will modify the wayward brain biology, bringing the patient back to health and productive life. Meanwhile, the family, educated and guided by the therapist, will not interfere with recovery and will know how to encourage and support each other as their loved one undergoes treatment.

We argue against those who say the above is hyperbole, and that integration promises to soften the medical edge and allow the ebb and flow of relationship in the mix. While we believe many therapists will continue to do what they do best, form relationships and seek clients' opinions to shape the course of therapy, this way of working will be marginalized in a medical climate. As we have hoped to describe, a move toward biology and toward medical prescription of treatment in psychotherapy has effects, ones that counter client resourcefulness and de-energize the process. Meanwhile, relationship- and context-based therapists will play supportive roles to those more medically trained.

We cannot predict the future, but we can learn from the science at our doorstep. This science consistently indicates a picture far different from the one described above as well as the one described in the three articles. We believe the future envisioned above is myth (albeit one with real effects) and radically misses the chief complexities of our work-the uniqueness of each client's experience, imbedded in context, and idiosyncratic path for change. We recommend that the medical model, as it gains ever more momentum in our field, be questioned for its assumptions, method, and implications. We call for recognition of the fact that psychiatric drug therapy is a profit-driven industry, built on questionable science. To be trigger-happy to bring medication into the discussion automatically is to be under the influence of bad science and great marketing. What is required is a reconnection with what therapists know and have experienced over and over, both in their clients and in

themselves–that most people can and will develop solutions to even the most daunting dilemmas, given support and encouragement. Clients' roads to recovery take sometimes unorthodox routes, yet change will and does occur naturally and universally. We privilege this stance as a way to "level the playing field," and to resist the noisy and ever-present chant to medicate. Fortunately, this stance finds support, not only in our everyday wisdom and practice, but in robust empirical evidence. It is time for therapists to learn the data, reinvigorate their belief in therapy, and offer clients real choices for change.

NOTES

1. Despite our critical perspective of psychotropic medications, we are not anti-drug any more than we are anti-CBT or any other viable approach to human distress. Further, we fully recognize that some clients are helped by medication and freely choose drugs as a first line of defense. In truth, we honor client preferences, even for medication, and stand against any practice that does not center clients' desires about how they may be helped. At the same time, we believe clients should have unbiased information about the actual risks and benefits of psychotropic drugs.

2. Validity asks the question: How useful is diagnosis to treatment? For example, in the prevailing diagnostic guide for BPD, there are 126 possible ways to arrive at a diagnosis. All it takes is to meet five out of nine criteria. If one can be diagnosed as BPD 126 possible ways, how distinctive or valuable can such a diagnosis be?

3. Preston cites this study as support for an augmentation strategy for partial responders using SSRI plus atypical antipsychotic (e.g., Zyprexa). Of 28 participants entering the double-blind phase of the study and comprising 3 groups, 25 completed. A sole sentence in the discussion section contains the acknowledgment that, because of the small group size, the results must be considered preliminary.

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