
PRACTICE

The Other Side of Psychopharmacology: A Review of the Literature

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A number of literature reviews exist that support the use of psychotropic medications. This article provides a review of the disconfirming literature regarding psychopharmacology use. Comparing the first review of psychopharmacology published in the counseling field two decades earlier to what is known currently, I examine recent developments in psychopharmacology research focusing on the safety, efficacy, side-effects, and theoretical assumptions of various classes of psychotropic medications. This article concludes by addressing counselor identity, practice and training concerns vis-à-vis psychiatric medications and the medical model.

Ponterotto (1985) published the first article review of psychopharmacology within the counseling literature. He proposed that counselors must become familiar with the current medications (i.e., antipsychotics, antidepressants, anti-anxiety, and lithium salt agents) used to treat psychiatric disorders, especially given these medications' "increased technology," "more sophisticated empirical validation procedures," and "treatment efficacy" (p. 109). Although many new medications have come onto the market since 1985, more recent literature reviews (e.g., King & Anderson, 2004) discuss the benefits of the use of psychotropic medications with very little discussion addressing the conflicting evidence.

Although Hansen (2005) recently discussed the role of the medical model within the counseling profession and the impact that this adoption will have on our future identity as counselors, there is little discourse concerning the problems associated with psychotropic medications and the adoption of psychopharmacology practices as part of the professional counselor agenda. In this

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article, I address this problem and encourage counselors to call into question the uses of technology (e.g., brain scans), research methodology, and treatment efficacy of these medications based on the examination of the existing research. Specifically, I suggest counselors investigate rigorously the uses and consequences of these medications regardless of their support or skepticism. In this effort, this article serves as a review of the disconfirming literature of psychopharmacology for mental health counselors to consider. As a caveat, I admit that this article is inherently biased and does not provide supportive evidence for psychopharmacology, which is written elsewhere.

In keeping with the organization of Ponterotto's (1985) article, this article provides counselors with access to information about the safety, side-effects, and efficacy problems regarding the classes of psychotropic medications he presented (i.e., antipsychotics, antidepressants, antianxiety, and lithium salts). In addition, I discuss misconceptions about the mental illnesses these medications treat. I also provide information about the critical skills counselors need to have to examine psychopharmacological research, and I offer guidelines for counselors concerned with the role of psychopharmacology in practice. I first address the major assumption used in the support of psychopharmacology.

The Major Assumption

Before discussing the use of psychotropic medications in general, one must consider the major assumption on which the uses of these medications are based: psychiatric disorders must have a specific biological etiology—neuropathological, neurochemical, or genetic explanation (Double, 2004; Kendler, 2005; Stahl, 2000). However, no valid diagnostic tests exist to determine a physical disease process for the great majority of diagnoses found in the *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR)*, text-revision (American Psychiatric Association [APA], 2000) (Ducommun-Nagy, 2003; Valenstein, 1998). Those disorders listed in the *DSM-IV-TR* for which a clear, undeniable disease process is present (e.g., Alzheimer's disease and other various forms of dementia) or a clear genetic defect has been located (i.e., Rett's disorder) fall under the purview of neurology, not psychiatry (Ducommun-Nagy, 2003; Encyclopedia of Mind Disorders, 2005; Glasser, 2003). Psychiatrist Kenneth Kendler (2005), co-editor-in-chief of *Psychological Medicine*, stated, "We [psychiatrists] have hunted for big, simple neuropathological explanations for psychiatric disorders and have not found them. We have hunted for big, simple neurochemical explanations for psychiatric disorders and have not found them. We have hunted for big, simple genetic explanations for psychiatric disorders and have not found them" (pp. 434-435).

Despite the lack of clear evidence for neuropathological, neurochemical, or

genetic explanations for psychiatric disorders, the beliefs in such are heavily perpetuated by psychopharmacologists and physiological psychiatrists (Valenstein, 1998), who differ from the declining number of psychiatrists and psychiatric nurse practitioners who appreciate the contextual factors affecting mental health. Psychopharmacologists and physiological psychiatrists believe that mental health problems reduce down to chemical and electrical exchanges between brain cells (neurons). With this philosophy, psychotropic medications are marketed aggressively and prescribed indiscriminately (Rosenheck, 2005; Schultz, 2004, Wazana, 2000) with the message that these medications will correct alleged brain defects related to psychiatric disorders. The following sections draw on existing psychopharmacological literature on common medications to examine this orthodoxy.

ANTIPSYCHOTIC (NEUROLEPTIC) MEDICATIONS

In this section, I address the claims made by Ponterotto (1985) regarding antipsychotic medications in his original article. I also discuss recovery issues related to schizophrenia. Ponterotto stated the following regarding antipsychotic medications:

1. Antipsychotic medications have been “proven effective in the management of various psychotic disorders, including psychotic depression, manic-depression, and *particularly schizophrenia* [italics added]” (p. 109).
2. Antipsychotic medications serve as prevention and to decrease pre-existing symptoms from emerging.
3. Antipsychotic medications restore normal cognitive functioning; decrease psychotic thinking, projection, suspiciousness, perplexity, delusional thoughts, hallucinations, illogical thought processes, the inability to separate relevant from irrelevant details, excitement, rambling, tangential speech, and impulsive behavior; and restore normal psychomotor activity.
4. Antipsychotic medications are safe, non-addicting, and non-lethal.

Efficacy

Current psychiatric texts (e.g., *Textbook of Psychiatry* [1999], the *Massachusetts' General Hospital Handbook of General Hospital Psychiatry* [1997], and *Principles and Practice of Psychopharmacology* [1993]) highlight the benefits of antipsychotics that Ponterotto (1985) mentioned. Often these texts attribute the decline of admissions into psychiatric hospitals to the benefits of antipsychotic medications, despite evidence to the contrary (Whitaker,

2004).¹ For example, only 30% to 50% of clients on antipsychotic medications experience any level of remission of psychotic symptoms (Jackson, 2005; Whitaker, 2002), and up to 74% of clients discontinue their medication within 18 months (Lieberman et al., 2005).

Ponterotto (1985) looked promisingly into the future when he mentioned that new antipsychotic medications were on the horizon. His prediction that within the next five years new medications would be available has now come to fruition. A number of medications, including Risperdal (risperidone), Zeldox (ziprasidone), Seroquel (quetiapine), Zyprexa (olanzapine), and Abilify (aripiprazole) have since come on the market, each touting how it is better than the other. On September 19, 2005, however, the National Institute of Mental Health (NIMH) concluded that the newer atypical antipsychotic medications did not perform any better than the older conventional antipsychotic medications. Moreover, Jackson (2005) outlined how the newer medications produce many of the same side-effects associated with conventional medications. Unfortunately, it is the side-effects of these medications that may have given us our perception of psychotic people. Whitaker (2002) wrote:

The image we have today of schizophrenia is not that of madness—whatever that might be—in its natural state. All of the traits that we have come to associate with schizophrenia—the awkward gait, the jerking arm movements, the vacant facial expression, the sleepiness, the lack of initiative—are the symptoms due, at least in large part, to a *medication-induced* [italics added] deficiency in dopamine transmission (p. 164).

Recovery from Psychosis

The World Health Organization (WHO) examined the success rates for the treatment of major psychotic disorders, specifically schizophrenia. Leff, Sartorius, Jablensky, Korten, Ernberg (1992) performed a 5-year follow-up of clients diagnosed with schizophrenia in the following cities using standard DSM criteria: Aarhus, Denmark; Agra, India; Cali, Colombia; Ibadan, Nigeria; London, Moscow, Prague, and Washington, D.C. Clients in developing countries (i.e., Columbia, India, and Nigeria) experienced a higher rate of recovery

¹ The field of social work led the movement during the 1950s towards the deinstitutionalization of those with mental disorders. Social workers showed that they could provide individual and family psychotherapy, vocational and social rehabilitation, and coordinate social services such as housing, legal and medical assistance, and transportation needs. In addition, the federal government in 1963 began subsidizing local governments for relocating those within psychiatric hospitals into nursing homes, shelters, and other alternative placements. In 1972, the federal government amended the Social Security Act to authorize those with mental disorders to receive disability payments, which extended the number of options for clients. Finally, a large number of those with mental disorders found themselves in places where they could obtain free meals and housing—the correctional system (Healy, 2004; Kaner, 1991; Whitaker, 2004)

from schizophrenia than those in developed countries (e.g., The United States). Although the WHO study did not identify the cause for the discrepancy, the study did allude to the differences in psychiatric treatment. In the developing countries, only 16% of clients were maintained on antipsychotic medication versus 61% of clients maintained on these medications in the developed countries (Whitaker, 2004). Regarding outcomes, measured by symptomatic status at time of follow-up, time spent in a psychotic episode and pattern of course, clients from Agra and Ibadan fared better than clients from the cities in developed countries, especially with regards to good social outcomes (Leff et al., 1992).

Contrary to what some may expect, up to 68% of clients diagnosed with schizophrenia experience partial to full recovery (Bleuler, 1974; Ciompi, 1988; Harding, Brooks, Ashikaga, Strauss, & Breier, 1987; Harrow, Grossman, Jobe, & Herbener, 2005; Huber, Gross, Schuttler, 1975; Mathews, Basil, Mathews, 2006; Tsuang, Woolson, Fleming, 1979) and an estimated 25% to 40% of acute psychotic clients recover without any antipsychotic medications (Bola & Mosher, 2002). The greatest predictors of who recovers include *little or no medication*, emphasis on hope, assistance in non-hospital environments, closer proximity to home, and a supportive environment (Bola, Mosher, & Cohen, 2005; Mosher, 1999).

Relapse Prevention and Latent symptoms

The success of a medication is often based on its ability to prevent relapse. However, neuroleptics may *increase* the likelihood of relapse and *worsen* symptoms (Gur, Maany, Mozley, Swanson, Biker, & Gur, 1998; Zarate & Tohen, 2004). For example, antipsychotic medications are known to increase the number of dopamine receptors or neuronal receptors' sensitivity to dopamine (Davis & Rosenberg, 1979; Moore, 1986) thus creating neurological changes in the brain (Chakos, 1994; Gur et al., 1998; Harrison, 1999; Jellinger, 1977). The neurons grow additional dopamine receptors to compensate and adapt to the excessive dopamine in the brain caused by the medication. If a client with schizophrenia discontinues taking neuroleptic medication, the brain no longer has the increased level of dopamine that accommodated the increase number of receptors; in short, there is a deficiency of neurotransmitters for the number of receptor cells perhaps increasing the likelihood for future psychotic behavior (i.e., discontinuation syndrome).

Such anatomical changes (i.e., changes in size, density, and properties of neurons and glia, especially within the striatum and frontal cortex, and significant enlargement and scarring in the caudate) may account for the differences, as measured by brain scans, between the brains of schizophrenic clients who have been medicated for years, and a control group which has not been medicated (Double, 2004; Jackson, 2005a, 2005b). Given that within the United States,

the psychiatric community adheres to a strict biochemical explanation for schizophrenia and psychotic behavior, and medicates as a first line of defense, such treatment may inadvertently lead to greater chronicity (Whitaker, 2002; 2004) by increasing the rate of relapse due to the brain's response (i.e., neuroplasticity) to neuroleptics. Cultures that do not appear to maintain clients on neuroleptic medications (as in the WHO study) evidence higher rates of recovery.

When clients discontinue medication, they may experience complications (e.g., tardive psychosis; Silvestri et al., 2000). Pharmacological researchers report that the complications upon discontinuation of the medication are a return of the psychotic symptoms (Gitlin et al., 2001). The client is then re-medicated. However, studies suggest that the symptoms are just as likely due to withdrawal from the medications—known as neuroleptic discontinuation syndrome (Tanter & Healy, 1998). Such evidence of neuroadaptation within the brain precisely meets the definition of addiction (Shafer & Albanese, 2005). Moreover, Zarate and Tohen (2004) found that clients maintained prophylactically on antipsychotic medication demonstrated more detrimental effects than those who discontinued the medication, such as more depressive symptoms, higher rates of dysphoria and parkinsonism, and greater discontinuation rates.

Psychopharmacologists (Pagliaro & Pagliaro, 1999) report that medications can unveil pre-existing latent psychosis (i.e., the client has dormant psychosis that is triggered by medication). However, psychiatrists Breggin (1991) and Glenmullen (2000) proposed that psychotic behavior is one side-effect of the medication. According to these authors, psychotic behavior is likely due to the anatomical changes, specifically supersensitivity of the dopamine receptors, caused by the medication. Such reactions are reported to be clear evidence of a withdrawal syndrome and physical dependency (Jackson, 2005; Shafer & Albanese, 2005).

Cognitive Functioning

Ponterotto (1985) mentioned that antipsychotic medication restores normal cognitive functioning as well as providing other benefits. Any cursory examination of the literature shows evidence consistent with these comments, especially with atypical antipsychotic medications. Yet, the evidence is inconclusive and suspect. For example, Keefe, Silva, Perkins, and Lieberman (1999) found extensive methodological flaws among all 15 studies from 1990 to 1998 that examined the cognitive benefits of antipsychotic medications. Other authors have reported that antipsychotic medications that block D2 dopamine receptors may not produce any effect on cognitive functioning (Berman et al., 1986; Faherland, Mackeprang, Gade, & Glenthøj, 2004), or, if there is an effect, it is negligible (Serper et al., 1994). Evidence also suggests that conventional antipsychotic medications may actually exhibit a deleterious effect on cognitive

functioning (Sweeney et al., 1991). The atypical antipsychotics have been widely reported to improve cognitive functioning (Woodward, Purdon, Meltzer & Zald, 2005), but Jackson (2005) reported that the cognitive benefits may in fact be an illusion stating, "it might not be the case that newer [antipsychotic] drugs are such potent cognitive restorers, as much as it is the case that the older drugs are so cognitively toxic" (p. 223). Finally, extrapyramidal symptoms (EPS), which I describe in the next section, interfere with a client's ability to complete cognitive tasks that require motor output.

Safety, Addictive Potential, and Lethality

A discussion of side-effects associated with antipsychotics must begin with an understanding that there is no requirement for physicians to report side-effects to the Food and Drug Administration (FDA) after a medication has been approved by the FDA (Jackson, 2005). In addition, statistics on side-effects are likely either underreported, undisclosed, or simply unknown by the pharmaceutical industry because of how studies are designed. For example, many pharmaceutical studies on psychiatric medications last no more than 4 to 8 weeks and use comparatively small sample sizes (Safer, 2002). These methodological characteristics may contribute to inaccurate findings whereby the studies may not be long enough for side-effects to present and therefore go unreported (Jackson, 2005).

Despite Ponterotto's (1985) claim that antipsychotic medications are relatively safe and non-addictive, there is considerable evidence to the contrary. An overwhelming number of clients prescribed antipsychotic medications will develop significant side-effects (Glazer, 2000) with some serious side-effects not disclosed in the literature. For example, Whitaker (2002) reported that 1 in 145 clients died who participated in trials for Risperdal (risperidone), Zyprexa (olanzapine), Seroquel (quetiapine), and Serlect (sertindole), but these deaths were not reported in the scientific literature. The longer clients are medicated with antipsychotic medications, the more severe the symptoms and neurological changes (Zarate & Tohen, 2004). Similarly, recent studies have linked the use of antipsychotics to metabolic changes including onset of diabetes mellitus and hyperlipidemia (Marder et al., 2004; Melkersson, Hulting, & Brismar, 2000; Wirshing, Boyd, Meng, Ballon, Marder, & Wirshing, 2002). Such concerns led the FDA to request that pharmaceutical companies warn consumers of the increased risk for diabetes (Food & Medication Administration, 2004).

Ponterotto (1985) claimed that a brain disorder that is caused by antipsychotics—tardive dyskinesia (TD)—only occurs in clients who have taken antipsychotics for many years. Glazer (2000b) found among 362 clients who were followed for a period five years, increasing incidence of TD the longer clients took antipsychotics. Thirty-two percent of these clients on the medications for 0 to 5 years experienced TD; 49% experienced TD with 5 to 10 years

of use; 57% with 10 to 15 years of use; 65% with 15 to 20 years of use; and 68% with 20 to 25 years of use. Some psychopharmacologists also have claimed that TD is a “pre-existing neuropathology” that only became evident because of the superior quality of the medication (Fenton, 2000). This hypothesis is not supported by any conclusive evidence and may result in clients feeling blamed or labeled defective for symptoms caused by the medication (Prosky & Keith, 2003).

In addition, other neurological symptoms manifest secondary to antipsychotic medication use: parkinsonism (slowed movements, decreased facial expression, resting tremor, and shuffling gait); dystonic symptoms (sustained muscle spasms that impact the neck or shoulder); and akathisia (intense feelings of restlessness) (Breggin, 1991; Breggin & Cohen, 1999; Gualtieri & Sovner, 1989; Preston, O’Neal, & Talaga, 2000; Whitaker, 2002). Additional symptoms described by Whitaker (2002) include blindness, fatal blood clots, arrhythmia, heat stroke, swollen breasts, leaking breasts, impotence, obesity, sexual dysfunction, blood disorders, painful skin rashes, seizures, and potential for having offspring with birth defects. Despite Ponterotto’s (1985) claim that these symptoms are rare and only occur after years of usage, more recent evidence suggests that such symptoms can occur quickly after initial dosages or upon discontinuation (Miller, 1999). Moreover, with the sharp increase of antipsychotic use among children and adolescents (Olfson, Blanco, Liu, Marengo, & Laje, 2006), these side-effects must be carefully weighed and monitored by counselors who work with children and adolescents.

When Ponterotto (1985) wrote about lethality, he was referring to the potential of death due to overdose. Yet, there is evidence that death can occur as a side-effect of antipsychotic medications. For example, Jindal, MacKenzie, Baker, and Yeragani (2005) reported that medicated clients diagnosed with schizophrenia are 1.4 times more likely to die from cardiac arrest than clients diagnosed with schizophrenia who are antipsychotic medication free. An increased risk of mortality is also especially high among the elderly who are often prescribed antipsychotic medications excessively (Breggin, 1991; FDA, 2005), which is especially disconcerting when none of these medications were specifically approved for the treatment of geriatric disorders such as dementia (Aschenbrenner, 2005). The FDA required recently a black-box warning, the most serious warning put forth by this agency, for the following atypical antipsychotics when used for the elderly: Clozaril (clozapine), Risperdal (risperidone), Zyprexa (olanzapine), Seroquel (quetiapine), Geodon (ziprasidone), and Abilify (aripiprazole). Finally, Healy and colleagues (2006) examined the lifetime suicide rates of clients with schizophrenia pre and post introduction of chlorpromazine (Thorazine) where they found a 20-fold increase *after* chlorpromazine’s introduction.

ANTIDEPRESSANTS

Many new antidepressant medications have come onto the market since Ponterotto's (1985) original article in which he discussed only tricyclic antidepressants and monoamine oxidase inhibitors (MAOI). Newer classes of antidepressants include heterocyclic antidepressants, selective serotonin reuptake inhibitors (SSRI), serotonin receptor antagonists, selective norepinephrine reuptake inhibitor (SNRI), and others (e.g., Effexor [venlafaxine], Cymbalta [duloxetine], Buspar [bupropion], and Remeron [mirtazapine]) (Jackson, 2005).

Given that tricyclic antidepressants prescriptions account for only two percent of prescriptions and MAOIs account for even fewer (Levin, 2005; Stafford, MacDonald, & Finkelstein, 2001), this section will focus primarily on the newer and most popular antidepressants, which raise similar concerns associated with the older medications and appear to be no more effective (Mechanic, 2000). In particular, I address Ponterotto's (1985) claims and current popular assumptions about antidepressant medications, which include the following:

1. Antidepressants are not stimulants.
2. Antidepressants activate or reactivate latent psychosis.
3. Antidepressants reduce suicidality.
4. Psychotherapy is not appropriate for endogenous (biological) depression.
5. Chemical Imbalance theory explains depression.
6. Antidepressants rarely produce neurological damage.

Antidepressants are Activating

Although it is unclear what exactly Ponterotto (1985) meant when he wrote that "antidepressants are not stimulants" (p. 110), other authors have recently suggested that antidepressants have activating properties. For example, psychiatrist Peter Breggin (2005) stated that antidepressants can cause a stimulant syndrome. Others have drawn similar conclusions (Glenmullen, 2000; Stahl, 2004). Brambilla, Cipriani, Hotopf, and Barbui (2005) found that Prozac, for example, had activating side-effects that include insomnia, agitation, tremors, and anxiety, as well as adverse gastrointestinal effects. Given the increase in these concerns, the FDA recently warned of the following antidepressant side-effects: anxiety, agitation, panic attacks, insomnia, irritability, hostility, aggressiveness, impulsivity, akathisia, hypomania, and mania (FDA, 2004b).

Activation or Reactivation of Latent Psychosis

Similar to the notion that antipsychotic medications can activate or reactivate latent psychosis, such claims have been made about antidepressants. Breggin

(1991) suggested that this conclusion appears to function as a means by which the pharmaceutical companies can distance themselves from the possibility that the psychosis is actually a deleterious result of the medication. Antidepressants have been implicated in inducing psychotic symptoms resulting in suicide attempts (Mizoguchi & Monji, 2005; Pompili, Tondo, & Baldessarini, 2005). This particular problem has been publicized since the FDA warned about the increased potential for suicidal behavior and ideation among children and adults (Food and Medication Administration, 2004b). The FDA (2005a) published, *List of medications receiving a boxed warning, other product labeling changes, and a Medication Guide pertaining to pediatric suicidality*, which contains 34 medications that must be monitored closely when prescribed to children.

Antidepressants also have been linked to triggering manic episodes as previously indicated. When this occurs, clients maybe misdiagnosed with Bipolar I Disorder (American Psychiatric Association, 2000) and prescribed an anticonvulsive medication like Depakote (valproate) or lithium. Psychopharmacologists have reported that the effectiveness of the medication served to unveil latent psychosis in the client. (Boulton, Baker, Martin-Iverson, 1991; GlaxoSmithKline, 2005). However, Glenmullen (2005) reported that antidepressants may trigger psychotic behavior as a side-effect.

Psychotherapy for Endogenous Depression

Ponterotto (1985) drew a distinction between the treatment protocols for endogenous (not related to external events therefore biological) as opposed to exogenous (event induced) depression stating that endogenous depression is rarely helped by psychotherapy and that the best course is pharmacotherapy. Vetter, Pritzbuier, Jungmann, Moises, Kropp, and Köller (2000) took a completely different view stating that psychotherapy is considered “indispensable” for clients described as having endogenous depression. Moreover, the widespread, indiscriminant prescribing of antidepressants for endogenous depression, and depression in general, may be counterproductive. Moncrief and Kirsch (2005) reported in the *British Medical Journal* the following summary points about antidepressants that the pharmaceutical industry does not disclose:

1. Claims that antidepressants are more effective in more severe conditions have little evidence to support them (which is particularly important when considering endogenous depression).
2. Recent meta-analyses show selective serotonin reuptake inhibitors have no clinically meaningful advantage over placebo.
3. Methodological artifacts may account for the small degree of superiority shown over placebo.

4. Antidepressants have not been convincingly shown to affect the long-term outcome of depression or suicide rates.
5. Given doubt about their benefits and concerns about their risks, current recommendations for prescribing antidepressants should be reconsidered.

Chemical Imbalance

One of the most troubling phenomena is the propagation that mental health disorders are caused by chemical imbalances as reflected in Ponterotto's (1985) article. This popular assumption is particularly true for depression. The counseling profession is indoctrinated continuously by the media, medical professionals, and counselors alike that the chemical imbalance theory is in fact true (Lacasse & Leo, 2005; Moncrieff & Kirsch, 2005; Schultz, 2004), even though a leading psychiatric textbook acknowledges that "...these theories are unproven and perplexing..." (Colp, 2000, p. 3309).

There is growing pressure to support such theories through the suggestions that counselors have an obligation to understand and make referrals for psychopharmacology (Council for Accreditation of Counseling and Related Educational Programs [CACREP], 2001; King & Anderson, 2004; Ponterotto, 1985) without also a need to examine critically the assumptions on which the support is based. Moreover, there does not appear to be a discussion about the long-term legal, ethical and practical impact on the profession for aligning counselors with psychopharmacology, with even a growing number of psychiatrists becoming alarmed (Glasser & Carlson, 2005).

Breggin and Cohen (1999) wrote, "Biochemical imbalances are the only diseases spread by the word of mouth" (p. 6). Neuroscientist Elliot Valenstein wrote, "Although it is often stated with great confidence that depressed people have a serotonin or norepinephrine deficiency, the evidence actually contradicts these claims" (p. 292). Likewise, Horgan (1999) stated, "Given the ubiquity of a neurotransmitter such as serotonin and the multiplicity of its functions, it is as meaningless to implicate it in depression as it is to implicate blood" (p. 336). Even Peter Kramer, who is often credited with popularizing the antidepressant Prozac with his book *Listening to Prozac* (1994), has since stated in an editorial in the *New York Times* that "...the theories of brain functioning that led to the development of Prozac must be wrong or incomplete" (p. 8, 2002).

The ability to link depression to any specific neurotransmitter like serotonin is currently impossible, particularly given that 95% of serotonin is found *outside* the brain within gastrointestinal system (Glenmullen, 2000). Even if there were evidence of neuropathology, there is no evidence that scientists can study the actual *en vivo* transmission of neurotransmitters between neuronal synapses (Waters, 2006). Therefore, it is safe to assume that psychiatrists cannot identify defective synapses and design medications that will only affect those defected synapses while leaving normal cells unharmed. Moreover, medical profession-

als have not developed normal ranges for various neurotransmitter concentrations to which laboratory tests could be compared if they existed (Valenstein, 1998).

Despite these impossibilities and lack of clear evidence, the chemical imbalance assumption prevails. Lacasse and Leo (2005) appropriately called the pharmaceutical's line of reasoning *ex juvantibus* (i.e., reasoning 'backwards' to make assumptions about disease causation based on the response of the disease to a treatment). An example of this line of reasoning is Horst's (1990) statement: "It is suggested that the organic basis for anxiety and depression may be clarified by *learning the mechanism of medication action* [*italics added*]" (p. 634). Similar *ex juvantibus* reasoning would conclude that shy people suffer from alcohol deficiency because they become extroverted when intoxicated. Although alcohol was used for treating psychiatric disorders in the 1800s, counselors likely would not recommend it as a treatment of choice or even in conjunction with psychotherapy today.

Psychopharmacologists, despite speculation among some psychiatrists (Sherman, 2001), attribute and simplify matters of the psyche and soul to chemical and electrical interchanges in the brain by claiming excessive or deficient neurotransmitters. Physiological psychiatrists have yet to produce a single independent test to measure such biological irregularities ("Study," 2006) as is done with diabetes, which mental disorders are often inappropriately compared (see National Association for the Mentally Ill [NAMI], 1997). Suppose that there is a chemical irregularity, how can a client know exactly? What parameters are there to indicate to clients that they have a chemical imbalance? What independent proof does one have that the antidepressant has "balanced" the brain chemicals thus permitting the client to discontinue the medication? No such safety mechanisms currently exist. It is left to behavioral checklists, such as the *DSM-IV-TR*, and a professional's philosophy to determine if someone has mental disorder warranting a chemical agent with no independent proof that that agent is "correcting" any abnormality (Cosgrove, Krinsky, Vijayaraghavan, & Schneider, 2006).

The indiscriminate medicating of clients with chemical agents without clear biological tests to confirm diagnoses is particularly disconcerting, in light of the potential side-effects, given that 80% of the response to antidepressant medication is duplicated in placebo control groups (Kirsch, Moore, Scoboria, & Nicholls, 2002), and that 57% of pharmaceutical studies fail to show a significant difference between placebo and medication treatment (Kirsch, Scoboria, & Moore, 2002). Much of this disconfirming data has only become available through petitioning the FDA using the Freedom of Information Act (Kirsch, 2005). Glenmullen (2005) advised that antidepressants have too many side-effects and too little supporting evidence to support their widespread use.

Antidepressants and Neurological Damage

Ponterotto (1985) claimed that “antidepressants rarely produce extrapyramidal side-effects” (p. 112). As discussed with antipsychotics, extrapyramidal side-effects are irreversible movement disorders (e.g., parkinsonism, akathisia, acute dystonic reactions and tardive dyskinesia) related to dysfunction of the extrapyramidal motor system (Kaplan & Sadock, 1991).

Leo (1996) examined cases of extrapyramidal symptoms (EPS) within the literature and found that akathisia was the most common (45%), followed by dystonia (28%), parkinsonism (14%), and tardive dyskinesia (11%). Leo reported that his analysis was made difficult because of a lack of consistency in how EPS was defined (see Leo [2001] for a description of the pathophysiology of EPS).

The extrapyramidal side-effect of antidepressants that is noteworthy is akathisia and its association with the increased risk of suicide. Akathisia is a type of EPS characterized by restlessness and severe agitation whereby the client is compelled to move constantly (Kaplan & Sadock, 1991). As discussed earlier, the FDA recently issued a black-box warning for antidepressants to warn clients of an increased risk of suicide. Although Jackson (2005) describes in detail the pathophysiology of antidepressants and akathisia, she summarized the relationship with the following: akathisia is associated with increased risks of suicide and violence; antidepressants are linked with akathisia; therefore, antidepressants can trigger suicide and violence in clients. It is with hindsight that one can say EPS is more of concern than Ponterotto (1985) could have possibly known.

ANTI-ANXIETY (ANXIOLYTIC) MEDICATIONS

Anxiety-related symptoms are currently the most frequently seen problem among mental health professionals, even more than depression (NIMH, 2001). The benzodiazepines continue to be prescribed frequently for anxiety disorders just as they were when Ponterotto (1985) wrote his article. Benzodiazepines (e.g., Valium [diazepam], Xanax [alprazolam], and Ativan [lorazepam]) have in large part replaced the use of barbiturates and antihistamines that were discussed by Ponterotto (1985); therefore barbiturates and antihistamines are not discussed here. Buspar (buspirone) is a non-benzodiazepines that has also been approved for the treatment of Generalized Anxiety Disorder (Fuller & Sajatovic, 2001). In addition, SSRIs have been used increasingly for the treatment of anxiety disorders (see concerns about SSRIs above; Preston, O’Neal, & Talaga, 2000). A number of antipsychotic (see concerns about antipsychotics above) and anticonvulsive medications (see Lithium Salts below for more information) also have been used off-label (Hitchens, 2003). Across these various treatments, it is estimated that 70% of clients with anxiety disorders will

experience relapse (or withdrawal reaction) after discontinuing their medication (Preston, O'Neal, & Talaga, 2000).

In this section, I focus primarily on the benzodiazepines. In particular, I address Ponterotto's (1985) claims about benzodiazepines, which include the following:

1. Benzodiazepines are relatively safe and effective.
2. Benzodiazepines are non-addictive (if used appropriately).

Safety and Effectiveness

The safety issues of benzodiazepines are manifold. As discussed above, any estimation of side-effects are likely to be incomplete given that there is no mandatory requirement for physicians to report medication induced side-effects. Breggin and Cohen (1999) cited Maxmen and Ward's (1995) analysis of medication-induced and neurological disorder occurrence caused by benzodiazepine: confusion and disorientation (6.9%); hallucinations (5.5%); anxiety and nervousness (4.1%); depression (8.3%); and irritability, hostility, and anger (5.5%). Manic side-effect has been particularly associated with the use of Xanax (alprazolam) and Ativan (lorazepam). This side-effect may inadvertently lead to a misdiagnosis of Bipolar I disorder in a similar way discussed with the use of antidepressants (Breggin & Cohen, 1999; Monterrey-Yanes, 1998), which may lead to an additional prescription for anticonvulsants to treat this side-effect.

Similar to the recent FDA warnings regarding antidepressant use and newborns (FDA, 2005b), Walling (2000) and others (Oberlander, Misri, Fitzgerald, Kostaras, Rurak, & Riggs, 2004) reported that benzodiazepine use among pregnant mothers results in serious side-effects. Walling also reported an increase risk of oral cleft lesions of the fetus when used in the first trimester and reported that withdrawal symptoms can occur in neonates in late pregnancy usage. Additionally, the use of SSRIs in conjunction with benzodiazepines have demonstrated an increased risk of fetal deformities (Oberlander et al., 2004).

Non-benzodiazepines have produced similar concerns (Breggin & Cohen, 1999). These medications share similar properties as benzodiazepines and antipsychotic medications while also having different pharmacological action (Long, 2005). Breggin and Cohen (1999) reported the following symptoms associated with the non-benzodiazepine, Buspar (buspirone): headaches, dizziness, and nausea, along with tension or anxiety, abnormal dreams, delirium, and psychotic mania. In addition, since Buspar binds to central dopaminergic receptors, there is an increased chance of neurological damage (e.g., dystonia, parkinsonism, akathisia, and tardive dyskinesia; Long, 2005).

Moreover, the use of medications like the benzodiazepines may actually decrease the effectiveness of psychotherapy. Finn (2001) found that clients with anxiety disorders (panic disorder, social phobia, and generalized anxiety disorder)

der) who were medicated with benzodiazepines (clonazepam, and alprazolam) retained less information, delivered as part of cognitive-behavioral therapy program, compared to clients with anxiety disorders who were not medicated. Finn concluded that benzodiazepines can contribute to significant cognitive impairment resulting in reduced psychotherapy effectiveness.

Addictive Quality

Psychopharmacological authors have begun to select various names to label the addictive properties of psychiatric medication, including benzodiazepines. The current label of choice is *discontinuation syndrome* (Jackson, 2005), which is apparently perceived as less stigmatizing by researchers than addiction or withdrawal. For example, Chouinard (2004) listed discontinuation syndrome as one of the side-effects of benzodiazepines along with dependence, rebound anxiety, and memory impairment. Psychiatrist Carl Salzman (1998) distinguishes benzodiazepines' addictive properties from street drugs by stating that addiction and dependence are not one and the same. Although technically correct, in that dependence indicates a pharmacological phenomenon, they are not separate issues entirely. Salzman proposed that addictions only occur when drugs are taken for non-medical purposes, for the purpose of achieving a pleasure response, and usually as a part of a polysubstance pattern. However, counselors and addiction specialists alike report that doctor-prescribed-medications have become addicting to their clients (DuPont & DuPont, 1998).

An additional concern regarding benzodiazepines is their strong withdrawal effect on clients, which is the case with any medication that affects the GABA [γ (gamma) aminobutyric acid] systems. For example, DuPont and DuPont (1998) reported an increase in seizures associated with the discontinuation of benzodiazepines. These authors also reported a risk of misuse and abuse when clients increase their dosages without the supervision of their physician leading to greater difficulty when the client discontinues the medication. When this occurs, the client may require in-patient treatment (DuPont & DuPont, 1998). In fact, the Drug Abuse Warning Network (DAWN), operated by the Substance Abuse and Mental Health Services Administration (SAMHSA), reported that 19% of those seeking detoxification services in 2003 sought the service for benzodiazepine dependence (U.S. Department of Health and Human Services, 2003). Moreover, benzodiazepine misuse or abuse accounted for 17% of emergency room visits in 2003. Benzodiazepines and antidepressant use was related to 45% of the suicide attempts in 2003 (U.S. Department of Health and Human Services, 2003).

LITHIUM SALTS (MOOD STABILIZERS)

Mood stabilizer use for Bipolar I, II, Bipolar NOS, and Cyclothymia has grown in popularity recently (Healy, 2006). Many clients are told that they will

need to take these medications for the rest of their lives as part of maintenance therapy (Preston, O'Neal, Talaga, 2000), but there is little evidence to support this and no evidence to support prophylactic use for Bipolar II, Bipolar NOS, and Cyclothymia (Healy, 2006). In addition Preston, O'Neal, Talaga (2000) wrote regarding Bipolar Disorder, "Medication-free periods are seldom beneficial and often result in symptom relapse" (p. 167). However, others suspect that the issue of symptom relapse may be attributed to withdraw-induced decompensation (Healy, 2006).

Lithium, or lithium carbonate, the first medication popularly used to treat Bipolar disorder is a natural compound that acts as a mood stabilizer. Lithium's therapeutic dose is very close to the toxic dose and thus requires blood tests to insure safety. In this section, I address Ponterotto's (1985) comments about lithium and include information about the newer mood stabilizers—the anti-convulsants. Ponterotto's comments of note include the following:

1. Lithium can be administered concomitantly with other psychotropic agents.
2. Lithium produces no anticholinergic, sedative, or stimulating effects.
3. Clients on lithium rarely complain of side-effects or of feeling "medicated."

Lithium and Concomitant Psychotropic Use

Much has changed since Ponterotto's (1985) article. Psychopharmacology has produced a whole host of psychotropic agents. Many of these agents are contraindicated for concomitant use with lithium. Fuller and Sajatovic (2001) reported the following dangers of lithium combined with other psychotropic medications: tricyclic antidepressants, SSRIs, and Haldol (haloperidol) use with lithium increases the risk of neurotoxicity; concomitant Thorazine (chlorpromazine) use may increase serum concentrations of both medications; and concomitant use of MAOIs increase the risk of fatal malignant hyperpyrexia. As with any medication, careful consideration must be made when medications are combined with other agents. Depakote (valproic acid), originally approved for seizures, is an anticonvulsant medication for the treatment of Bipolar Disorder that has similar concerns (Breggin & Cohen, 1999).

Anticholinergic, Sedating, or Stimulating Effects

In order to address Ponterotto's (1985) specific points, I address anticholinergic, sedating, or stimulating effects individually.

Anticholinergic Effect

Anticholinergic effects refer to symptoms such as dry mouth and blurred vision due to the blockade of cholinergic nerves. Contrary to Ponterotto's (1985) claim, Hoencamp, Haffmans, and Dijken (1994) specifically linked anti-

cholinergic effects with lithium in a double-blind study of refractory depressed out-patients.

Sedation

A medication's sedation potential can impact various affective, behavioral, and cognitive functions. Breggin and Cohen (1999) reported that lithium and other similar medications (e.g., Depakote) "smooth out" clients by "suppress[ing] the normal electrical transmission of brain cells, limiting the individual's capacity to feel or to react. Lithium literally replaces basic elements in the brain's electrical transmission system, including sodium and potassium ions, thereby slowing down nerve conduction" (p. 36). Blumberg and colleagues (2005) found preliminary evidence that the use of these medications affected the emotional centers of the brains of clients diagnosed with bipolar disorder. Fuller and Sajatovic (2001) reported that 18% to 30% of clients prescribed Depakote (valproate) experience somnolence. Bourgeois (2005) reported an increased risk of cognitive disorders, especially delirium in the elderly, with lithium medication use. Likewise, Elliot (1986) reported a decline of cognitive function following lithium administration for restraint purposes, to the already cognitively compromised. Bell and colleagues (2005) examined the effects of lithium and Depakote (valproate) on cognitive functioning and found that these two medications cause a range of cognitive impairments in healthy participants.

Stimulating Effect

Breggin and Cohen (1999) reported that all mood stabilizers cause some form of sedation, which researchers reframe as a clinical benefit. However, different clients will react differently to brain destabilizing medications in that some clients may experience a stimulating effect. For example, Fuller and Sajatovic (2001) reported that 13% to 18% of clients on Depakote (valproate) experience dizziness; 9% to 15% experience insomnia; and 7% to 11% experience increased agitation. The aforementioned reactions are notably less than those associated with the sedation effect of Depakote (valproate).

UNDERSTANDING PSYCHOPHARMACOLOGY RESEARCH

The challenge of understanding psychopharmacology research is manifold. First, CACREP accredited counseling programs do not require a foundation in behavioral neuroscience. Behavioral neuroscience education would allow counselors to understand, in part, the biological implications of psychopharmacology research (CACREP, 2001; Ingersoll, 2000). Second, the pharmaceutical industry plays a major role in how the dissemination of pharmacological infor-

mation is delivered (or not delivered) to physicians. Information is often provided along with many gifts, lunches, and “free seminars,” all of which have been shown to influence physician prescribing practices (Wazana, 2000). Third, the ability to find literature touting the benefits of psychopharmacology is effortless. Pharmaceutical advertisements are found in most forms of media: magazines, Internet, television, professional journals, and pamphlets located at the doctor’s office. Therefore, it is up to the counselor to take exceptional effort to read broadly on the topic of psychopharmacology in order to obtain a comprehensive view.

In this section, I summarize problems associated psychopharmacology research, psychopharmacology’s influence, and how confusing it can be for counselors who perceive benefits as a result of their personal and clinical experiences. The psychopharmaceutical literature has a number of questionable methodological concerns that once understood can be easily noticed when reviewing such literature (Jackson, 2005). For example, psychiatrist Daniel Safer (2002) of John Hopkins University outlined in detail how the pharmaceutical-industry-sponsored-research findings are modified to support their medications for financial benefit. In fact, in a recent analysis of clinical medication trials in psychiatry, Perlis and colleagues (2005) found that the randomized, double-blind, placebo-controlled studies examined were 4.9 times more likely to report positive results when a conflict of interest was reported (i.e., research funding was provided by the pharmaceutical industry), and that 60% of studies report conflicts of interest. Perlis and colleagues’ findings suggest that the existing literature is biased in favor of the pharmaceutical industry when authors rely on funding provided by the industry.

An additional problem with pharmaceutical research, and research in general, is publication bias as it relates to the non-publication of studies that find negative results (Shields, 2000). Shields (2000) wrote, “As many as 50% of studies may not be published in a particular area of research. Importantly, there is more than a two-fold likelihood that statistically nonsignificant studies (null studies) will not be published or communicated” (p.771). Furthermore, despite the development of research guidelines established to encourage the publication of negative findings (Wager, Field, & Grossman, 2003), only six out of 75 pharmaceutical manufacturers endorsed these guidelines; endorsement still does not guarantee compliance (Singh, 2003). In addition, researchers who receive industry-sponsored funding are generally required to sign a non-disclosure agreement, which is often enforced when non-significant findings are found. The non-disclosure of negative findings from industry-sponsored research, which produces the bulk of pharmaceutical research as part of the process to obtain FDA approval (Jackson, 2005), may be purposeful and make decision making difficult by professionals and consumers alike (Kirsch, 2005).

Additional methodological modifications used by pharmaceutical

researchers reported by Safer (2002) include (1) pharmaceutical researchers comparing newer medications to unusually high dosages of older medications, causing the older medications to have significantly more side-effects and thus making the newer medication appear safer; (2) pharmaceutical researchers administering a number of self-report measurements but only publishing data on those measurements that support the researchers' position; (3) pharmaceutical researchers increasing rapidly the dosages of the competitor's medication to induce more severe side-effects compared to the researchers' medication; (4) pharmaceutical researchers masking unfavorable side-effects or not asking about certain side-effects (e.g., sexual dysfunction) and reporting only data that were disclosed voluntarily by the client; (5) pharmaceutical researchers publishing the same data in different journals to increase the appearance of empirical support; (6) pharmaceutical researchers publishing articles using ghost writers employed by the pharmaceutical manufacturers; and (7) pharmaceutical researchers omitting negative information in the abstract that is in the manuscript to capitalize on the fact that professionals are busy and may only read the abstracts.

Additional problems arise with how physicians are educated about medications. Counselors and clients alike would hope that medical students, for example, would graduate without undue influence of the pharmaceutical industry. Unfortunately, this is not so. The pharmaceutical industry now plays a major role in the education of medical students through the use of industry-sponsored lectures and lunches (Brodkey, 2005). This influence by the pharmaceutical industry has shown to have an effect on the practicing behaviors of physicians in that they are more likely to prescribe newer, more expensive medications recently marketed to them than older, less expensive medications with similar pharmaceutical profiles (Wazana, 2001).

Given the pervasiveness of psychopharmacology in the national understanding of mental health, counselors may accept what is handed to them because they have little access to disconfirming data. Moreover, counselors may also feel pressured to adopt the medical model for financial reasons (Hansen, 2005). The pressure for a share of the market and need for professional recognition may be what has promoted the medical model within the counseling profession and the demise of the counseling profession's historical values (Hansen). However, counselors must be aware that any perpetuation of the chemical imbalance theory may actually inadvertently decrease self-reflection and personal efficacy and increase suffering and ignorance (Lachter, 2001).

The acceptance of biological explanations of mental disorders and the pursuant medication occurs with little consideration for the long-term impact on the psychology of the client and the integrity of the counseling profession. Robert Whitaker, investigative journalist and author of *Mad in America* (2002), stated, "The drug companies are setting forth an unrealistic vision of what it

means to be human. They're defining normal stresses and worries as pathological, and the only reason they're doing it is because it leads to more business" (Willams, 2005, para. 6). Moreover, Jackson (2005) reported that the current influence of the psychiatric and psychopharmacological establishment has made the study of "non-pharmacological management of psychiatric conditions" (p. 181) more difficult by writing standard treatment protocols that emphasize pharmacological interventions.

Counselors may be confused by the premise of this article because of their personal experience of seeing clients who improved using various brain medications. *My concern is that counselors may confuse benefit with the suppressing-effect of the medication.* Psychotropic medications, by their very definition, disrupt and suppress normal brain functioning within the cortex as suggested by research cited in this article. I agree with Glasser (2003) who acknowledged that the symptoms listed in the *DSM-IV-TR* (2000) are real, but to claim that they have a biological cause due to some defect and that the medication is correcting that defect would be inaccurate and misleading given the current state of the science in this area.

Moreover, Glasser (2003), as well as other psychiatrists and counselors (e.g., Breggin, 1991; Burstow, 2005; Dorman, 2003) claim that psychiatric symptoms are a function of one's attempt to deal with stressful life events. Such coping strategies develop within the cortex. Clients are less able to execute these strategies when the cortex is suppressed chemically by pharmaceuticals therefore giving the illusion that the medication "cured" the client (Dorman, 2003). When medication is discontinued, clients often experience withdrawal symptoms and/or a return of the psychological symptoms for which they requested treatment originally. Either way, the problems, or the inner subjective experience of the client, have gone unchanged and unexamined (Hansen, 2005).

SUGGESTED GUIDELINES

Given the large number of severe side-effects (e.g., neurological damage and withdrawal syndromes) and questionable methodological characteristics of pharmaceutical research, counselors may have concerns about supporting the use of medications for disorders that have not been shown to have specific biological etiologies borne out of neuropathological, neurochemical, or genetic defects. To help counselors who do not advocate the use of psychotropic medications, Breggin and Cohen offered the following suggested guidelines for counselors (1999, pp. 198–201):

1. Inform your clients about the prevailing biopsychiatric viewpoint.
2. Clarify the reasons for which you do not professionally agree with or encourage the use of medication.

3. Recommend consultations and readings from both viewpoints.
4. Do not pressure your clients to go along with your particular philosophy of therapy.
5. Avoid making referrals for psychiatric medications if you believe they will not be helpful.
6. Unless they have been taking medications for a very short time, always warn clients about the dangers of abruptly stopping any psychiatric medication.
7. If you have knowledge about adverse effects, share it with your clients.
8. If you are a nonmedical counselor with clients who want to withdraw from psychiatric medications, refer them to a physician who will manage potential side-effects and prescribe lower doses that allow the clients to wean off successfully.
9. If your clients are favorably inclined, consider involving their families, friends, and other resources [during the withdrawal process].
10. If the counseling is not going well, and cannot be fixed, refer the client to another counselor rather than encouraging the use of psychiatric medications.
11. Make notes in your counseling record to indicate that you have had conversations with your clients about medications.

Psychiatrist Paul Schaefer (2003) also offered guidelines for nonmedical counselors. He recommended that counselors always question the diagnoses of clients who are discharged from psychiatric facilities. Schaefer has suggested that recently discharged clients have a new psychiatric evaluation with a psychiatrist who can develop a relationship with the client and understand the symptoms within the context of the family. Furthermore, counselors are encouraged to develop strong skills in the use of the *DSM-IV-TR* (2000) by using published casebooks that teach it, because all psychopharmaceutical research is developed around the symptom clusters described in the *DSM-IV-TR* (2000). Finally, as reflected in this article, counselors should become familiar with the classes of medications described herein in order to “interface with the medical establishment and the insurance reviewer with greater ability to challenge the denial of service” (p. 156).

Counselors may have legal and ethical concerns about discussing medication use with their clients. Ingersoll (2000) wrote that there are “no clear prohibitions against a nonmedical mental health professional talking with clients about psychotropic medications” (Are There Legal-Ethical Problems with Counselors Talking to Clients about Psychotropic Medication section, para. 1). Counselors are advised not to make specific recommendations about the use or nonuse of particular medications. However, counselors are encouraged to know the cur-

rent medications that clients are prescribed so that side-effects can be monitored and reported when necessary.

There are a number of books by psychiatrists and neuroscientists that are written for the layperson and the professional alike. When appropriate, counselors can refer clients to such information, and then clients can discuss questions with their prescribing professional. I contend that it is best to have a cooperative relationship with a medical professional who supports the counselor's position as well. Finally, clients should always be monitored by a physician when they choose to withdraw from medication.

CONCLUSION

As indicated through the comparison of Ponterotto's (1985) article with the psychopharmaceutical literature today, there is little doubt that the role of psychopharmacology continues to be debated, both within the professional realm of counselors and in the personal lives of clients. It is only with a 20-year retrospective that such a comparison can be made. Although the tone of this article may sound anti-medication or anti-pharmaceutical given the prevailing framework within the profession, my objective is to provide counselors with a review of disconfirming evidence voiced by psychiatrists and neuroscientists while using more recent research to illustrate these points. Moreover, a secondary goal is to provide counselors with guidelines for interacting with their clients vis-à-vis psychotropic use and suggest recommendations for training and continuing education for counselors. With the call from academics and CACREP to learn about and make referrals for psychotropic medications (CACREP, 2001; Ingersoll, 2000), this article encourages counselors to get a balanced view about psychopharmacology and the medical-model in general (Lacasse & Gomery, 2003; Liburd & Rothblum, 1995).

It is my belief that the counseling profession must reject the allure of simple biological explanations for human behavior and return to the roots from which our profession grew that honors the human within a context (Hansen, 2005; Liburd & Rothblum, 1995). A biological view must be included in the overall understanding of mental health with the acknowledgment that it is incomplete by itself (Prosky & Keith, 2003). *More importantly, the counseling profession must be cautious about supporting the psychiatric-medical model, or any model, when it is not prepared to produce its own body of research to test the assumptions of that model.*

Finally, counselors must examine the consequences and the impact of associating with and imposing particular assumptions about the biological etiology of mental disorders on clients without evidence that such approach serves their best interest (see Lachter, 2001; Read & Harré, 2001). For example, psychiatrist Daniel Dorman, assistant professor at the University of Los Angeles School of Medicine, wrote, "Declaring that someone has a disease of the mind

that requires treatment with medications is to tell her she has a permanent and profound flaw, that she will never join humanity” (p. 63). The counseling profession was built on confirming in clients the opposite: that their pain is real, understandable, and most of all, that they are not broken and in need of fixing, but remain wholly connected in humanity. It is through that connection to humanity that counselors promote the healing power of relationships and walk with their clients out of their darkness. Reclaiming this healing power that is so closely tied to our heritage and rejecting the medicalization of the counseling profession is paramount for the future of counseling to remain true to its founding principles.

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