

AN ANALYSIS OF PSYCHOLOGIST POSTDOCTORAL  
PSYCHOPHARMACOLOGY TRAINING MATERIALS  
FOR CRITIQUES OF NEUROBIOLOGICAL HYPOTHESES OF DEPRESSION'S  
ETIOLOGY, CRITICAL ANALYSES OF THE DSM'S RIGOR, AND FOR  
CONSUMER/SURVIVOR/EX-PATIENT CONTENT

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Christopher Rowe

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AN ANALYSIS OF PSYCHOLOGIST POSTDOCTORAL  
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DOCTOR OF PSYCHOLOGY

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July 27, 2016

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## ABSTRACT

AN ANALYSIS OF PSYCHOLOGIST POSTDOCTORAL  
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There is widespread agreement that neurobiology plays a role in psychological distress and that psychiatric diagnosis and associated psychopharmacological interventions can be helpful. However, there are also unresolved issues surrounding the limits of empirical support for current diagnostic criteria, shortcomings in neurobiological explanations of psychopathology, and unanswered questions about the mechanism, safety, and efficacy of psychiatric medications. This has implications for treatment errors which can precipitate negative socio-economic and health consequences, particularly for vulnerable groups like the Consumer/Survivor/Ex-Patient (c/s/x) population. It is for these reasons that the training psychologists receive to prescribe should, in addition to integrating the critiques of conventional thinking about the etiology and diagnosis of mental distress, discuss the c/s/x movement. Extant research on psychologist postdoctoral psychopharmacology training has focused on legitimacy, safety, feasibility, and training considerations. This

study used content analysis and was the first to examine a selection of psychologist postdoctoral psychopharmacology training materials to understand the extent to which they were (a) integrating critiques of neurobiological hypotheses for depression's etiology, (b) challenging the rigor of the Diagnostic and Statistical Manual of Mental Disorders (DSM) and, (c) informing students about the consumer/survivor/ex-patient movement. Results indicated that the examined body of materials did not consistently and comprehensively critique the majority of neurobiologically based etiological hypotheses for depression that were being disseminated. Next, challenges to the DSM's empirical rigor within the examined materials primarily focused on construct validity versus inter-rater reliability, and without the provision of statistical analyses. Finally, the only substantial c/s/x content within the examined materials was limited to one book which students were not required to read. The limits of these findings and a variety of socio-cultural, ethical, legal, and professional advocacy considerations are discussed. The electronic version of this dissertation is at AURA: Antioch University Repository and Archive, <http://aura.antioch.edu/> and OhioLINK ETD Center, <https://etd.ohiolink.edu>

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## Chapter I: Introduction

### Background

A compelling body of scientific evidence points to a variety of plausible neurobiological explanations for the etiology of psychological distress and the potential benefits of psychotropic drug interventions (Julien, Advokat, & Comaty, 2011; Schatzberg & Nemeroff, 2009; Stahl, 2013). The value of using formal diagnostic criteria to conceptualize, research, and discuss mental health problems in addition to aiding in the selection of psychopharmacological treatments has also been well documented (American Psychiatric Association, 2000; Fischer, 2012; Nemeroff et al., 2013).

Conversely, there are unresolved issues surrounding the limits of empirical support for current diagnostic criteria, shortcomings in neurobiological explanations of psychopathology, and unanswered questions about the mechanism, safety, and efficacy of psychiatric medications (Moncrieff, 2009; Whitaker, 2010). This has implications for treatment errors which can precipitate negative socio-economic and health consequences, particularly for vulnerable groups like the Consumer/Survivor/Ex-Patient (c/s/x) population (Chamberlin, 1990; Kallert, Mezzich, & Monahan, 2011; LeFrancois et al., 2013; Whitaker, 2010). It is for these reasons that the training psychologists receive to prescribe should, in addition to integrating the critiques of conventional thinking about the etiology and diagnosis of mental distress, discuss the c/s/x movement.

The extent research on the psychologist prescriptive authority movement has focussed on its legitimacy (Evans & Murphy, 1997; Fox et al., 2009; Heiby, 2002; Kirsch, 2003; Sammons, Levant, & Paige, 2003), the feasibility of training, (Tulkin &

Stock, 2004; U. S. General Accounting Office [GAO], 1997, 1999; Vector Research, Inc., 1996), safety considerations (American College of Neuropsychopharmacology, 1998, 2000; Evans, 2003; GAO, 1997, 1999; Lavoie & Fleet, 2002; Newman, Phelps, Sammons, Dunivan, & Cullen, 2000; Robiner et al., 2002; Vector Research, Inc., 1996), and curricula requirements (Levant & Shapiro, 2002; McGrath, 2010; Sammons & Brown, 1997; Resnick, Ax, Fagan, & Nussbaum, 2012; Robiner, et al., 2002; Sechrest & Coan, 2002; Tulkin & Stock, 2004).

This study was the first to examine a selection of psychologist postdoctoral psychopharmacology training materials to understand the extent to which they were integrating critiques of neurobiological hypotheses for depression, challenging the rigor of conventional diagnostic practices, and addressing a particularly vulnerable population with mental health concerns (i.e., the consumer/survivor/ex-patient movement).

I chose to focus on depression's etiology because the disorder is the most commonly diagnosed form of mental distress (Moncrieff, 2009; Whitaker, 2010), with some experts speculating that by the year 2020 depression will be the second leading cause of disability worldwide (Julien et al., 2011). Additionally, neurobiologically based hypotheses for depression are used to support claims about psychiatric medications' mechanism of action as well as the rationale of prescribing this class of drugs (Julien et al., 2011; Schatzberg & Nemeroff, 2009; Stahl, 2013).

Next, as noted, in spite of technological advancements with neuroimaging and associated research, experts continue to disagree about the particular neurophysiological determinants of depression (i.e., the role of neurotransmitters, receptors, neurotrophins, neuropeptides, etc.) and the extent to which these hypotheses support the rationale,

mechanisms, and safety of antidepressants with the potential for serious iatrogenic effects (Andrews, Kornstien, Halberstadt, Gardner, & Neale, 2011; Berton & Nestler, 2006; Healy, 2008; Krishnan & Nestler, 2008; Moncrieff, 2009; Rose & Abi-Rached, 2013; Valenstein, 1998; Whitaker, 2010).

Similarly, substantial limitations with the DSM's inter-rater reliability and construct validity have also been identified (Caplan & Cosgrove, 2004; Frances, 2013; Kirk & Kutchins, 1994; Valenstein, 1998). Additionally, there are concerns that shortcomings of the manual's rigor and potential errors in characterizing the extent and type of psychological distress associated with the DSM's empirical limits might negatively impact employment opportunities, insurance coverage, child custody and other legal proceedings, economic opportunities, and human rights (Caplan, 2012; Caplan & Cosgrove, 2004; Frances, 2013; Kallert et al., 2011; Whitaker, 2010; Zur & Nordmarken, 2010).

The c/s/x population represents a particularly important group to consider within the context of this study because the c/s/x movement was initiated in response to allegations of human rights abuses stemming from psychiatric treatments—treatments that have been based upon questionable scientific evidence. Further, the c/s/x population is especially vulnerable to treatment errors because its members tend to suffer from intersecting forms of discrimination such as poverty, racism, homophobia, and sexism—in addition to the stigmatization of mental illness (LeFrancois et al., 2013).

The c/s/x movement has taken a strong stance against coercive psychiatric practices including forced drugging, forced confinement, and misinformation (i.e., insufficiently informed consent) about the etiology, diagnosis and pharmacological

treatment of mental distress (Campbell & Gardner, as cited in Sammons et al., 2003; Oaks, as cited in Kallert et al., 2011). This has ethical and legal implications for the process of obtaining informed consent for psychiatric services such as diagnosis and psychotropic interventions (Bassman, 2005; Gopal et al., 2012; Gottstein, 2008; Kallert et al., 2011; Mendez, 2013). The importance of conservative prescription practices and informed consent, in light of gaps in the body of etiological and diagnostic evidence used to support psychotropic interventions, is highlighted by a growing body of research documenting the potential for iatrogenic risks associated with antidepressant use (i.e., increased risk of stroke, suicidality, mortality, and complications during pregnancy) (Andrews et al., 2011).

In the United States, forced drugging is an accepted practice for mitigating a person's potential risk of harm to self and/or other(s). However, concerns have been raised by the c/s/x movement and others, in some cases litigiously, about the constitutional legality of these practices which can profoundly impact a person's human rights, physical, and psychological health (Gottstein, 2008; Mendez, 2013; Oaks, 2011).

In the same vein, the American Psychological Association's *Ethical Principles of Psychologists and Code of Conduct* (2010) and *Practice Guidelines for Psychologists' Involvement in Psychopharmacological Issues* (2011) tenets uphold the importance of informed consent that acknowledges the limits of evidence upon which interventions are based. Further these documents outline the importance of a culturally sensitive approach to practice that would dictate being knowledgeable about marginalized groups like the c/s/x population and its movement. For example, the importance of listening to patients' perspectives, developing multi-cultural sensitivity, and obtaining sufficiently informed



consent by reviewing the strengths and limits of evidence supporting diagnoses, etiological hypotheses, and psychotropic treatments (American Psychological Association, 2010, 2011).

In terms of advocacy, professional allies of the c/s/x population (e.g., psychiatrists, psychologists, physicians, social workers, and prescribing psychologists) have identified the importance of considering the history, mission, and needs of the c/s/x movement and population when it comes to the provision of collaborative mental health care practices (MindFreedomInternational, 2015; Sammons et al., 2003).

It is for these social, economical, health, ethical, legal and professional advocacy related reasons that psychologist postdoctoral psychopharmacology training materials warrant examining to determine the extent to which they are integrating information about the c/s/x population and critiques of conventional perspectives about the etiology of depression and rigor of the DSM. The following paragraphs will provide a critical examination of the literature on curricula research and the three topics of analysis in addition to outlining the rationale for the study in greater detail.

### **Critical Review of the Literature**

The following section will critically examine the literature surrounding (a) curricula research, (b) neurobiological hypotheses for the etiology of depression, (c) the rigor of the DSM, and (d) considerations pertaining to the c/x/s population and the c/s/x movement. Additionally, the rationale for this study will be discussed in greater detail.

## Curricula Research

One method for determining how students within a particular discipline are being trained is to examine curricula. According to Douglas Clements (2007) “curriculum is a written instructional blueprint and set of materials for students’ acquisition of certain culturally valued concepts, procedures, intellectual dispositions, and ways of reasoning” (p. 36). An area of focus for curricula research within the discipline of psychology has been diversity—in particular, ethnicity and gender (Dionne & Albanese, 2005; Hogben & Waterman, 1997; Peterson & Kroner, 1992). The American Psychological Association has established task forces to address the issue of multicultural sensitivity within the discipline. For example, the *Report of the APA Task Force on the Implementation of the Multicultural Guidelines* (2008) noted that the discipline of Psychology:

has traditionally been defined by and based upon Anglo Western middle class, Eurocentric perspectives and assumptions. The traditional approaches to psychological research, education and practice have not always considered the influence and impact of culture, race and ethnicity, and their roles in psychological theory, research, and therapy have largely gone unexplored. There has been a growing need to develop a deeper knowledge and awareness of race and ethnicity in psychology and to integrate race and ethnicity into the practice, research, education and ethics of psychology. (Historical Summary para. 1)

To this end, the American Psychological Association’s Division of State, Provincial, and Territorial Affairs, published a Diversity handbook with the goal of involving “more ethnic minority psychologists in membership and leadership positions in State Provincial or Territorial Psychological Associations” (2009b, Diversity Defined para. 4). In the same vein, some of the discipline’s history textbooks have been critiqued for ignoring psychology’s participation within the eugenics movement and in the development and utilization of racially discriminatory testing practices (Cushman, 1995;

Harris, 1997). Additionally, concerns about occupational biases (Firmin, Johnson, & Wikler, 2009) and over-reliance on secondary sources (Devitt, Honts, & Vondergeest, 1997) within psychology textbooks have been raised.

In discussing the impetus for co-authoring the first edition of *Psychology*, social psychologist and feminist Carol Tavris stated that:

Most of all, we wanted to build a book around the principles of critical thinking. We wanted to show students that psychology can teach them how to think better: how to ask questions, how to think about answers and examine the evidence for them, what explanations are possible, what emotional biases we bring to our explanations, and so on. (Shermer, 1999, *The Measure of a Woman*, para. 39)

More recently, Tavris (2004) asserted that the failure to sufficiently address core principles of critical and scientific thinking was “widespread in graduate clinical psychology programs and psychiatric residencies, where students can earn a PhD or an MD without ever having considered the basic epistemological assumptions and methods of their profession” (p. xi). Citing ethnographic research, Tavris (2004) asserted that “rarely do [psychiatric residents] learn to be skeptical about questions, analyze research, or consider alternative explanations or treatments” (p. xi).

The researcher could only find one study examining the degree to which curricula were integrating alternative explanations about psychopharmacotherapy. The study was conducted by social workers Lacasse and Gomory (2003) in response to concerns that their discipline was diverging from its “commitment to exploring non-mainstream positions” (p. 384). The researchers sought to establish whether the 35,000 masters of social work students being trained in the United States at the time were “receiving the best scientific information, evaluation of evidence, and critical analysis currently available on mental health” (p. 384).

Lacasse and Gomory (2003) acquired 71 psychopathology course syllabi from fifty-eight of the top 80 graduate schools of social work listed in the U.S. News and World Report's rankings for the year 2000. The researchers examined the syllabi and required readings for the 2001/2002 academic year and, using content analysis, focused attention on four topics: (a) concepts of mental health issues, (b) the reliability and validity of psychiatric diagnoses, (c) biological etiology, and (d) drug treatment. The study looked at the syllabi's course descriptions, goals and objectives, lecture topics, organization and structure, and required readings to ascertain the degree to which evidence-based critiques of the aforementioned mental health issues were being addressed.

Results indicated that less than half of the syllabi (47.9%,  $n = 34$ ) required students to read an article of any kind—meaning that most of the psychopathology courses relied solely on secondary sources (Lacasse & Gomory, 2003). Next, with regard to the conceptualization of mental disorders, the authors found that the syllabi rarely mentioned any critics of the biomedical model. For example, Lacasse and Gomory noted that Thomas Szasz, a psychiatrist with extensive publications critiquing mainstream psychiatry, was only listed as the author of a required reading in 5.6% ( $n = 4$ ) of syllabi, and placed on the recommended reading list or bibliography within 2.8% ( $n = 2$ ) of the syllabi.

In total, 39.4% ( $n = 28$ ) of the syllabi identified one or more alternative non-medical perspectives (i.e., psychodynamic (31%), ecosystems/family systems (12.7%), cognitive behavioural (9.9%), behavioural (4.2%)) for the etiology of mental distress. The researchers also noted the absence of works by social workers Jerome Wakefield and

Thomas Scheff, well known within the discipline for their non-medical conceptualizations of mental illness.

In terms of content addressing the DSM's empirical rigor, the study found that 87.3% (n = 62) of syllabi did not mention the words "reliability" or "validity" (Lacasse & Gomory, 2003). A more detailed analysis of relevant required readings conducted for 95.8% (n = 69) of the courses, revealed that 28.1% (n = 20) of the syllabi had assigned a textbook which did not address any empirical problems with the DSM-IV. In addition, these courses did not assign any articles addressing this issue.

The required textbook readings for 10 of the syllabi (14.1%) included content on the DSM's reliability and validity (Lacasse & Gomory, 2003). The researchers described this content as "ranging from a short acknowledgment of extant critiques to briefly summarizing the empirical literature" (p. 391). While Lacasse and Gomory noted that 29.6% (n = 21) of the syllabi required one or more readings "which offered some critical discussion relevant to the practice of psychiatric diagnosis," they posited that there was an absence of any empirical data driven approaches to accomplish this (p. 392).

For the biological etiology of depression, Lacasse and Gomory (2003) discovered that 71.8% (n = 51) of courses used materials which supported the chemical imbalance hypothesis for mental distress, while slightly more than one quarter of the syllabi (25.3%, n = 18) "included course goals or descriptions representative of the chemical imbalance theory" (p. 394). The researchers asserted that 43.6% (n = 31) of required textbooks contained "bioreductionistic content" (p. 394). Of the five syllabi (7%) which included readings that critiqued the biomedical model of mental distress—three were recommended and two were required (Lacasse & Gomory, 2003).

The researchers identified “misleading content” in certain textbooks (Lacasse & Gomory, 2003). For example, one text used photographs of positron emission tomography (PET) to differentiate between the brains of people who had been diagnosed with Attention Deficit Hyperactivity Disorder (ADHD) and those whose brains were considered to be healthy. The authors of the text in question attributed these differences to problems with cerebral glucose metabolism resulting in impulsivity—a conclusion with little empirical evidence to support it (Moncrieff, 2009; U.S. Department of Health and Human Services, 1999).

Findings for the fourth topic (psychotropic medications), indicated that less than half (47.9%,  $n = 34$ ) of the syllabi explicitly stated that this subject would be addressed. Twenty-one of the courses (29.6%) that made no mention of medications within the syllabi assigned a textbook with some psychopharmacology content. In total, three syllabi (4.2%) contained a reading which discussed safety concerns surrounding psychotropic drugs; one of these readings was required for one course.

Based on these findings, Lacasse and Gomory (2003) concluded that:

There is little evidence that [social work] graduate psychopathology courses cover viewpoints other than the most conventional and institutional—that of biomedical psychiatry. A small handful of secondary (textbooks) rather than primary (research articles) sources provide the majority of the mental health content in these courses. (p. 383)

The researchers referred to an “apparent one-sided approach” towards psychopathology courses for graduate social work students and posed the following question:

Given the tentativeness of what is ‘known’ about mental disorderliness based on current research, and the emphasis placed, even by mainstream psychiatric experts, on not disregarding alternate perspectives, explanations, and treatments,

many of which are considered strengths of social work, should we not then offer these alternatives for the critical consideration of our students? (Lacasse & Gomory, 2003, pp. 401–402)

While these findings raised concerns about the extent to which the examined social work programs were integrating critiques of conventional perspectives about psychopharmacology, the study had a number of limitations. For example, it utilized a cross sectional design and academic syllabi can change significantly from year to year (Lacasse & Gomory, 2003). In addition, only 58 of the 153 Council on Social Work Education's accredited master's programs were ultimately analyzed. Further, the analyses of the curricula materials were not exhaustive meaning that relevant content may have been missed. Finally, content analysis requires interpretation meaning that results can be impacted by the researcher's subjective biases.

In terms of psychologist postdoctoral psychopharmacology training, members of the medical establishment have questioned the safety of psychologists prescribing and the sufficiency of the associated programs' prerequisites, didactic training, and internships (Heiby, 2002, 2009; Lavoie & Fleet, 2002; McGrath, 2010; Robiner et al., 2002). Additionally, Heiby (2009) suggested that prescriptive authority for psychologists will negatively impact collaborations between psychologists and physicians.

The limited body of research examining the safety of prescriptive authority for psychologists has not identified any risks (American College of Neuropsychopharmacology, 1998; GAO, 1997, 1999; Vector Research, Inc., 1996). However, these findings were based on a small number of subjects ( $n = 10$ ) from a military training facility. Further, the applicability of this data to prescribing psychologists working at community settings with comparably less medical oversight has

been questioned (Robiner et al., 2002). Nonetheless, proponents of the RxP movement have asserted that “hundreds of thousands” of prescriptions have been written by clinicians with prescriptive authority in New Mexico without any serious adverse effects (McGrath, 2010, p. 40).

Research into the feasibility of psychologist postdoctoral psychopharmacology training programs has yielded mixed results. These inconsistencies may be linked to financial variables and assumptions about the extent to which additional psychotropic drug prescribers are needed (GAO, 1997, 1999; Vector Research, Inc., 1996). For example, the GAO studies, whose findings were less positive than the Vector Research Institute’s analysis, included start-up and program evaluation costs within their calculations while the latter study did not. Next, only the GAO studies assumed that the military possessed a sufficient number of psychiatrists—an assertion that has been challenged (McGrath, 2010).

At least four psychologist postdoctoral psychopharmacology training facilities have closed their doors or suspended enrollments since the inception of these programs in the 1980s. As of July, 2016, four programs were in operation (i.e., Alliant International University, Fairleigh Dickinson University, Prescribing Psychologists’ Register, and the University of Hawaii at Hilo). The Southwestern Institute for the Advancement of Psychotherapy (2016) program at New Mexico University was temporarily closed due to reorganization efforts. McGrath noted in 2010 that approximately 1,500 psychologists had completed civilian sector psychologist postdoctoral psychopharmacology training while less than 10% of these individuals were actively prescribing. He concluded that



“For good or ill, then, the ‘truth’ of RxP will be determined politically: either it will win the legislatures or fade away” (p. 43).

Psychologist postdoctoral psychopharmacology training curricula have undergone significant changes since the first iteration of this training was established by the United States military in 1989 (McGrath, 2010; Resnick et al., 2012). For example, the Department of Defence’s Pharmacology Demonstration Project (PDP), which ran from 1991 to 1997, reduced its didactic requirements from 1365 credit hours to 660 credit hours during that period (McGrath, 2010; Sammons & Brown, 1997). These changes stemmed from a decision to omit content that was deemed to be outside the scope of training psychologists to prescribe psychotropic drugs. In comparing the American Psychological Association’s first recommended curriculum for training in psychopharmacology with the Department of Defense’s PDP curriculum, Dunivan and Orabona (1999) noted a “considerable reduction of hours (i.e., biochemistry, human anatomy, health assessment, clinical medicine)” (p. 516).

The American Psychological Association’s first Task Force on Prescription Privileges initially recommended training programs provide 300 hours of didactic coursework. More recently, this was increased to 400 credit hours (American Psychological Association, 1996, 2009a). At the same time, its recommendations for didactic training are still less than 70% of the PDPs maximum (1,365 credit hours) and roughly 40% less than the PDP program’s minimum (660 credit hours) didactic requirements (Sammons & Brown, 1997). Current programs require that students complete between 400 and 450 credit hours.

While the initial American Psychological Association Task Force on Prescription Privileges recommended that applicants demonstrate “knowledge of human biology, anatomy and physiology, biochemistry, neuroanatomy, and psychopharmacology” as “a necessary prerequisite” for postdoctoral prescription training, the Task Force did not specify what this entailed or how it would be regulated (1996, p. 2). To this end, Dr. Robert McGrath, Director for the Postdoctoral M.S. Program in Clinical Psychopharmacology at Fairleigh Dickinson University stated that there were “certain inconsistencies and impracticalities in the requirements of the curriculum,” and noted that “it was ambiguous whether certain elements of the practicum were required or voluntary . . . no program was able to comply with the guidelines exactly” (2010, p. 27). In an apparent effort to resolve these concerns, the APA Task Force on Prescription Privileges ultimately waved prerequisite courses and integrated them within the curricula of psychologist postdoctoral psychopharmacology training programs (American Psychological Association, 2009a; McGrath, 2010).

By comparison, other disciplines have more rigorous enrolment requirements for prescription training. For example, the University of California’s Davis School of Medicine required that Physician Assistant Program applicants first complete classes in human anatomy (with lab), physiology (with lab), general chemistry (with lab), microbiology or bacteriology (with lab), in addition to mathematics and social science courses (University of California, 2015).

Stricter yet were the University of New Mexico’s (2015) College of Pharmacy’s requirements for the Doctor of Pharmacy program which stipulated that applicants first complete courses in general chemistry (8 semester hours with labs), organic chemistry

(8 semester hours with labs), molecular cell biology (4 semester hours), genetics (4 semester hours), microbiology (4 semester hours with lab), anatomy and physiology (6 semester hours), and biochemistry (3 semester credit hours), in addition to classes in microeconomics, calculus, statistics, physics, communication, and critical thinking prior to enrollment.

Internship requirements have also undergone changes since the PDP. For example, while all three iterations of the PDP required and the first RxP task force specifically recommended that students work with at least one hundred clients, this suggestion was dropped. Next, the American Psychological Association's most recent Prescription Privilege Task Force (2009a) recommended that "the trainee gains supervised clinical experience with a sufficient range and number of patients in order to demonstrate threshold performance levels for each of the competency areas" (p. 10) without clarifying the number of clients this would entail.

Clinical psychologist William Robiner and colleagues (2002) questioned the extent to which, "such condensed training overcomes current shortcomings to achieve knowledge and clinical proficiency equivalent to that of other prescribers, especially psychiatrists, and ensure competent prescribing that the public should reasonably expect of its doctors?" (p. 235). Similarly, Heiby (2002) noted that psychologist postdoctoral psychopharmacology curricula involved less formal training in hard sciences than other prescribing professions, including those which require ongoing supervision subsequent to licensure (i.e., nurse practitioners).

Conversely, proponents of RxP noted that psychologist postdoctoral psychopharmacology training programs provided over three times more psychopharmacy

course work than physicians and nurse practitioners (Muse & McGrath, 2009). In response, Stuart and Heiby (2007) argued that educating physicians more effectively in psychopharmacology would be more cost effective than training psychologists to prescribe.

To date, psychologists with appropriate training and credentials can prescribe within New Mexico, Louisiana, Illinois, Iowa, and the territory of Guam (American Psychological Association, 2014, 2016). Psychologists with prescription privileges can also prescribe within the Indian Health Services, United States Public Health Service, and the Department of Defence. However, despite research into the prescriptive authority for psychologists movement's legitimacy, the feasibility and safety of training psychologists to prescribe, and the composition of psychologist postdoctoral psychopharmacology curricula requirements, no studies have analyzed the extent to which these training materials are integrating relevant critical perspectives.

We do not know, for example, the extent to which psychologist postdoctoral psychopharmacology curricula are integrating critiques about neurobiologically based hypotheses of depression, or challenges to the empirical rigor of the DSM. Further, it is unclear whether or not these training materials are making efforts to address the implications that these critiques and associated risks of treatment errors have for the consumer/survivor/ex-patient (c/s/x) population and movement.

### **Neurobiological Etiology of Depression**

Until recently, the monoamine hypothesis was the most commonly cited neurobiological hypothesis for depression (Stahl, 2013). The monoamine hypothesis posits that depression stems from a chemical imbalance within the brain. More

specifically, the premise asserts that reduced levels of serotonin (5HT), noradrenalin (NE), and dopamine (DA) are implicated in depressive symptomatology. Despite initial optimism about this hypothesis, Stahl wrote in 2000 that “So far, there is no clear and convincing evidence that mono-amine deficiency accounts for depression; that is, there is no ‘real’ monoamine deficit” (p. 601).

In Stahl’s *Essential Pharmacology: Neuroscientific Basis and Practical Applications*, the author concluded that research in support of the monoamine hypothesis for depression has “unfortunately yielded mixed and sometimes confusing results” (Stahl, 2013, p. 262; Stahl, 2008, p. 483). Stahl (2013) also noted that studies have not consistently found deficiencies in serotonin (5HT), norepinephrine (NE), and dopamine (DA) metabolites. Additionally, there are concerns that neurotransmitter levels cannot be measured within the blood brain barrier of live human subjects (Lacasse & Gomory, 2003).

The neurotransmitter receptor hypothesis took the chemical imbalance premise a step further suggesting that depleted neurotransmitter levels trigger a homeostatic response involving the up-regulation of postsynaptic neurotransmitter receptors (Stahl, 2013). Evidence for this hypothesis has been mixed. For example, consistent molecular lesions in monoamine receptors have not been found, leading Stahl (2013) to conclude that “There is no clear and convincing evidence that abnormalities in monoamine receptors account for depression” (p. 267). More compelling albeit, inconsistent findings, indicated that polymorphisms in the serotonin 1A receptor gene may be implicated in mood disorders (Albert, 2012; Fisher et al., 2013; Kishi et al., 2012). Investigations into the etiology of depression have also focused on longer-term changes to molecular and

cellular plasticity (Krishnan & Nestler, 2008). Particular attention has been afforded to P11, a calcium binding protein that interacts with serotonin 5-HT1b. Using in-situ-hybridization, researchers found that P11 was down-regulated in depressed individuals' cingulate cortex's (Svenningsson et al., 2006).

In addition, the role of transcription factor Cyclic-AMP-response-element-binding-protein (CREB) has been studied. Findings with rodents suggest that stress activates CREB within the nucleus accumbens, triggering depression-like-responses (Pitinger & Duman, 2008). Conversely, data has shown that antidepressants can precipitate an upregulation of CREB, indicating that changes in neuroplasticity may be site specific or mediated by some other mechanism (Krishnan & Nestler, 2008).

More recently, research has focused on the means by which neurotrophins impact neuroplasticity and mood. This hypothesis stems from pre-clinical data which indicated that stress could inhibit brain-derived neurotrophic factor (BDNF) mediated signaling within the hippocampus, leading to atrophy and apoptosis (Krishnan & Nestler, 2008; Stahl, 2013). Related research has shown that chronic antidepressant treatment can lead to increased BDNF signaling; an outcome that some interpret as further evidence in support of the neurotrophic hypothesis for depression (Duman & Monteggia, 2006; Krishnan & Nestler, 2008).

There is also a substantial amount of research data which contradicts or fails to support these correlations between neurotrophins and mood (Krishnan & Nestler, 2008). For example, depression is not consistently induced by blocking BDNF or neurogenesis in the hippocampus (Airan et al., 2007; Santarelli et al., 2003). Next, Dias and colleagues found that chronic fluoxetine administrations in rodents did not result in significant

impacts to axon-specific BDNF transcription levels (Dias, Banerjee, Dunman, & Vaidya, 2003). Others have found that increased levels of serotonin in the hippocampus actually decreased levels of BDNF (Vaidya, Marek, Aghajanian, & Duman, 1997).

In a review of the BDNF hypothesis for depression, Groves (2007) described the extant data as inconsistent and contradictory and asserted that “Like the monoamine hypothesis proposed over 40 years ago, we may have to accept that the role of BDNF lies more in the genesis of depressive symptoms than at the core of disease pathophysiology” (p. 1085).

Further complicating these interpretations about the volume and density of neural matter within hippocampal and pre-frontal cortex regions are unresolved methodological problems pertaining to the research itself (i.e., co-morbid diagnoses and dissimilar medication histories across subjects) (Krishnan & Nestler, 2008). In addition, antidepressants are capable of iatrogenically altering brain matter. For example, researchers from Duke University found that antidepressant use, especially tricyclic’s, was associated with neuronal white matter degradation among elderly subjects (Steffens et al., 2008). Thus, it is possible that antidepressants might cause some of the neuronal abnormalities observed within neuroimaging studies of depressed individuals’ brains, challenging conventional explanations for these results.

Links between mood and various neuroendocrine and neuroimmune mechanisms have also been linked to depression. For instance, chronic administrations of glucocorticoids in rodents have produced depressive-like behaviours (Gourley et al., 2008). Of related interest are findings that patients with Cushing’s syndrome exhibit

excessively high concentrations of circulating cortisol in conjunction with depressive features and hippocampal atrophy (McEwen, 2007; Nestler et al., 2002).

Interestingly, hypercortisolaemia has been associated with severe depressive symptomatology while hypocortisolaemia was linked to atypical depression. This indicates a potential relationship between glucocorticoid profiles and the severity of mood symptomatology.

Neuromodulators of immunity known as cytokines have been implicated in depression's etiology. In rodents, drug induced release of cytokines interferon- $\alpha$ , tumor necrosis factor- $\alpha$ , and IL-1 $\beta$ , were correlated with social withdrawal and reductions in exploratory and sexual behaviours (Dunn, Swiergiel, & de Beaurepaire, 2005). In the same vein, blocking pro-inflammatory cytokine-mediated signaling or targeted deletions of cytokine mediating genes, has yielded anti-depressant-like effects in rodents (Krishnan & Nestler, 2008). However, direct administrations of interferon- $\alpha$  and IL-1 $\beta$  have not been found to produce depression like symptoms (Dunn et al., 2005). Moreover, human studies have yielded inconsistent data surrounding any relationships between mood and serum cytokine concentrations (Dunn et al., 2005). Further, legitimate concerns have been raised about the validity of using results from animal models of psychological distress to explain the etiology of psychological distress in humans (Rose & Abi-Rached, 2013).

Estrogen has also been shown to have neurobiological impacts on mood levels. Stahl (2008) referred to the hormone as a trimonoaminergic modulator and described its capacity to mediate the activities of gamma-aminobutyric acid (GABA) and glutamate. Additionally, drugs that increase the availability of GABA are typically associated with



feelings of relaxation and well-being (Julien et al., 2011). By proxy, estrogen tends to inhibit GABA activity. Next, age specific rates of depression have been positively correlated with the percentage of estrogen production over the course of a woman's lifespan, with the highest rates for both occurring during childbearing years (Stahl, 2008).

As a result of GABA being inhibited, pyramidal neurons become activated and release glutamate (Stahl, 2008). Disturbances in glutamate metabolism and associated impacts on N-methyl-D-aspartate (NMDA) receptors, as well as trophic changes, were linked to depressive symptomatology and suicidality (Paul & Skolnick, 2003). In addition, it has been established that prolonged excitation of nerve cells can be toxic and atrophic (Julien et al., 2011).

Estrogen also regulates gene production including brain-derived neurotrophic factor (BDNF) (Stahl, 2008). BDNF, as mentioned earlier, is thought to be critical for neurogenesis and preventing atrophy and apoptosis in areas of the brain like the hippocampus. Interestingly, administrations of estradiol (a type of naturally occurring estrogen) have been shown to reduce depressive-like-behaviours in older female mice (Walf & Frye, 2009). Conversely, human research has shown that estroidal was not an effective intervention for low mood in older postmenopausal women (Morrison et al., 2003).

Support for an epigenetic etiology of depression includes the disorder's high discordance rates among monozygotic twins, individual variations in depressive-like behaviors among inbred rodents and higher rates of depression among women (Krishnan & Nestler, 2008). Covalent change alterations of DNA (i.e., methylation), post-translational changes to histone (a highly alkaline protein), and non-transcriptional

silencing mechanisms such as micro RNAs have been noted too (Krishnan & Nestler, 2008).

In addition, epigenetic findings suggest that rats born to less attentive mothers (i.e., less maternal licking and grooming), are more likely to exhibit reduced expressions of glucocorticoid receptors and an associated increase in the methylation of cytosine, a glucocorticoid receptor gene promoter (Tsankova, Renthal, Kumar, & Nestler, 2007). Thus, early neglect may contribute to genetic modifications and subsequent depressive symptomatology. These abnormalities were shown to arise within the first week of life and could, according to the researchers, be mediated by cross-fostering (Tsankova et al., 2007).

Trichostatin A, a histone deacetylase inhibitor (HDAC) has also been shown to reverse the course of methylation and HDAC inhibitors appeared to have antidepressant-like effects on rodents that underwent trials using social defeat assays (Weaver et al., 2004). Interestingly, mice that underwent complete eradications of HDAC5, showed an increased vulnerability to social defeat (Weaver et al., 2004). Also, while the antidepressant drug Imipramine can raise hippocampal HDAC5 levels, the drug also significantly reduces the expression of HDAC5 within the nucleus accumbens (Krishnan & Nestler, 2008). Such outcomes highlight the regional specificity of drug effects and the brain's apparent leanings towards homeostasis.

Additional insights about depression's etiology have come from research into the neurobiological correlates of resiliency. For example, gene expression analyses of rodent brains provided distinct translational profiles consistent with overcoming adversity (i.e., the ventral tegmental area, nucleus accumbens, and hippocampus) (Krishnan & Nestler,

2008). While these profiles suggest that resilience and disorders like depression may stem from specific neurobiological processes, the extent to which these results can be generalized to human beings is unclear.

In an effort to link particular symptoms of depression to “inefficient information processing” within various regions of the brain, Stahl (2013) conceptualized the disorder’s symptomatology via a “symptoms and circuits” model (pp. 273–278). More specifically, Stahl tied the core symptoms of major depressive disorder (weight/appetite changes, insomnia or hypersomnia, psychomotor agitation or retardation, fatigue, feelings of guilt or worthlessness, executive dysfunction, and suicidal ideation) to particular brain circuits within the prefrontal cortex, basal forebrain, striatum, nucleus accumbens, thalamus, hypothalamus, amygdale, hippocampus, brainstem, spinal cord, and cerebellum. Stahl (2013) also linked depressive symptoms to aminergic “dysfunction” (p. 278).

In discussing the rationale for this hypothesis, Stahl asserted that “The trend is from categorical DSM diagnoses to dimensional symptoms matched to circuits in the RDoC [Research Domain Criteria] approach” (S. Stahl, personal communication, November 5, 2014). The National Institute of Mental Health (NIMH) 2008 Strategic Plan noted that

RDoC attempts to bring the power of modern research approaches in genetics, neuroscience, and behavioral science to the problems of mental illness, studied independently from the classification systems by which patients are currently grouped. The approach provides a framework to develop hypotheses and evaluate results from studies that investigate the mechanisms of psychopathology. (NIMH, 2014, Background para. 2)

The NIMH Plan also stated that the “RDoC research starts with basic mechanisms and studies dysfunctions in these systems as a way to understand homogeneous symptom sets that cut across multiple disorders, rather than starting with clinical symptoms and working backwards” (2014, Abstract, para. Methods and Findings). The references to “mechanisms of psychopathology” and “dysfunctions” within the plan suggests that the “systems and circuits” approach to depression is based on certain etiological and epistemological assumptions about genetic and neurobiological correlates for psychological distress (2014). For example, decisions about what types of data to gather (i.e., neurobiological markers), how to accomplish this (i.e., neuroimaging), and how these results are interpreted (i.e., via the medical model).

Genetic research for depression has also focused on a class of genes known as neuropeptides, which have been found to modulate neuronal communication. For example, neuropeptide nerve growth factor (VGF), which is transcribed by cAMP response element binding protein (CREB), was shown to be down-regulated in the hippocampus of rodents undergoing stress inducing activities (learned helplessness and forced swim) (Thakker-Varia et al., 2007; Thakker-Varia & Alder, 2009). Conversely, VGF was found to be upregulated in rodents given antidepressants or engaging in voluntary exercise opportunities (Thakker-varia & Alder, 2009; Thakker-Varia et al., 2007).

Another study investigated the relationship between neuropeptide Y (NPY) and responses to written words (Mickey et al., 2011). These researchers used fMRI to measure how the emotional valence of words (negative or neutral) impacted brain activity among 44 individuals diagnosed with major depressive disorder and 137 healthy controls

(84% had an NPY genotype expression of low, intermediate, or high). Findings indicated that healthy individuals were significantly more likely to exhibit activation of the frontal cortex during exposure to negatively valenced words and that this activity was negatively correlated with the strength of geno-type predicted NPY expression.

The researchers hypothesized that “these genetically influenced neural response patterns” play a role in mitigating risk for certain types of major depressive disorder (Mickey et al., 2011). Ultimately, this data may help to explain individual differences in resilience, emotional experience, and heterogeneous responses to pharmacological interventions for depression. At the same time, these findings are inconclusive and questions remain about the efficacy of using neuropeptides as an antidepressant agent (Thakker-Varia & Alder, 2009).

The aforementioned findings represent a substantive body of research affording a variety of compelling and plausible neurobiologically based hypotheses for the etiology of depression. The advent of neuroimaging with pneumoencephalography in 1918 and subsequent developments with computerized axial tomography (CAT), single photon emission computerized tomography (SPECT), positron emission tomography (PET), magnetic resonance imaging (MRI) and functional magnetic resonance imaging (fMRI), have been critical to the acquisition of this knowledge.

In 2008, Nikos Logothetis completed a database keyword search using the terms, “fMRI,” or “MRI,” or “functional magnetic imaging,” which yielded over 19,000 peer reviewed articles. Roughly two years later, the same search conducted by Rose and Abi-Rached (2010) resulted in 53,662 papers. Neuroimaging technology has permitted scientists to examine and conceptualize the brain in novel ways, facilitating new

hypotheses about cognitive-emotional experiences including mental illness and resilience (Satel & Lilienfeld, 2013). Similarly, human brain injury studies and animal research have provided scientists with opportunities to uncover neural correlates that might predispose us towards mental health or psychiatric illness (Rose & Abi-Rached, 2010). This includes insights about neuroanatomy, neurotransmitters, neurotransmitter receptors, neurotrophins, the hypothalamic-pituitary-adrenal axis, neurogenesis, neuropeptides and the neuroendocrine system, immunological factors, genetics, glutamate, GABA, and enzymes' role in behaviour and well-being.

Nonetheless, efforts to definitively establish which neural mechanisms play a causal role in depression and other psychological disorders have been unsuccessful. More than 15 years ago, Rockville (1999) stated within *Mental Health: A Report From the Surgeon General*, that “there is no definitive lesion, laboratory test, or abnormality in brain tissue that can identify the [mental] illness” (p. 44). This report also noted that the etiology for depression remains “unknown” (p. 49). Similarly, Krishnan and Nestler (2008) highlighted the “enormous gaps in the knowledge of depression and its treatment” (p. 901).

Additionally, legitimate concerns have been raised about the particular research methodologies upon which neurobiological hypotheses of depression have been based. For example, brain scan data used to support claims of irregular regional neural activity in disorders like AD/HD, bipolar disorder, depression, and schizophrenia has been criticized (Rosa & Abi-Rached, 2013).

In discussing the effects of brain lesions and the limits of neuroanatomical reductionism, neuropsychologist Russell Poldrack asserted “Given that many cognitive

processes may be distinguished not by activity in specific regions but by patterns of activity across regions, there is reason for caution regarding many of the inferences that have been driven by highly modular approaches” (as cited in Rosa & Abi-Rached, 2013, p. 76).

Rose and Abi-Rached (2013) questioned whether scientists have ascertained the most appropriate scale for measuring mental processes with fMRI. Further, these authors posited that scanning facilities could impact neuroimaging results:

The scanning facility is not a ‘non-place’ for those who are being scanned, it is a particular and rather unusual arrangement of space, persons, machinery, sounds, and sights, not to mention the experience of being in the scanner itself. What are the subjects thinking and doing when they are in the scanner? The scanned brain is in the human body, the body of a human being who is being paid to lie in the machine to perform a task that the brain will never be confronted with in the real world outside. . . . What is the salience of the image when it emerges from the secluded and simplified world of the laboratory, and makes claims for its relevance to the understanding of conduct in the wild and messy world of ordinary life? (pp.76–77)

In their critical analysis of neuroimaging research, Satel and Lilienfeld (2013) noted that fMRI data represents correlative information about brain activity and prohibits causal conclusions about behaviours or psychological operations. These authors also noted the complexities involved in trying to deconstruct complex mental states and emotions into specific neural correlates—an effort that typically involves regional brain mapping or, more recently, pattern analysis.

Further, the correlation between blood flow and neurobiological activity is complicated by the delay (i.e., 2 to 5 seconds) between neural activation and subsequent increases in blood circulation to those neurons (Satel & Lilienfeld, 2013). Consequently, any rapid changes in neural activity could be missed. While EEG can capture some of

this undetected data, the technique has its own limitations. For example, EEG cannot effectively differentiate between the actions of excitatory and inhibitory neuronal impulses (Halgren & Pashler, as cited in Pashler, 2013).

Statistical methods used to examine neuroimaging findings can be problematic as well. For example, the task of interpreting neuroimaging data is highly complicated and analytic methods lack standardization. This can undermine the reproduction and comparison of neuroimaging studies. Further, when analyses run concurrent statistical tests on the same BOLD signals, the risk of false positive results increases (Satel & Lilienfeld, 2013).

In 2009, reflecting on the impacts that neuroimaging had on clinical practice over the past 20 years, Thomas Insel, Director of the NIMH, stated that:

what promise have we realized in the diagnosis and treatment of individuals with serious mental illness? In contrast to the steadily decreasing mortality rates of cardiovascular disease, stroke, and cancer, there is no evidence for reduced morbidity or mortality from any mental illness. (pp. 128–129)

Insel (2009) also asserted that:

Despite high expectations, neither genomics nor imaging has yet impacted the diagnosis or treatment of the 45 million Americans with serious or moderate mental illness each year. While we have seen profound progress in research (with molecular, cellular, and systems neuroscience revealing new, unexpected insights about the brain), the gap between the surge in basic biological knowledge and the state of mental health care in this country has not narrowed and may be getting wider. (p. 128)

Behavioral neuroscientist Jacqueline Crawley (2007) expressed concerns about interpreting the data from animal models of human distress. In her textbook, *What's Wrong With My Mouse? Behavioral Phenotyping of Transgenic and Knockout Mice*, Crawley (2007) recommended that researchers “Tread softly when approaching a mouse model of a human psychiatric disease” (p. 227). Crawley also stated, “Don’t



anthropomorphize! Emotions are personal, internal, and highly species specific. There is no way for a human investigator to know whether a mouse is feeling afraid, anxious, or depressed” (p. 261).

In addition, the validity of genetic findings for psychopathology has been challenged (Farber, 1981; Jackson, 1960; Joseph, 2002; Kamin, 1974; Leo, 2003; Leo & Joseph, 2002). For example, in discussing family research from the 1920s, Joseph (2002) asserted that “The ‘evidence’ of [psychiatrist] Myerson’s era consisted of family pedigrees, preconceived notions, and prejudice” (p. 72). Further, with regard to twin studies Joseph noted that:

Problems include the lack of an adequate and consistent definition of the trait under study, non-blinded diagnoses, inadequate or biased methods of zygosity determination (i.e., the determination of whether the twins are MZ or DZ), the unnecessary use of age-correction factors, the use of non-representative sample populations, and the lack of adequate descriptions of the methods used in some of the studies. (Joseph, 2002, p.74)

Joseph (2002) has also challenged the “equal environment assumption” and posited that the environment of monozygotic twins was more similar than for fraternal twins (p. 73). For example, he stated that:

The problem with the traditional EEA definition is that most people—including many prominent twin researchers (Bouchard, 1993; Gottesman, et al., 1972; Kendler et al., 1994; Morris-Yates et al., 1990; Rose, et al., 1990; Scarr, 1968; Scarr et al., 1979)—recognize that it is false since MZs are treated more similarly by their parents and by the social environment, spend more time together, and share a closer emotional bond. (Joseph, 2002, p. 74)

In addition, it was posited that the proband method of analysis can inflate twin concordance rates relative to using the pairwise method (Joseph, 2002; Leo, 2003).

In reviewing Caspi and colleagues' suppositions about the implications of the 5-HTT promoter polymorphism on one's susceptibility for depression, Leo, noted that "68% of the population carries at least one copy of this allele . . . even people without the short form became depressed; serotonin processing has been implicated in numerous DSM-IV conditions; and this allele is most likely involved in many other traits—some of which might be considered beneficial" (Leo, 2003, p. 411).

Concerns also exist about the drug trial publication process (Dubovsky & Dubovsky, 2007; R. Smith, 2005; Turner, Knoepflmacher, & Shapley, 2012; Turner, Matthews, Linardatos, Tell, & Rosenthal, 2008). For example, Dubovsky and Dubovsky (2007) identified problems with ghost writing and the implications of journal articles attributing authorship to individuals who did not actually write the article and/or have any final say about its contents.

Richard Smith (2005), who was employed as an editor for the British Medical Journal for 25 years, noted that industry funded studies typically yielded results favoring the pharmaceutical company which funded them. For example, he described a 1994 study by Rochon and colleagues which analyzed 56 published studies on nonsteroidal anti-inflammatory drugs for arthritis, and noted that none of the published results were unfavorable to the pharmaceutical company which funded the respective trials.

R. Smith (2005) was also concerned about editors' reliance on authors for the provision of relevant studies in reviewing the literature. Further, editors may be faced with a conflict of interest when the decision to publish or decline a study has the potential

to impact a journal's financial viability. In a satirical paper, Sacket and Oxman listed tactics used to inflate drug trial results (i.e., using selective, non systematic reviews to bias support for proposals “invoking fallacious ‘placebo effects’ and ‘assay sensitivity’ arguments in order to avoid head to head comparisons,” manipulating data by selecting surrogate and composite end points that support desired results, adjusting the requirements for “superiority” and “non-superiority,” adding effective co-interventions to the drug being studied but not to the control group using the comparator treatment, unblinded outcome assessments that favor the drug treatment group, initiating sub-group analyses designed to establish statistically significant effects favoring the experimental drug, providing statistics for the relative but not absolute risk reduction and the number needed for treatment, and over interpreting the results of an indeterminate trial (Sackett & Oxman, 2003, p. 1443).

In a study investigating the publication of antidepressant drug trials, Turner (a medical reviewer at the US Food and Drug Administration from 1998 to 2001) and colleagues noted selective publishing of results and outcomes can inflate estimates of efficacy thereby impacting the risk-benefit ratio (Turner et al., 2008). In reviewing 74 FDA-registered studies of 12 antidepressant drugs, involving 12,564 patients, these researchers found that 31% of the examined trials were not published. Conversely, of the 37 studies viewed by the FDA as having positive results, only one did not get published. The article posited that “studies viewed by the FDA as having negative or questionable results were, with three exceptions, either not published (twenty-two studies) or published in a way that, in our opinion, conveyed a positive outcome (eleven studies)” (Turner et al., 2008, p. 252).

Turner and colleagues (2008) suggested a discrepancy between FDA analyses and the published literature on drug trials. For instance, these researchers noted that the FDA had concluded that 51% of antidepressant drug trials were positive while the published literature suggested that 94% of the conducted studies on antidepressant's efficacy were positive (Turner et al., 2008). A more recent study by Turner et al. (2012) examined publication bias in antipsychotic drug trials and found that for the 20 published trials examined "the five that were not positive, according to the FDA, showed some evidence of outcome reporting bias" (2012, Abstract, Methods and Findings).

These researchers noted that the accuracy of their findings was limited by the small number of trials examined (Turner et al., 2012). However, Turner and colleagues (2012) also asserted that "Although the magnitude of the publication bias seen here is less than that seen in a similar study of antidepressant drugs, these findings show how the selective reporting of clinical data undermines the integrity of the evidence base and can deprive clinicians of accurate data on which to base their prescribing decisions" (Turner et al., 2012, Editor's Summary, para. What Do These Findings Mean).

Gaps in the neuroscientific research on depression's etiology mean that treatment errors are possible and that the limits of these research findings used to support neurobiological hypotheses for depression should be considered within the context of training psychologists to prescribe—particularly given the risks that treatment errors pose for patients and especially for marginalized groups like the c/s/x population. Potential health risks of antidepressants will be discussed in detail within the section on the c/s/x movement.

## **Diagnostic and Statistical Manual of Mental Disorders**

This dissertation examined the extent to which psychologist psychopharmacology training materials were integrating critiques of the DSM's empirical rigor because the manual plays such a central, albeit controversial role in contemporary mental health care services within the United States (American Psychiatric Association, 2000, 2013; Frances, 2013). The manual is used to determine the existence of a mental disorder, inform the selection of pharmacological interventions, and clarify treatment needs for insurers. In addition, the DSM aids in establishing prevalence rates for mental disorders and in the selection and assignment of research participants within drug trials. Kirk and Kutchins (1994) stated that the "DSM represents a major way of organizing psychiatric knowledge, research efforts, and treatment approaches" (p. 71). According to Jenkins and Vaida (2007), the diagnosis of psychiatric conditions has "a significant impact on medication selection, dosing, and frequency" (p. 42).

Members of the DSM-IV-TRs task force posited that "more than any other nomenclature of mental disorders, DSM-IV is grounded in empirical evidence" (American Psychiatric Association, 2000, p. xxiv). The DSM-IV-TR's task force was made up of 27 members and integrated materials from 13 work groups. Each work group had five or more members whose reviews were evaluated by up to 100 advisers. Conferences, and workshops, external reviews of drafts, and liaisons with over 60 organizations and associations, all aided in the development of the text's methodological and conceptual frameworks (American Psychiatric Association, 2000). The manual stated that this "systematic and explicit process" was a major innovation of the DSM-IV-TR

(p. xxiv). The manual's authors also described having made an effort to "extract, aggregate, and interpret [data] in a comprehensive and objective fashion" (p. xxvi).

In addition, the task force solicited input from individuals who they anticipated would be critical of the literature reviews used to inform the manual's content (American Psychiatric Association, 2000). Next, data reanalyses and field trials were used to address cases in which evidence was deemed to be insufficient. In total, 12 field trials involving more than 70 sites with over 6,000 subjects were completed. According to the DSM-IV-TR's authors, the primary goal of the text revision was to correct factual errors, reflect new information, enhance the document's educational value, and better align the DSM-IV-TR with the International Classification of Disease and Related Health Problems (ICD-10) manual (American Psychiatric Association, 2000).

The DSM-IV-TR task force acknowledged the limitations of using a categorical classification system. For example, they identified problems with diagnosing "boundary cases" where the criteria for a particular disorder were just shy of being met (American Psychiatric Association, 2000, p. xxxi). To address this, the DSM-IV-TR stressed that diagnosis required a thorough assessment by clinicians with sufficient training.

The matrix of socio-cultural, environmental, biological, and psychological variables impacting mood complicates the creation of a fixed and universal diagnostic system. Critics have argued, for example, that the DSM's nosology is based upon consensus versus objective scientific data (Valenstein, 1998; Whitaker, 2010). Nancy Andreasen, chair of the DSM-IV-TR work group for Schizophrenia, and co-author of *Introductory Textbook of Psychiatry* (3rd ed.) with Donald Black, stated that "The [DSM-IV-TR's] selection of signs and symptoms is often relatively arbitrary" and "they will

remain arbitrary as long as we are ignorant about pathophysiology and etiology” (2001, p. 35).

Michael First, co-chair and editor of the DSM-IV-TR, reiterated Andreasen’s statement in positing that:

DSM is a labeling system that is inherently superficial, and it is a convenient fiction to suppose that patients’ problems can be broken down into discrete categories. We don’t understand the etiology of mental illness and lab findings are practically never found that are diagnostically useful. (as cited in Lacasse & Gomory, 2003, p. 383)

According to Caplan and Cosgrove (2004), diagnostic labels “are defined by whoever does the defining, and the power to make a definition stick usually resides in groups that have the most social, political, and/or economic power” (p. xx). Similarly, in describing how various diagnostic decisions were made during the development of the DSM-III, Michael First stated that “A lot of what’s in the DSM [III] represents what Bob [Robert Spitzer] thinks is right . . . He really saw this as his book, and if he thought it was right he would push very hard to get it in that way” (as cited in Spiegel, 2005, p. 5).

Spitzer and co-researcher Joseph Fleiss (1974) were influential in challenging the reliability of the DSM-II and stressed the importance of revising the manual to address this. In highlighting their concerns about the DSM-II’s reliability, Spitzer and Fleiss (1974) recomputed the findings of six prior reliability analyses of the DSM-II and concluded that:

There are no diagnostic categories for which reliability is uniformly high. Reliability appears to be only satisfactory for three categories: mental deficiency, organic brain syndrome (but not its subtypes), and alcoholism. The level of reliability is no better than fair for psychosis and schizophrenia and is poor for the remaining categories. (p. 344)

In an effort to promote greater reliability in the DSM-III, the revision task force initiated a number of field trials. When the DSM-III was published in 1980, and in reference to these field trial findings, the manual's authors reported "far greater reliability than had previously been obtained with the DSM-II", and noted that "reliability for most classes in both phases is quite good" (American Psychiatric Association, 1980, pp. 5, 468). Roughly two years later, Hyler, Williams, and Spitzer (1982) asserted that "the reliability of the major diagnostic classes of DSM-III was extremely good" (p. 1276).

Nonetheless, Kirk and Kutchins (1994) asserted that there was no evidence that any formal comparisons between the DSM-III and previous versions of the manual had been conducted. Further, in their own analysis of the DSM-III's field trial data, Kirk and Kutchins (1994) asserted that reliability spanned "the entire spectrum from chance to perfect agreement" (para. 30). Further, the researchers concluded that the DSM-III's kappa's were "wildly uneven and unstable" (para. 33). These authors speculated that methodological problems precipitated these inconsistencies and noted that, of the 13 kappas provided for children's Axis I disorders, three were based on a single patient, one on two patients, and two kappas relied on four patients.

Kirk and Kutchins (1994) also questioned the requirements on which the field trials' kappas were based. For instance, in order to achieve a kappa of one (perfect agreement) the observers had only to agree that a mood disorder existed versus clarifying the particular type of mood disorder. Kirk and Kutchins suggested that it would have been more accurate for the DSM-III's authors to refer to the field trial data as being similar to previous studies.



Another study examined DSM-III-R's reliability and documented similar problems with the manual's rigor. This analysis was particularly noteworthy because a number of the study's researchers played a significant role in the DSM-III's development (Williams et al., 1992). The study was carried out at six sites in the United States and one in Germany and involved interviews of 390 psychiatric patients and 202 non-psychiatric participants. The diagnoses were conducted by experienced and specially trained mental health professionals utilizing:

a finely tuned classification system (DSM-III-TR) developed over a ten year period by outstanding psychiatric researchers; specially behaviorally oriented diagnostic criteria, a carefully developed structured interview (SCID), careful selection and training of experienced professional interviewers, and the competent supervision research team that is perhaps the most experienced at conducting diagnostic studies in the world. (Kirk & Kutchins, 1994, para. 37)

Data analysis of the study's psychiatric patient sample yielded kappa scores ranging from .40 (fair agreement) to .86 (almost perfect agreement) with a weighted balance of .61 (substantial agreement), when aggregated across five of the sites (Williams et al., 1992). For the non-patient community sample, the kappa scores from two sites ranged from .19 (slight agreement) to .59 (moderate agreement) with an average of .37 (fair agreement). Kirk and Kutchins (1994) asserted that, even with substantial efforts to maximize reliability, the results were unconvincing.

Further, the authors posited that the reliability levels were likely higher than those which would have been expected from a more normative clinical setting (Kirk & Kutchins, 1994)—a conclusion reiterated by Robert Spitzer, who stated in reference to the DSM-IV-TR that “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" (as cited in Spiegel, 2005, p. 63).

In 1974, Spitzer and Fleiss had posited that the DSM could not be valid if its classification system was unreliable. Roughly 27 years later, in reference to the DSM-IV-TR's validity, Andreasen and Black (2001) asserted that "Biologically oriented psychiatrists have objected to the lack of validity in the DSM as well. In this instance they point to the arbitrary nature of the definitions, which are not rooted in information about biological causes" (pp. 35–36).

Concerns surrounding the potential for the DSM-IV-TR to oppress marginalized populations have been raised by Caplan and Cosgrove (2004) who stated that "psychiatric diagnosis has been conceived of and applied in extremely biased ways and is surprisingly unwarranted by scientific research" (p. xix.). In addition, the authors asserted that the DSM-IV-TR's biases are potentially dangerous because diagnostic labels can negatively impact employment, health insurance coverage, and legal proceedings (e.g., child custody).

Eriksen and Kress (2008) argued that locating the source of psychological distress within the individual misdirects attention away from the socio-political determinants of psychological well-being. In this way, forms of oppression like racism, sexism, homophobia, ageism, classism, ableism and mother-blaming, purportedly become embedded within diagnostic and treatment guidelines (Caplan & Cosgrove, 2004).

Caplan and Cosgrove (2004) also noted the apparent absence of educational materials for psychotherapists that adequately address socio-cultural biases among clinicians, and the impacts such biases have on diagnosing psychopathology in clients.

Similarly, Ofer Zur, Director of the Zur Institute, an online continuing education resource for mental health clinicians, asserted that:

Most undergraduate, graduate, and post-graduate education neglects critical aspects of training in regard to the complex process of diagnosis. Few programs inform students that DSM diagnostic criteria generally lacks empirical support, that some criteria is the result of political or popularity ‘voting’, that scientific method and evidence has been largely disregarded in its development and that issues such as gender and cultural sensitivity are grossly underrepresented. (Zur & Nordmarken, 2010, p. 6)

A paper co-authored by two members of the DSM-V task force made some predictions about the then unpublished DSM-V’s reliability tests. This article stated that “To see a kappa for a DSM-V diagnosis above 0.8 would be almost miraculous; to see  $\kappa$  between 0.6 and 0.8 would be cause for celebration. A realistic goal is a kappa ( $\kappa$ ) between 0.4 and 0.6, while a  $\kappa$  between 0.2 and 0.4 would be acceptable” (Kraemer, Kupfer, Clarke, Narrow, & Regier, 2012, p. 14). Further, in response to concerns surrounding the DSM-V’s rigor, Kraemer and colleagues (2012) asserted that:

Many medical diagnoses go into common use without any evaluation, and many believe that the rates of reliability and validity of diagnoses in other areas of medicine are much higher than they are. Indeed, psychiatry is the exception in that we have paid considerable attention to the reliability of our diagnoses. It is important that our expectations of DSM-V diagnoses be viewed in the context of what is known about the reliability and validity of diagnoses throughout medicine and not be set unrealistically high, exceeding the standards that pertain to the rest of medicine. (p. 15)

The DSM-V was published in 2014. It is noteworthy that the DSM-V kappa guidelines used for establishing inter-rater agreement levels were adjusted relative to previous revisions of the manual (Carney, 2013; R. Cooper, 2014; Regier et al., 2013). In responding to this observation R. Cooper (2014) stated that:

While many psychiatrists have become used to thinking of Spitzer’s threshold of 0.7 as the cut-off point for a “good” kappa, there are precedents for employing

lower benchmarks in the statistical literature. Influentially, Landis and Koch (1977) count 0.21-0.4 fair, 0.41-0.6 moderate, 0.61-0.8 substantial, and 0.81—almost perfect. Altman (1991), condemns only kappas of less than 0.2 as poor, and considers anything above 0.61 as good. Fleiss, Levin and Cho Paik (2003) counts kappa's below 0.4 poor, those Between 0.4 and 0.75 fair to good, and those above 0.75 excellent. Clearly there are no universally agreed standards for what would count as a “good” Cohen’s kappa. (para. 13)

Based on these adjusted kappa levels, the DSM–V Task Force concluded that “most diagnoses adequately tested had good to very good reliability” (Regier et al., 2013, p. 59).

However, if one applies the older structure for establishing DSM kappa levels, there are some cases in which the inter-rater reliability for disorders has decreased relative to previous revisions of the manual. For example, inter-rater agreement for Schizophrenia was determined to have a kappa value of 0.81 in the DSM-III trial, and 0.46 in the DSM-V trial (Regier et al., 2013). In this regard, major affective disorders have also seen a decrease from a kappa of 0.8 to a kappa of 0.28, respectively.

Next, according to the previous measure of inter-rater reliability, the DSM-V’s kappa score for mixed anxiety-depressive disorder is negative (i.e., worse than chance alone)—meaning that clinicians would likely have achieved greater inter-rater agreement through guesswork (R. Cooper, 2014). In total, of the twenty diagnoses studied for the DSM-V field trials, three (15%) resulted in kappa scores that were greater than 0.6. According to the DSM-IV model for establishing inter-rater reliability, this meant that 17 (85%) of the kappa scores had agreement levels at or below a moderate level of inter-rater agreement (R. Cooper, 2014).

The DSM has provided generations of clinicians across a variety of disciplines with a framework for evaluating the type and severity of mental distress. Similarly, the manual has helped to facilitate important research efforts into the etiology, diagnosis, and

treatment (pharmacological and non-pharmacological) of psychological suffering. In addition, the DSM has helped to facilitate the provision of services, including financial support, for many individuals suffering with psychological distress.

However, as noted herein, there are also well documented problems with the DSM's validity and inter-rater reliability. Further, the implications of these shortcomings must be considered when it comes to training psychologists for prescriptive authority—particularly since the DSM plays an integral role in determining which psychotropic drugs with potentially dangerous side effects, should be prescribed.

### **C/S/X Movement**

This study examined psychologist postdoctoral psychopharmacology training materials for c/s/x content because the c/s/x movement represents a population of marginalized individuals who are particularly vulnerable to the negative consequences of diagnostic and treatment errors resulting from unresolved issues pertaining to etiological hypotheses for depression and the rigor of the DSM (LeFrancois et al., 2013; Reaume, 2002; Whitaker, 2010). Further, integrating c/s/x voices within the context of training psychologists for prescriptive authority is consistent with both disciplines' ethical guidelines, represents a valuable opportunity for shared advocacy, and has important ethical and legal implications within the context of obtaining informed consent from patients.

The c/s/x movement is an international patient-lead phenomenon comprised of diverse needs, experiences and perspectives reflected in the movement's monikers; *users*, *consumers*, *ex-patients*, and *survivors* (Morrison, 2006). Through this lens, people who make use of mental health services are viewed as 'users' or 'consumers' while

‘ex-patients’ are individuals who were treated, in some cases involuntarily, within psychiatric facilities. The term *survivor* refers to an identity based upon the concept of resilience and having endured previous psychiatric treatment, in some cases prior to deinstitutionalization. These nomenclatures have been criticized. For example, the term “consumer” has been challenged for its capitalist overtures and “survivor” because of its associations with the Holocaust (Reaume, 2002, p. 423).

The c/s/x movement’s origins have been linked to the civil rights, gay liberation, feminist, and antipsychiatry movements of the 1960s and 1970s (Vogt, 2007). Vogt conceptualized the 1970s and early 1980s as a more radical period of mental health activism in which the majority of c/s/x initiatives excluded professionals and non-patients because they tended to “interfere in consciousness raising . . . and usually have mentalist<sup>1</sup> attitudes” (Chamberlin, 1978, p. 86). However, some c/s/x organizations developed partnerships with mainstream mental health initiatives as a means to procure funding and sustainability (Chamberlin, 1978; Vogt, 2007). These collaborations were controversial among c/s/x activists. For example, Clay (2005) posited the risk of “‘co-option’ of a peer program’s basic principles and unique beliefs” by mainstream mental health providers who had their own agendas (p. 243). In addition, Clay suggested that the imposition of “external rules and procedures” by mainstream providers stifled the efficacy of some c/s/x programs (p. 242).

The c/s/x population has been characterized as being particularly vulnerable to negative outcomes from treatment errors because its members, in addition to being

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<sup>1</sup> Mentalist: discrimination against a person based on the perception that they are unstable or mentally ill

stigmatized for mental distress, typically suffer from the impacts of low socioeconomic status. This includes stigmatization for being poor, difficulties securing employment and affordable housing, and access to post-secondary education and appropriate health care. Further, many c/s/x individuals face additional intersecting forms of marginalization such as racial discrimination, homophobia, and/or misogyny (LeFrancois et al., 2013). Next, as a result of being impoverished, access to more expensive non-pharmacological interventions (i.e., psychotherapy) may be restricted. In cases of generational poverty, financial support from immediate or extended family members may be limited too. In this regard, Menzies, LeFrancois, and Reaume described “the critical need to examine psychiatric practices that mediate and amplify a host of crosscutting socio-economic and political forms of discrimination” (LeFrancois et al., 2013, p. 16).

In conceptualizing the c/s/x movement, Usar (2014) noted:

Advocacy, choice, peer support, self-help, self-definition and self-determination, in particular claiming “voice” and talking from the standpoint of marginalized experience have been the guiding principles of the c/s/x movement since its inception in the second half of the 20<sup>th</sup> century. Rooted in broader struggles, for social justice these principles have been successfully used by people who are labeled with mental illness to speak their truth, share their stories, voice their opinions, and challenge the psychiatric system and its knowledge. Collectively, people have organized to expose systemic discrimination and abuse they face within the psychiatric system and the larger society, disputed the bio-medical understanding of “mental illness”, and created self-help alternatives to coercive treatments. (p. 2)

The fight against coercive psychiatric practices has been an integral part of the c/s/x movement’s *raison d’être*, development and activism (Oaks as cited in Kallert, Mezzich, & Monahan, 2011). In the United States, forced drugging is primarily used for mitigating a person’s risk of harm to self and/or other(s) (Treatment Advocacy Center, 2016). However, there are also cases in which patients are forcibly drugged based upon

assumptions that they lack the capacity to determine the best course of treatment (Treatment Advocacy Centre, 2016). Concerns have been raised, in some cases litigiously, about the constitutional legality of these practices which threaten liberty and dignity (Gottstein, 2008; Mendez, 2013; Oaks, 2011). The recent United Nations report by the *Special Rapporteur on Torture and other Cruel, Inhuman or Degrading Treatment or Punishment* asserted:

Despite the significant strides made in the development of norms for the abolition of forced psychiatric interventions on the basis of disability alone as a form of torture and ill-treatment and the authoritative guidance provided by the CRPD (Convention on the Rights of People with Disabilities), severe abuses continue to be committed in health-care settings where choices by people with disabilities are often overridden based on their supposed “best interests”, and where serious violations and discrimination against persons with disabilities may be masked as “good intentions” of health-care professionals. (Mendez, 2013, p. 4)

Similarly, psychologist and c/s/x activist Ronald Bassman (2005) asserted:

Many of my peers, those of us who have been treated for serious mental illness, are not comforted by the popular belief that we have moved far beyond the days of frontal lobotomies, insulin shock, metrazol shock, electroshock, teeth extractions, and organ amputations. Though the sojourns of mental patients into community life freed them from confining institutions, if you listen you will hear stories of chemical lobotomies which have become their new prisons with invisible, yet substantial walls (Blanch, Fisher, Tucker, Walsh, and Chassman, 1993; Ridgway, 2001; Chamberlin, 2002). (p. 492)

Another important issue pertaining to coercive prescription practices involves risks associated with psychotropic medications’ iatrogenic effects (Gottstein, 2008). For example, neuroleptics have been linked to degradation of the frontal cortex and antidepressants have been shown to negatively impact bone density and blood platelet characteristics, in addition to increasing the risk of stroke, suicidality, mortality, and complications during pregnancy (Andrews et al., 2011). The following body of research highlights the potential dangers of antidepressants in light of c/s/x concerns and the



potential risks that this vulnerable population faces as a result of treatment errors pertaining to psychotropic interventions for depression.

Pregnancy. Meta-analytical findings indicate an apparent link between SSRI exposure during the third trimester and pulmonary hypertension in newborns; a condition that is fatal for approximately 10% of babies who suffer from the illness (Ellfolk & Malm, 2010). Other data suggested that antidepressants are correlated with preterm deliveries and low birth weight, although these findings currently lack sufficient evidence for any definitive conclusions (Ellfolk & Malm, 2010). Withdrawal symptoms correlated with maternal SSRI use have been observed in newborns, however, the associated risks and long-term implications, are unclear (Ellfolk & Malm, 2010; Moret, Isaac, & Briley, 2009).

In-vitro studies suggest that SSRIs paroxetine and, potentially, fluoxetine and citalopram have teratogenic potential (Sloot, Bowdeb, & Yih, 2009). Additional animal research using mice indicated that permanent neurobehavioral alterations subsequent to in-utero exposure to these medications can and do occur (Ansorge, Morelli, & Gingrich, 2008; Holmes, Murphy, & Crawley, 2003). Nonetheless, human data surrounding long-term developmental risks associated with maternal SSRI use during pregnancy remains inconclusive (Ellfolk & Malm, 2010).

For example, a meta-analysis of seven studies (n = 1774) by Einarson and Einarson (2005) did not find increased risk of birth complications from antidepressants. These conclusions are limited by the study's methodology which involved using a number of different antidepressants that impacted a variety of different pathways. Also, the authors were directly involved in three of the seven studies reviewed raising the

potential for bias in the selection, presentation, and interpretation of data. Finally, the review did not investigate the potential for any long-term neurobehavioral effects for babies whose mothers were using SSRIs or other antidepressants.

Electrolytes and bone density. Another health risk correlated with SSRIs is hyponatremia, a condition which can increase the chances of falls due to dizziness associated with electrolyte imbalances. A number of reviewers outlined how this can be problematic for older individuals, particularly when it comes to compounded effects from polypharmacy (e.g., diuretics) (Andrews et al., 2011; Jacob & Spinler, 2006; Moret et al., 2009). There is also evidence that SSRIs degrade bone mineral density which could conceivably increase the likelihood of bone fractures, particularly among the aged (Moret et al., 2009).

A randomly selected prospective cohort study involving 5,008 community dwelling adults (50 years old or older) found that daily SSRI use ( $n = 137$ ) was associated with a two-fold increased risk of fractures (J. Richards et al., 2007). However, limitations of this study included the fact that the duration of participants' daily SSRI use was unknown. Hence, it was not possible to link the extent of SSRI use with fractures. Also, depression was diagnosed by questionnaires and not via an interview with a psychiatrist. Other reviewers have suggested that the sympathetic nervous system activity, glucocorticoids, and inflammatory cytokines associated with depression, may underlie decreases in bone density (Chau, Atkinson, & Taylor, 2012; Yirmiya & Bab, 2009).

Internal bleeding. A number of studies indicated that SSRI users were more likely to be hospitalized for abnormal bleeding and lose more blood during surgery than

non-antidepressant users (Meijer et al., 2004; van Haelst et al., 2010). A study which analyzed data from a cohort of 64,000 new antidepressant users found 196 cases with abnormal bleeding and a positive correlation between this problem and SSRI use (Meijer et al., 2004). Limitations of the study included the relatively small number of cases and the fact that hospital records were used for diagnosis. Also, instances of bleeding where hospitalization did not occur would have been missed meaning there was a possibility that risk levels were underestimated.

SSRI use has also been linked to upper gastrointestinal bleeding—a risk that appears to be amplified by concurrent prescriptions for anti-thrombotic pharmaceuticals like non-steroidal anti-inflammatory drugs (NSAIDs) (Dall, De Muckadell, Lassen, Hansen, & Hallas, 2009). Dall and colleagues compared data from 3,652 individuals diagnosed with serious upper gastrointestinal bleeding (USB) to 36,502 controls matched for age and gender. The researchers found that SSRI use was associated with USB and noted that the results were consistent with antiplatelet effects already associated with this class of drug. These results may have been skewed by selection and information biases, however. For example, the researchers noted that their database did not include information about potential confounds like alcohol use, smoking, physical frailty, or the consumption of non-prescription ulcerogenic drugs.

Stroke. SSRIs have been associated with increased risk for ischemic and hemorrhagic stroke (Trifiro, Dieleman, Sen, Gambassi, & Sturkenboom, 2010). Trifiro and colleagues identified 996 cases of ischemic stroke from a primary care database. In comparison with controls matched for age (65 years and older), sex, and index date, SSRI use was associated with greater risk for ischemic stroke, particularly over the short-term.

This was an observational study, however, and the researchers lacked confirmation about whether prescriptions were actually filled and taken. Thus, it is possible that poor compliance confounded results.

Other findings indicated that antidepressant use may actually improve survival rates in stroke patients (Jorge, Robinson, Arndt, & Starkstein, 2003). Nonetheless, even if SSRIs can offer anticoagulant benefits for such patients, this upshot may be offset by the harmful anti-clotting effects mentioned earlier (Andrews et al., 2011).

**Behaviour.** Behavioral inhibition problems, attentional deficits, increased suicidal behaviours, and higher mortality rates have been linked to antidepressants. For example, a review noted that SSRIs and tricyclic antidepressants were correlated with difficulty inhibiting socially appropriate behaviours. Other research has shown a positive correlation between motor vehicle accidents (MVAs) and antidepressant use (Chang et al., 2013; Gibson et al., 2009). However, these findings should be viewed cautiously given that compliance with medication use and unmeasured environmental stressors could ostensibly have impacted the rate of MVAs.

**Suicidality.** Large scale meta-analytic studies supported claims surrounding the existence of age-dependent risk for suicidal behaviours among SSRI users (Fergusson, Doucette, & Cranley-Glass, 2005; Stone et al., 2009). The meta-analysis by Stone and colleagues (2009) found that antidepressant use in children, adolescents, and young adults (< 25 years old) was associated with increased risk of suicidality, but that this risk declined with age. Indeed, for patients over the age of 65, suicidal ideation appeared to decrease with SSRI use. It should also be noted that, for younger patients, the harmful effects of antidepressant drugs showed the strongest correlations with suicidality among

those who were being treated for disorders other than depression. Conversely, among older individuals, the beneficial outcomes were associated with their being treated for major depressive disorder (Stone et al., 2009). Thus, suicidality risks linked to antidepressants may be age and disease dependent. Stone and colleagues noted the primary limitation of their meta-analysis was that it failed to sufficiently address the differing types of patients and variety of circumstances for which antidepressants may be prescribed. In particular, they asserted that patients exhibiting a high risk for suicide would probably not have been enrolled in a placebo controlled study for ethical reasons. Also, most of the examined studies involved the treatment of acute versus chronic conditions. Finally, there was a sparse amount of relevant data since few suicides occurred during the studies.

Proponents of antidepressants have argued that, left untreated, depression itself can result in suicide (Julien et al., 2011; Stahl, 2008). However, a meta-analysis of 19 epidemiological studies on depression and suicide risk showed that there was little evidence to support the hypotheses that suicide risk could be reduced or increased via antidepressant use (Baldessarini et al., 2007).

Mortality. Adding to the controversy surrounding suicidality and notwithstanding the potentially favorable effects of SSRIs in this regard for elderly patients, are findings linking antidepressants to an overall increase in mortality rates. The recent Woman's Health Initiative study, for instance, showed that antidepressant use was associated with being one and a half times more likely to die, even if the participants did not meet the criteria for depression (Smoller et al., 2009). Also of note was the fact that less than 1%

of participants' deaths were attributed to suicide, suggesting that the mortality rate for this sample was primarily based on physical health issues.

The complexity of empirical literature on antidepressant related suicide and mortality makes it difficult to draw more definitive conclusions about the associated risks that these drugs pose (Baldessarini et al., 2007; Reeves & Ladner, 2010). Methodology wise, it is impossible to control for the sheer number of variables that can ultimately lead to an untimely death or health problem. For example, it is conceivable that environmental teratogens may play a role in accounting for the adverse health symptoms experienced by babies whose mothers happened to be using antidepressants during the child's neonatal development (Ellfolk & Malm, 2010). Further, the data was correlative and findings from animal studies may not be applicable to human beings.

These health and safety concerns should also be considered in light of the unprecedented numbers of people being prescribed antidepressants. For example, in 2010, more than 250 million prescriptions for antidepressants were dispensed in the United States alone (IMS Institute for Healthcare Informatics, 2011). In Canada, 19% of British Columbian women—roughly one in five—received a prescription for an SSRI during a 12 month period between 2002 and 2003 (Currie, 2005).

Consequently, it warrants considering the potentially negative implications of these risks on vulnerable groups like the c/s/x population. A population, for whom the health and safety risks of antidepressants (prescribed amidst unresolved etiological and diagnostic questions) have the potential to compound existing hardships born from poverty and other forms of marginalization. In the same vein, and also in line with the assertion that psychologist postdoctoral psychopharmacology training should inform

students about the c/s/x movement, are the disciplines' (psychology and prescribing psychology) ethical guidelines. Guidelines which assert the importance of addressing the strengths and weaknesses of evidence used to justify interventions, when obtaining informed consent from patients.

For example, the American Psychological Association's *Ethical Principles of Psychologists and Code of Conduct* (2010) states:

(b) When obtaining informed consent for treatment for which generally recognized techniques and procedures have not been established, psychologists inform their clients/patients of the developing nature of the treatment, the potential risks involved, alternative treatments that may be available, and the voluntary nature of their participation. (See also Standards). (p. 13)

Similarly, the *Practice Guidelines for Psychologists' Involvement in Psychopharmacological Issues* states:

Rationale. The APA (2002b) Ethics Code requires psychologists to obtain informed consent before any professional interaction whenever possible. The decision to prescribe medication for a patient optimally results from collaboration between that patient and the psychologist, rather than from a unilateral decision by the prescriber. A collaborative decision depends upon appropriate education of the patient about alternative treatments and full informed consent. (American Psychological Association, 2011, p. 844)

In addressing the implications of informed consent with regard to forced and voluntary use of psychotropic medications the same guidelines state:

Psychologists in forensic settings may work with individuals who are unable or unwilling to provide informed consent. In these circumstances, it is incumbent upon the psychologist to be aware of both institutional rules and regulations and APA ethical expectations for how to handle the administration of medications in the absence of consent. Despite differences in the context of the treatment, the psychologist endeavors to provide the same level of education and disclosure about medication and its efficacy, iatrogenic effects, and medication procedures as he or she would for any other patient.

The use of medication increases the universe of topics to be addressed as part of the informed consent process. (American Psychological Association, 2011, p. 844)

Based on both of the aforementioned guidelines for informed consent the prescribing psychologist would presumably need to convey that the etiology of depression has not been established and that there are considerable limits to the rigor of DSM diagnoses used to inform psychiatric interventions and decisions about forced and voluntary drugging. The ability to relay this information as a prescribing psychologist would arguably require that it had been addressed with sufficient breadth during clinical training and within the materials used to accomplish this. Further, failure to address these issues within the scope of obtaining informed consent could present ethical and legal problems in cases where treatment errors occur.

Consider the American Psychological Association's (2011) *Practice Guidelines Regarding Psychologists' Involvement in Pharmacological Issues* here:

Guideline 2. Psychologists are urged to evaluate their own feelings and attitudes about the role of medication in the treatment of psychological disorders, as these feelings and attitudes can potentially affect communications with patients.

Rationale: There is some evidence to suggest the clinician's faith in the treatment can be an important predictor of treatment response (Jacobson & Hollon, 1996). Unfortunately, treatment with medication has at times been associated with both excessive optimism and skepticism (e.g., Kramer, 1993; Valenstein, 1998), and both positions have been exaggerated by media attention. Psychologists will inevitably form their own opinions about medications. These opinions can in turn affect patients' decisions about taking a prescribed medication, and even medication effectiveness, if they are not addressed openly in the process of discussing psychopharmacological interventions.

Implications: Psychologists who are aware of their attitudes and feelings towards medications, and who openly accept the possible validity of alternative viewpoints, are in the best position to discuss the potential risks and benefits of using medication in a balanced manner. Psychologists are encouraged to explore their own feelings about medication, and to consider the possible role of those feelings in discussions about pharmacotherapy with the individuals they serve. (p. 839)



Given that the practices of prescribing psychologists are being supervised by physicians, the *Code of Ethics for the American Medical Association* (2006) also warrants mentioning within the context of discussing informed consent and training considerations:

The patient's right of self-decision can be effectively exercised only if the patient possesses enough information to enable an informed choice. The patient should make his or her own determination about treatment. The physician's obligation is to present the medical facts accurately to the patient or to the individual responsible for the patient's care and to make recommendations for management in accordance with good medical practice. The physician has an ethical obligation to help the patient make choices from among the therapeutic alternatives consistent with good medical practice. Informed consent is a basic policy in both ethics and law that physicians must honor, unless the patient is unconscious or otherwise incapable of consenting and harm from failure to treat is imminent. (AMA, 2006, Opinion 8.08 – Informed Consent para. 1)

In this regard, Oaks has written about “coercion by misinformation” and noted:

One could argue that in coerced psychiatric procedures, some of the most powerful individuals in our society have authority over some of the most disenfranchised, and discredited citizens. Because of this power imbalance, the veracity of claims by mental health professionals ought to be held to the highest academic standards, because an error may destroy what many of us hold most precious: our liberty. (as cited in Kallert et al., 2011, p. 189)

Another ethical reason for integrating information about the c/s/x movement within psychologist postdoctoral psychopharmacology training materials is that the discipline of psychology's ethical principles and code of conduct and the practice guidelines for prescribing psychologists both uphold the importance of cultural sensitivity. It warrants noting here that the c/s/x movement has its own cultural mores reflected in the movement's history, distinctive beliefs, unique forms of artistic expression, activism, and nomenclature (LeFrancois et al., 2013; Usar, 2014).

*Principle E: Respect for People's Rights and Dignity* from the American Psychological Association's *Ethical Principles of Psychologists and Code of Conduct* states:

Psychologists are aware of and respect cultural, individual and role differences, including those based on age, gender, gender identity, race, ethnicity, culture, national origin, religion, sexual orientation, disability, language and socioeconomic status and consider these factors when working with members of such groups. (American Psychological Association, 2010, p. 4)

Next the third tenet of the *Practice Guidelines for Psychologists' Involvement in Psychopharmacological Issues* states:

When clinicians work with patients or clients from different linguistic, ethnic, or cultural groups, clinicians recognize that the presentation or description of the clinical syndrome may reflect culturally-specific referents and may not conform to those of the dominant group. (American Psychological Association, 2011, p. 840)

This is reiterated in the guidelines' ninth tenet which notes that "Psychologists are encouraged to explore issues surrounding patient adherence and feelings about medication" and the tenth guideline, which states that "Psychologists are urged to develop a relationship that will allow the populations they serve to feel comfortable exploring issues surrounding medication use" (American Psychological Association, 2011, pp. 842–843).

Opportunities for shared advocacy efforts also dictate that psychologist postdoctoral psychopharmacology training materials should integrate information about the c/s/x movement. For example, there are already professional allies (e.g., various psychiatrists, psychologists, physicians, and social workers) and clinician activists, who have identified the importance of considering the c/s/x movement's history, mission, and needs when it comes to the provision and reformation of mental health care practices (MindFreedomInternational, 2010).

Additionally, prescribing psychologists have identified the importance of considering the c/s/x movement within the context of advocacy efforts. For example, Campbell and Gardner noted in *Prescriptive Authority for Psychologists: A History and Guide* that:

The story of psychology's consumer and professional alliances in the pursuit of prescriptive authority cannot adequately be told without a candid and personalized view of the journey taken by the pioneers and advocates in the struggle with mental illness and with the systems that have determined their course. Psychologists' alliances with consumers, families, and mental health professionals grow from mutual and intersecting goals for better legislation, improved mental health systems, and the underlying power of advocacy as the vehicle of change." (as cited in Sammons et al., 2003, p. 159)

At the same time Campbell and Gardner asserted:

Until recently, psychologists had a mixed record regarding involvement in the consumer mental health movement. Consumer's very negative view of mental health professionals is largely leveled at psychiatry as the professional force that directed patients into involuntary hospitalization and forced medication. On the other hand, consumers and families ask "Where are the psychologists and where were they then? (as cited in Sammons et al., 2003, pp.164–165)

In considering the opportunity for rectifying this, Campbell and Gardner posited that "The importance of capturing this bird's eye view of the consumer's experience within the mental health system is that the practices that cause dissatisfaction with psychiatric services invite change and movement into the psychosocial realm of psychological treatment" (as cited in Sammons et al., 2003, p.162). Similarly, the authors asserted "As psychologists adopt the role of mental health professionals with prescriptive authority, they are skilled and prepared to offer a psychosocial model of comprehensive treatment that corresponds to the long-standing needs and preferences of the consumer's group" (as cited in Sammons et al., 2003, p.162).

The value of teaching psychologists within postdoctoral psychopharmacology programs about c/s/x self-help initiatives also has implications for advocacy efforts. For example, c/s/x self-help offerings have the potential to provide valuable mental health resources, particularly for marginalized individuals who do not possess insurance for mental health support (i.e., counseling). Research by Nelson, Ochocka, Janzen, and Trainor (2006a, 2006b, 2006c, 2006d) identified the benefits of c/s/x initiatives on members' levels of community integration, psychiatric symptom reduction, reductions in the use of traditional mental health care services, and improvements in quality of life. A study by Chassot and Mendes (2014) examined how active participation (i.e., activism) within the British "user/survivor movement" impacted subjects' ability to conceptualize their experiences through positive reframing, increased self-esteem and afforded opportunities for acceptance and connections with fellow users/survivors difficult to achieve within social networks outside of that context.

Chassot and Mendes (2014) also noted diversity in terms of the types of support being offered by "user/survivor" organizations:

Although alternative models of understanding mental distress are important to some members, we also found that other participants valued access to mainstream psychiatric information through service user/survivor groups, which they used to better understand their conditions and to negotiate care with practitioners. (p. 9)

A large SAMHSA funded study of consumer operated service programs from 1998 to 2002 found that the examined drop-in programs provided a variety of services that typically paralleled traditional mental health and social services offerings (i.e., activity and support groups, telephone access, laundry facilities, computer use, and assistance with medication education, clothing, and bus/transportation passes) (Johnsen,

Teague, & Herr, 2005). The researchers also reported that both of the examined peer support programs “have a systematic approach consistent with the principles of empowerment and recovery” (Johnsen et al., 2005, pp. 232–233). Next, the study concluded that peer support initiatives provided additional assistance with managing personal concerns (i.e., employment issues, housing and health, recreation, and maintenance of personal relationships). By contrast, the educational programs focussed attention on helping consumers gain an “accurate and comprehensive knowledge about mental illness and psychiatric services” (Johnsen et al., 2005, p. 232).

There is a history of collaborative advocacy efforts between c/s/x organizations and mainstream institutions. For example, MindFreedomInternational has collaborated with the World Health Organization (Mezzich, 2007) and the United Nations (2006). In discussing their alliance with MindFreedomInternational’s efforts to protect human rights within the context of mental health care, Dr. Benedetto Saraceno, former Director of the World Health Organization’s Department of Mental Health and Substance Abuse asserted:

MindFreedom is doing remarkable work nationally and internationally to promote the protection of human rights in mental health. In my experience, MindFreedom has been a fair, transparent interlocutor. WHO has different views from MindFreedom in some areas, such as the definition of mental disorders or mental disability, or the assessment of the advantages and disadvantages of psychotropic medicines, but WHO also shares many common views with MindFreedom concerning the right of choice about treatment and, above all, the need of addressing the global emergency about the violation of human rights. On several occasions, MindFreedom’s Director David Oaks has expressed the same concerns as WHO and I think it is possible to build up a fair collaboration on those issues on which we agree, more than having no collaboration based on issues on which we disagree. (as cited in MindFreedomInternational, 2008, para. 1)

There are also examples of instances in which the c/s/x movement has been addressed within university curricula (Ryerson University, 2015; Trent University, 2015; York University, 2014). Similarly, in 2008, Simon Fraser University hosted the *Madness Citizenship & Social Justice Conference*. The conference's brochure noted:

Psychiatric users and survivors have become increasingly visible and proactive on various political fronts, organizing and aligning themselves with community groups, legal advocates and other constituencies in their collective struggles for adequate medical care, housing, education, employment, and legal and civil protections. By recruiting participants from these various constituencies, this conference will provide a sharp counterpoint to the prototypical meetings of clinical, professional and academic associations concerned with issues related to 'mental illness' and psychiatry. This event will be unique in offering a venue for the advancement of multi-vocal, progressive and counter-hegemonic perspectives on these critical topics and issues. The intention is to establish a context of mutual knowledge-sharing and empowerment, and to develop longer-term strategies that will permit activists, survivors, scholars and other participants to interact, share experiences, develop connections and engage in problem-solving praxis. (Simon Fraser University, 2008, p.1)

Integrating c/s/x perspectives into the materials used to train psychologists for prescriptive authority is also important because information about the population and its movement is so widely available online. For example, Usar (2014) has described how increasing numbers of c/s/x groups have developed websites in recent years to "publicize their work, as well as expand their outreach and advocacy activities" and for the purpose of providing "instant access to vital information, establishing links with various survivor/consumer groups and for keeping the advocacy momentum alive (Morrison, 2005, pp. 88–89)" (p. 21). Consequently, training for prescriptive authority which includes information on the c/s/x movement would ostensibly support prescribing psychologists in addressing questions and concerns that might result from clients' exposure to this information online.

In concluding their chapter on *Alliances with Consumer Groups and other Associations within Prescriptive Authority for Psychologists: A History and Guide*, Campbell and Gardner ended with a quote from Catherine Acuff who asserted that:

By creating collaborative partnerships with those in the c/s/x movement, by truly listening to their voices, by recognizing the individuality of those with whom we work, by rejecting pejorative labels and going beyond pathology based models, by being open to alternative therapies and self-help approaches, and by training our students and ourselves about the true hope of recovery, we can create a profession that is prepared for the future and the promise it holds (Acuff, 2000, p.1464). (as cited in Sammons et al., 2003, p. 175)

Further, it is conceivable that members of the c/s/x movement will seek out prescribing psychologists to explore alternatives to drug therapy, titrate medications, and/or combine the use of psychotropics with provisions for psychotherapy under a single provider (McGrath, 2010). Ultimately, if affiliations between prescribing psychologists and the c/s/x movement are weak or non-existent, opportunities for collaboration, shared learning, research, advocacy, and treatment could be missed.

Legitimate concerns surrounding the consequences of abolishing forced psychotropic interventions and forced hospitalization have also been raised. These arguments point to the ethical and legal complexities of maintaining the safety of patients and community members. Further, faced with dwindling community resources, compounded in some cases by addiction and poverty, there may be limited alternatives available for mental health practitioners, including prescribing psychologists, when it comes to mitigating risk. In the same vein, psychiatrists like Torrey (1997) and Satel (2000) have posited concerns about the potential for c/s/x organizations to dissuade individuals in need of psychiatric care from voluntarily receiving such services thereby increasing risk of harm to self or other.

To this end, Torrey and Satel have actively lobbied against government funding for c/s/x groups (McLean, 2003). Further, they have alleged that c/s/x organizations frequently represent extremist views whose arguments lack scientific support, and whose messaging amounts to misinformation about electro-convulsive-therapy, involuntary commitment, and psychotropic prescription drugs (Rissmiller & Rissmiller, 2006). The Treatment Advocacy Center which Torrey founded suggested that c/s/x initiatives aimed at individuals with mental distress increase the latter's risk of homelessness, imprisonment, and violence towards others (Treatment Advocacy Centre, 2011a). Similarly, former American Psychiatric Association medical Director, John Scully, called MindFreedomInternational's activism "ill-considered" and invited the group to reconsider its position and "join NAMI to help improve the care of our fellow citizens who suffer from serious mental illnesses" (MindFreedomInternational, 2003, p.1). Critiques of the c/s/x movement have also been levied by its own members. For example, Estroff (2004) noted that the movement's "exaggerated claims of agency may pose as many problems as erasure of agency for people with schizophrenia—and both are probably inaccurate" (p. 300).

The fact that the c/s/x movement is controversial does not negate the importance of addressing the phenomenon within the context of the materials used to train psychologists to prescribe. Rather, being exposed to information about this marginalized population would require that prescribers grapple with the history, perspectives and needs of a diverse, complex, and marginalized population that is, by virtue of its members' intersecting forms of discrimination, particularly vulnerable to treatment errors.



**Research Question**

To clarify the extent to which current psychologist postdoctoral psychopharmacology training materials are integrating critical perspectives and considering their implications for treating a marginalized population, this study sought to understand: (a) To what extent do psychologist postdoctoral psychopharmacology training materials critique neurobiologically based hypotheses for the etiology of depression, (b) challenge the DSM's empirical rigor, and (c) integrate information about the c/s/x movement?

## Chapter II: Method

### Operational Definitions

The following operational definitions were used within the study's content analysis.

- *Measures—Neurobiological Hypotheses of Depression* refers to any brain based hypothesis or theory about the cause of depression.
- *Measures—DSM*. The Diagnostic and Statistics Manual of Mental Disorders (DSM) is published by the American Psychiatric Association. There are five editions of the DSM, plus two formal revisions (I, II, III, III-R, IV, IV-TR, and V). Operationally, the DSM must be explicitly mentioned or cited in order for content to be assigned to a particular coding category for this topic. However, provisions were also made to document relevant content falling outside of the coding framework's requirements (i.e., content about the DSM that did not include any explicit mentions of the manual).
- *Measures—C/S/X Movement*. This operational definition pertained to any mentions of the consumer/survivor/ex-patient movement. The Encyclopedia Britannica (2016) defined social movement as a "loosely organized but sustained campaign in support of a social goal, typically either the implementation or the prevention of a change in society's structure or values." For the purposes of this study, the c/s/x movement has been conceptualized as an organized and sustained movement challenging the medicalization of mental distress including practices like forced drugging, forced hospitalization, forced electro-convulsive therapy, diagnostic procedures, and

problems with obtaining informed consent. Materials or statements by and about individuals who identified or were deemed to have identified as c/s/x activists were also included within this operational definition. In this regard, the term activist has been characterized within the Oxford Dictionary (2016) as a “person who campaigns to bring about political or social change.”

Consequently, for the purposes of this study, “c/s/x activist” is defined as an individual who campaigns for social change within the context of supporting the c/s/x movement as outlined above. The terms “consumer(s),”

“survivor(s),” or “ex-patient(s)” on their own did not meet the criteria for this operational definition unless the statement(s) in which they were embedded clearly referred to the c/s/x movement.

- *Measures—Citations/References.* Citations within an examined reading or video were documented in two scenarios. First, the researcher identified citations from the following list of critics: Joseph Glenmullen, Marcia Angell, Peter Breggin, David Cohen, David Healy, Joanna Moncrieff, Irving Kirsch, Robert Whitaker, Glen Spielmans, Jeffery Lacasse, Jonathan Leo, Jay Joseph, David Antonuccio, and Brett Deacon . The citation could be documented via APA format or the critic could be referenced by name. This list of critics was developed through consultation with a number of the experts and critical perspective holders identified within the dissertation’s acknowledgements section. Second, this study sought to determine whether or not any articles or books written by c/s/x activists were cited.

- *Measures—Critiques.* A “critique” was operationally defined as any statement that highlighted the limitations of a neurobiologically based hypothesis or theory for depression’s etiology, or content which was critical of the DSM in any way. For example, any content which identified problems with the DSM’s inter-rater reliability and construct validity, or limitations of using the manual (e.g., requires considerable training) or controversy (e.g., disagreements about a category based approach), was documented as a critique. A critique of the c/s/x movement was operationally defined as any content which challenged the legitimacy of the movement and its organizations. Similarly, a critique of this aforementioned content was operationally defined as any challenge levied against a negative appraisal of the c/s/x movement’s legitimacy.
- *Measures—Construct Validity for DSM.* In this context, construct validity pertained to whether or not DSM diagnoses actually measure what they are purported to by the manual.
- *Measures—Inter-rater Reliability for DSM.* In this context, inter-rater reliability was operationally defined as the likelihood that different clinicians or coders would obtain similar diagnostic results when using the DSM to diagnose the same patient.
- *Measures—Statistics on Inter-Rater Reliability or the Construct Validity of the DSM.* The operational definition for this measure pertained to the inclusion of any statistical data about inter-rater agreements (i.e., kappa

scores) for DSM diagnoses and/or the manual's construct validity (e.g., concordance rates between the DSM and the ICD-10).

- *Measures—Coding Categories.* Coding categories were developed for each of the three topics to identify and categorize information pertaining to a neurobiological etiology of depression, the DSM, and the c/s/x movement. For operational definitions pertaining to each of the topic's coding categories, see Appendices A (neurobiological etiology's of depression), B (the DSM), and C (the c/s/x movement).

## **Methodology**

**General description of content analysis.** According to social psychologist Steven Stemler (2001), content analysis is “a systematic, reliable technique for compressing many words of text [or video] into fewer content categories based on explicit rules for coding” (p. 1.). Neuendorf (2002) asserted that the researcher must initially determine what content will be examined and clarify the rationale behind the proposed analysis. This includes ascertaining which theories or perspectives justify the value of the proposed study. Following this, the researcher should select the research question(s) or hypotheses and conceptualize the variables to be studied. Next, the researcher must clarify the coding process including the establishment of operational definitions, known as coding schemes (Neuendorf, 2002). The researcher should also determine whether a complete census of the materials is viable.

In content analysis, an identifiable message or component of a message within the coding scheme is called a *unit* (Neuendorf, 2002). The unit helps identify which variables are being measured and provides the basis for reporting observations. While content

analyses frequently involve word counts, other forms of data can be used. For instance, Lacasse and Gomory's (2003) study examined the extent to which critics' works were being incorporated into social work syllabi.

According to Krippendorff (2004), the researcher must clarify the parameters of the variables being studied. In this way, operational definitions are used to dictate which of the written passages in books or spoken segments of video materials, will be analyzed.

The two primary means of organizing data via content analysis are *a-priori* and *emergent coding* (Stemler, 2001). With a-priori coding, a particular theory is used to establish categories prior to the main analysis. In emergent coding, categories are developed and refined throughout the analysis of data. The specificity of coding categories has important implications for data collection. For example, if the categories are too broad the researcher may face challenges in developing complex impressions about the data set. Conversely, if the categories are highly specific, the researcher may have difficulty formulating general impressions.

Inevitably, some relevant content will fall outside of the coding categories and the researcher must determine what to do with this information. Miles and Huberman (1994) noted,

Data reduction is not something separate from analysis. It is part of analysis. The researcher's decisions—which data chunks to code and which to pull out, which evolving story to tell—are all analytic choices. Data reduction is a form of analysis that sharpens, sorts, focuses, discards, and organizes data in such a way that "final" conclusions can be drawn and verified. (as cited in Namey, Guest, Thairu, & Johnson 2008, p. 139)

If the researcher concludes that the omission of relevant data that cannot be categorized within the topic's exemplars is unacceptable, additional sub-categories might be

developed for this purpose (Namey et al., 2008). At the same time, Namey and colleagues (2008) asserted that “There is no single “right” way to approach analysis of a large qualitative data set and often an assortment of complementary approaches, building one upon another and triangulating findings, is preferable” (p. 158).

One or more additional coders must be enrolled in the study, prior to utilizing the emergent or a-priori coding method, in order to establish that there is sufficient inter-rater reliability to proceed with the main analysis. This includes training the additional coder or coders and check inter-rater reliability via a preliminary analysis (Neuendorf, 2002). If the level of reliability is insufficient (i.e., below the minimal level set by the researcher) the coding framework may need to be revised and/or the second coder(s) retrained. Once a sufficient level of reliability has been established within the preliminary examination, it is time to proceed with the main analysis. The main analysis will also require periodic inter-rater reliability checks to confirm that adequate the required level of inter-rater agreement is maintained.

## **Method**

This study employed content analysis to examine the Division 55 Prescription Exam for Psychologists Review DVD as well the syllabi, and a selection of the required and recommended readings, and two videos from one psychologist postdoctoral psychopharmacology program. These materials were chosen based on the assumption that the program’s syllabi and associated readings accurately reflected the most pertinent aspects of training psychologists to prescribe psychotropic medications. Further, given that a significant portion of this content was purportedly based on the PEP exam’s requirements, it was assumed that the PEP preparatory DVD would offer similar insights

about psychologist postdoctoral psychopharmacology training materials. For example, the PEP exam is said to “Measure credibly the knowledge-base competency necessary to prescribe” (Sammons et al., 2003, pp. 179–180).

Through examining these resources the researcher sought to obtain a clearer picture about the extent to which psychologist postdoctoral psychopharmacology training materials were integrating critiques of neurobiological hypotheses of depression, challenging the rigor of the DSM, and addressing the c/s/x movement. In line with Neuendorf’s (2002) recommendations, the rationale for this study’s methodology stemmed from dialogues with experts in the field, an extensive literature review, and a preliminary analysis of the syllabi and two pharmacology textbooks (required readings) from a psychologist psychopharmacology program’s 2012/2013 curriculum (this curriculum was posted online). In developing the study’s prospectus the researcher communicated with a variety of experts identified within the manuscript’s *Acknowledgements* section, for their support with selecting the three topics of analysis, addressing methodological considerations, and in overcoming initial obstacles associated with the controversial nature of the topic.

Categories for each of the study’s three coding frameworks (i.e., for the DSM, neurobiological etiology of depression, and the c/s/x movement) were developed through reading a variety of written materials from within and outside of the examined psychologist postdoctoral psychopharmacology program’s curriculum. Some examples of non-program materials examined during the formation of coding categories included the *Clinical Handbook of Psychopharmacology* (2008); *Principles and Practices of Psychopharmacology* (2011); the DSM-IV-TR; Irving Kirsch's (2010) *The Emperor’s*



*New Drugs*; Elliot Valenstein's (1998) *Blaming the Brain*; David Healy's (2008) *Mania*; Joanna Moncrieff's (2009) *The Myth of the Chemical Cure*; Thomas Szasz's (2008) *Psychiatry: The Science of Lies*; Judi Chamberlin's (1978) *On Our Own: Patient-Controlled Alternatives to the Mental Health System*; and *Mad Matters: A Critical Reader in Canadian Mad Studies*, edited by Brenda LeFrancois et al. (2013).

Additionally, journal articles from the literature review, and an analysis of c/s/x organizations' websites including but not limited to, [www.MindFreedomInternational.org](http://www.MindFreedomInternational.org), [www.theicarusproject.net](http://www.theicarusproject.net), [www.mhselfhelp.org](http://www.mhselfhelp.org), and [www.psychrights.org](http://www.psychrights.org), were helpful in this regard. Further, the researcher attended two talks by c/s/x activists at the Unitarian Church in Vancouver, British Columbia, and spoke with these and other individuals who identified as c/s/x activists.

The coding framework for the neurobiological etiology of depression was comprised of 12 different hypotheses. In some cases, the researcher elected to combine hypotheses with overlapping mechanisms. For example, neurotrophins, proteins, and second messenger hypotheses were combined into one neurobiologically based theory for the etiology of depression as were the Neuroendocrine and Neuropeptides hypotheses. Similarly, all neurotransmitter hypotheses for depression were combined into one theory as were the neuroanatomical and circuit based hypotheses.

Differences in the nomenclature and approaches used to describe neurobiologically based hypotheses for the etiology of depression made the formation of coding categories more challenging. For example, in *Principles and Practices of Psychopharmacology*, the authors discussed a variety of "interactional theories of depression" that were not identified within the analyzed psychologist postdoctoral

psychopharmacology program's readings (Janicak, Marder, & Pavuluri, 2011). Next, in discussing the neurobiology of depression, Janicak et al.'s (2011) text referred to the "Permissive Hypothesis," "Adrenergic-Cholinergic Balance Hypothesis," and "Bidimensional Model Hypothesis," which were not identified as such within any of the other readings (pp. 206–207). Similarly, Julien and colleagues (2011) text, *A Primer of Drug Action*, was the only analyzed text whose examined readings referred to the "Neurogenic Theory"—a term that Stahl was unfamiliar with (S. Stahl, personal communication, November 5, 2014).

Next, while Stahl noted the role that various facets of the neurogenesis hypothesis (i.e., glutamate, apoptosis, neurotrophins, the neuroendocrine system and the HPA-axis) played in the etiology of depression, he did not explicitly reference the hypothesis within his chapter on depression. Ultimately, however, the researcher chose to interpret this content as implicit references to the neurogenesis hypothesis of depression and coded it accordingly. It is possible that Stahl would disagree with this decision. For example, he might assert that the content pertains to the disorder's pathophysiology versus its causes.

In the process of developing the DSM coding categories, the researcher found associated content ranging from general descriptions of the manual to information about its history/development, strengths/benefits, and criticisms/limitations/controversies. For the c/s/x movement, relevant content tended to focus on the movement's history, organizations, campaigns/events/activism, challenges to the legitimacy of the c/s/x movement, and personal stories of c/s/x activists.

The development of categories continued iteratively during the main analysis such that each reading had the potential to inform which categories were used in subsequent

analyses. This meant that the researcher periodically returned to previously analyzed materials and revise them based upon updated coding categories. Ultimately, the researcher stopped adding or changing categories when it appeared that a sufficient level of saturation had been reached (i.e., the coding categories managed to capture the primary content on the each of the three topics of analysis within each of the successively examined materials).

The researcher gathered data that was relevant to the topics of analysis, albeit falling outside of their respective coding frameworks, within a ‘comments’ section of the excel spreadsheets used for data collection. The researcher ultimately consolidated this information within a narrative summary which provided an overview of that content and the extent to which the information differed from the coded findings.

For additional thoroughness, and upon completion of the main analysis, the researcher searched the examined books’ indices for the following terms:

- ADHD
- Attention Deficit Hyperactivity Disorder
- ADD
- Attention Deficit Disorder
- Affective Disorders
- Anxiety, Bipolar, Consumer
- Depression
- Ex-Patient
- Major Depressive Disorder
- Major Depression

- Mood Disorders
- DSM
- Diagnostic and Statistics Manual of Mental Disorders
- Psychosis
- Psychotic Episode
- Psychotic Disorder
- PTSD
- Post Traumatic Stress Disorder
- OCD
- Obsessive Compulsive Disorder
- Personality Disorders
- Schizophrenia
- Survivor

Next, the researcher reread each of the examined psychologist postdoctoral psychopharmacology program's non-textbook articles. In addition, both of the examined videos were viewed four times.

### **Sampling**

The researcher initially telephoned the Directors of all four psychologist postdoctoral psychopharmacology programs in addition to sending them an email and registered letter (see Appendix D). Three of the programs responded—two agreed to participate in sharing their syllabi and all three Directors were willing to take part in the interview. The Director who was not agreeable to providing syllabi noted that this would go against their institution's policy (personal communication on January 1, and February

7, 2014). However, this Director did send a number of articles and a PowerPoint in response to the researcher's request for program materials that provided nonconventional perspectives pertaining to the three topics of analysis (personal communication on February 7, 2014).

In reviewing the two psychologist postdoctoral psychopharmacology programs which had expressed a willingness to fully participate within the study, the researcher determined that there was insufficient time to analyze them both. Further, it was noted that only one of the program's curriculums contained any readings from critics of conventional psychopharmacology. Additionally, this curriculum contained substantially more primary source articles. Consequently, the latter program was selected because it seemed more likely to have integrated critiques of conventional perspectives.

Required book readings were selected based on the relevance of their titles, and a brief examination of the texts' contents section and indices. The researcher ultimately analyzed ten of the 14 required Non-Semester Project (NSP) books, six of the ten recommended texts, and all six of the semester project books. In addition, all fifty-seven of the non-textbook readings were examined (see Appendices E, F, G, H, I, J, K).

Four of the required NSP book readings and four of the recommended textbook readings were omitted from the analysis for the following reasons (see Appendix L). Here, the researcher assumed that the required *Physical Examination and Health Assessment* (Jarvis, 2011a), *Clinician's Pocket Drug Reference* (Gomella, Haist, & Adams, 2009) and *Stahl's Prescriber's Guide* (Stahl, 2011), were unlikely to contain information about the etiology of depression, the DSM, or the c/s/x movement. One required reading on ADHD from Lewis' (2002) textbook, *Childhood and Adolescent*

*Psychiatry (3rd ed)* was excluded because the researcher was unable to obtain a copy of the book and was later informed by the examined program's Director that the reading was no longer part of the curriculum.

Taber's *Cyclopaedic Medical Dictionary* (Venes, 2013) and the *Pocket Companion for Physical Examination & Health Assessment* (Jarvis, 2011b) were omitted because their respective subject areas were deemed to be outside of the study's analysis. Next, the researcher omitted the recommended *Clinical Handbook of Psychotropic Drugs for Children and Adolescents* (Bezchlibnyk-Butler & Virani, 2007) because an examination of the required version of this text for the prescription consideration pertaining to adults (i.e., *Clinical Handbook of Psychotropic Drugs* by Virani, Bezchlibnyk-Butler, Jeffries, & Procyshyn, 2011) did not address etiological or diagnostic considerations pertaining to the DSM, nor provide any information about the csx movement.

Next, while students were encouraged to read the Carlat Report ([www.thecarlatreport.com](http://www.thecarlatreport.com)), *Journal of Clinical Psychiatry*, and review current literature on the National Library of Medicine's PubMed website ([www.ncbi.nlm.nih.gov/pubmed/](http://www.ncbi.nlm.nih.gov/pubmed/)) this body of information was too large to analyze for this study. It is possible that students would have accessed critical discourse about a neurobiological etiology of depression, challenges to the rigor of the DSM, and information about the c/s/x movement therein. However, a number of factors point to the unlikelihood of this. First, research on reading compliance suggests that only 50% of students actually complete reading assignments prior to a lecture (B. Cooper, 2015). An online survey-based study of 744 graduate students from American Psychological

Association accredited doctoral programs found that that only half of clinical psychology students completed assigned readings. Second, work and family demands can limit the amount of time available for reading (McMinn, Tabor, Trihub, Taylor, & Dominguez, 2009)—a consideration that seems particularly relevant to the population of psychologists returning to complete psychopharmacology training for prescriptive authority. Third, these reading were recommended and non-specific, meaning that students were not required to read these materials and they were not being guided to any specific articles. Consequently, students would need to search as well as read recommended articles with content that they may not be tested on.

In most cases, when the required chapters for a selected book were specified, only those portions of the reading were analyzed. However, in some cases an additional chapter or chapters was/were examined to inform the discussion. These latter cases were not tallied during data analysis. The book was read in its entirety when no specific chapters were identified within the syllabi (i.e., the recommended textbooks). All of the non-textbook readings were examined in their entirety.

### **Tabulation and Reporting**

**Syllabi.** Descriptive statistics were used to identify the types of materials being analyzed (i.e., books, journal articles, primary source studies, reports, guidelines, handbooks, newsletters, videos, magazines, and journals). In addition, the number of referenced works from the researcher's listed critics of conventional psychopharmacology beliefs and practices were tallied.

**Books/Articles.** Descriptive statistics for the examined books and non-textbook readings were formulated to determine which neurobiological hypotheses of depression

were discussed and critiqued. Further, to establish which aspects of the DSM were addressed and whether or not this included any information and critiques about the manual's inter-rater reliability and construct validity. Descriptive statistics were also used to document (a) the extent to which the researcher's listed critics were cited, (b) aspects of the c/s/x movement were discussed, (c) works from c/s/x-identified individuals were included, and (d) critiques of the c/s/x movement were challenged.

**PEP DVD & PEP practice questions.** Descriptive statistics for the PEP Review DVD were calculated by analyzing the PowerPoint's, the PEP Review DVD's supplemental required and recommended readings, as well as the 162 PEP Practice Questions from Dr. Marlin Hoover, for content on the study's three topics of analysis.

**Psychologist postdoctoral psychopharmacology program videos.** Two video presentations from the *Treatment Issues in Psychopharmacology: Affective Disorders* class were analyzed by the researcher. This included the *Biological Basis of Depression* and *Pharmacotherapy for Depressive Disorders*. The researcher used time as a measure for documenting the location and breadth of coded content within the videos.

### **Inter-Rater Reliability**

A second coder was enrolled to establish inter-rater agreement. Requirements for their participation included enrollment within the PsyD Program at Antioch University Seattle and completion of the program's psychopharmacology (PSYCH723) class. In total, the second coder required thirteen hours to train – this included the eight hours of preliminary (pilot) coding to establish sufficient inter-rater reliability (i.e., Cohen's  $Kappa \geq .41$ ) prior to proceeding with the main analysis. The main analysis took the second coder 22 hours to complete.



The researcher randomly selected 30% (n = 7) of the examined books and 30% (n = 11) of the examined articles for the second coder's analysis. By chance, the random selection of texts included two of the Semester Project books. The researcher also chose to include Robert Whitaker's book *Anatomy of an Epidemic* to measure inter-rater agreement for c/s/x content because it was the only training material to address the movement in a substantive manner. In addition, the second coder was provided with a PowerPoint from Marlin Hoover (developer of the PEP Training DVD), which contained a set of 162 Practice Questions for the Psychopharmacology Exam for Psychologists. The second coder was asked to determine whether these questions addressed the study's three topics of analysis.

In total, the second coder examined 25 pages of text for the practice analysis, 214 pages for the main analysis, and all 162 of the aforementioned Practice Questions for the PEP. The textbooks analyzed for the practice analysis included, *Neuroscience: Exploring the Brain* (2006), *Stahl's Essential Psychopharmacology: Neuroscientific Basis and Practical Applications* (2013), *The Myth of the Chemical Cure* (2009), *Prescriptive Authority for Psychologists* (2003), and *The Handbook of Clinical Psychopharmacology* (2008). The second coder was provided with a coding framework and algorithm for each of the three topics and for identifying references and citations (see Appendices A, B, C, D, M, N, O, P).

The researcher omitted the syllabi from the second coder's analysis because there was little indication that any of the study's topics were being addressed within this particular aspect of the curriculum. Consequently, it was determined that the second coder's time would be better spent examining readings that were identified within the

syllabi. The second coder did not analyze two of the program's examined videos because both presentations were obtained after the inter-rater reliability process had been completed. Additionally, the second coder did not analyze the PEP Training DVD because the researcher's own analysis did not find substantive content pertaining to any of the three topics of analysis within these materials. Further, feedback from the DVD's developer, Marlin Hoover, confirmed the researcher's findings in this regard (M. Hoover, personal communications, November 23, and December 10, 2015). Next, it was assumed that the practice test questions—for which inter-rater reliability was examined—would be exemplars of the PEP Training DVD content and, most importantly, represent the knowledge required for psychologists to safely and effectively prescribe.

Inter-rater reliability was determined through calculating Cohen's Kappa via SPSS software. Agreements required that the researcher and second coder selected the same coding categories, critiques, and listed critics. Further, that both coders agreed upon whether the inter-rater reliability/construct validity of the DSM, neurobiological hypotheses of depression, and legitimacy of the c/s/x movement, had been critiqued.

A number of different scales have been developed for interpreting kappa scores. For example, Cohen (1960) asserted that Kappa values of less than zero were indicative of no agreement while values of 0.01–0.20 represented none to slight agreement, kappas of 0.21–0.40 suggested a fair level of agreement, 0.41–0.60 moderate agreement, 0.61–0.80 substantial consensus, and kappas of 0.81–1.00 reflected almost perfect concurrence. Fleiss (1981) consolidated these agreements in recommending that a kappa less than .40 represented poor inter-rater agreement, .40–.75 reflected intermediate to good agreement,

while any kappa over .75 suggested an excellent level of consensus. Altman (1991) subsequently readjusted the kappa scale to include more levels of agreement such that any kappa scores under .20 were described as poor, .21-.40 as fair, .41-.60, as moderate, .61-.80 as good, and .81-1.00 as indicative of very good inter-rater agreement.

This researcher elected to follow Altman's scale for establishing inter-rater reliability levels because it provided more detail than Fleiss' approach. In this regard, at least a moderate level of agreement ( $k = .41$ ) had to be achieved for each of the examined materials during the practice analysis in order that the second coder could proceed with the main analysis.

The researcher initiated the inter-rater reliability process by reviewing the three coding frameworks with the second coder. Next, with support from the researcher, the second coder applied these coding frameworks to a variety of pharmacology textbooks. Once the second coder appeared to understand the coding frameworks the practice analysis was initiated. After establishing that sufficient kappa had been reached in the practice analysis, the second coder proceeded with the main analysis.

Inter-rater reliability checks were also conducted during the main analysis, after each of the materials had been examined, to establish whether sufficient reliability was maintained. The researcher informed the second coder when they had made administrative errors (i.e., if she missed a paragraph's heading that explicitly identified a particular neurobiological hypothesis for depression, or overlooked a citation for a critic or an explicit mention of the DSM). However, these administrative errors were included within the calculations for inter-rater reliability.

### **Random Selection of the Second Coder's Materials**

The second coder's materials were randomly selected by taking a sheet of paper with the readings' titles and cutting each of these titles into a symmetrically shaped piece of paper. These pieces of paper were subsequently placed within the appropriate envelope (i.e., for book readings or non-textbook articles). The second coder then removed 30% of these pieces of paper from each of the two large envelopes. During this process, the second coder selected the *Study Guide to Psychiatry: A Companion to the American Psychiatric Publishing Textbook of Psychiatry* (2009). The researcher chose to omit this reading from the analysis of inter-rater agreement because the second coder had already selected the *American Publishing Textbook of Psychiatry* (2009). Consequently, the second coder randomly selected another title (i.e., *A Primer of Drug Action*, 2011) from the same envelope. The researcher did not include the four *American Academy of Child and Adolescent Psychiatry Practice Parameters* within the random selection process because they were missed during his initial review of the syllabi.

An App was used to generate random page numbers (Random, 2012). These page numbers were only selected if they fell within the parameters of the required or recommended chapters/pages. The random number app was developed and operated by Dr. Mads Haahr, from the School of Computer Science and Statistics at Trinity College in Dublin, Ireland (Random, 2012).

Materials for the second coder were printed, photocopied, or supplied in the original book format. The researcher made sure that there was no highlighting, underlining, or notes from his own analysis within any of the materials being examined by the second coder. The second coder was supplied with a detailed description and

algorithm for each of the coding frameworks as well as an excel spreadsheet for data collection. For instances in which the second coder had questions about a particular section of text, the researcher directed them back to the coding frameworks and algorithms (see Appendices A, B, C, M, N, O, P).

### **Ethical Considerations**

The study had the potential to result in negative perceptions about the participating psychologist postdoctoral psychopharmacology program and its director depending on the findings and peoples' perceptions thereof. To address this potentiality, the study protected the program and its director's anonymity by not mentioning their names or the names of the presenters from the analyzed videos. Additionally, program syllabi were kept in a locked filing cabinet in the researcher's office where they will be stored for seven years and destroyed thereafter.

## Chapter III: Results

### Findings

**Inter-rater reliability.** Inter-rater reliability scores were calculated for the randomly selected materials. This included readings from the analyzed program's required and recommended Non-Semester Project textbooks and Semester Project books. A number of the Non-Textbook readings and all of Hoover's 162 PEP review questions were examined by a second coder to establish inter-rater reliability, as well.

**Practice analysis.** Texts used for the practice analysis included the *Handbook of Clinical Psychopharmacology for Therapists* (2008), *Prescriptive Authority for Psychologists: A History and Guide* (2003), *Myth of the Chemical Cure* (2009), *Neuroscience: Exploring the Brain* (2006), and *Essential Psychopharmacology: Neuroscientific Basis and Practical Applications* (2013). Stahl's (2013) book was the only text from the examined psychologist postdoctoral psychopharmacology program's readings list that was used for practice coding—note that the text was not randomly selected for the main inter-rater reliability analysis. The researcher selected the aforementioned texts to mitigate any practice effects that might have resulted from having the second coder interact with the same books being used for the main analysis.

During the practice and main analyses, the researcher reviewed inter-rater agreement levels subsequent to the completion of each of the materials examined by the second coder. The purpose of evaluating the second coder's assessments was to confirm whether or not sufficient levels of inter-rater agreement had been achieved to warrant the analysis of additional readings.

For the practice analysis, Kappa scores were “good” for the etiology of depression:  $\kappa = .782, p < .0005$  and “very good” for content on the DSM:  $\kappa = .888, p < .0005$  (when agreements for no content were included). To address the possibility that inter-rater agreements for no content might be skewing reliability levels the researcher recalculated kappas subsequent to omitting this data. This resulted in a “good” level of agreement for content on neurobiological hypotheses of depression ( $\kappa = .677, p < .0005$ ), and “good” agreement for DSM content ( $\kappa = .708, p < .0005$ ).

Kappa scores for c/s/x content were in the “good” range when agreements for no content were included ( $\kappa = .773, p < .0005$ ). However, given that the researcher and second coder only documented three and four instances of c/s/x content, respectively, a recalculation of kappa minus no-content-agreements was not completed.

Levels of inter-rater reliability were also calculated within the practice materials for agreements surrounding critiques of neurobiologically based hypotheses of depression, the rigor of the DSM, and the c/s/x movement. Here, levels of agreement were all in the “very good” range with consensus for critiques of neurobiologically based hypotheses of depression yielding a kappa of  $\kappa = .851 (p < .0005)$ . Challenges to the DSM’s rigor ( $\kappa = 1.00, p < .0005$ ), and critiques of the c/s/x movement ( $\kappa = 1.00, p < .0005$ ) both showed perfect agreement. Agreement for critiques of the DSM which had not focused on its rigor also yielded a kappa of  $\kappa = 1.00 (p < .0005)$ . There were no critiques of the c/s/x movement within any of the practice materials. Consequently a kappa score for this variable was not calculated.

There was complete agreement between the researcher and second coder for the absence of listed critics within content that met coding category requirements for each of

the three topics of analysis. In addition, there was full agreement for listed critics whose works were cited on topics outside of the study's analysis (i.e., three listed critics in *Prescriptive Authority for Psychologists: A History and Guide* (2003) and one in *The Myth of a Chemical Cure* (2009)). Both coders identified the same two listed critics whose works were cited on one of the two reference pages examined for the practice analysis.

There were four occasions during the practice coding exercise in which the second coder made an administrative error. In two instances, the mistake occurred because the second coder missed a heading which identified the primary etiology for depression that was discussed. In another case, the second coder interpreted content about Kraepelin as pertaining to the DSM's development. While Kraepelin is inextricably linked to the DSM's origins, this study only focused on content specifically related to development of the DSM itself (as outlined within the DSM coding framework). The second coder's fourth administrative error involved the omission of a listed critic. The administrative errors were treated like any other disagreements within the context of calculating kappas.

**Main analysis.** After completing the practice analysis the second coder moved on to the main analysis. Each reading was analyzed by the researcher in order to confirm that a sufficiently high level of inter-rater agreement had been reached prior to proceeding with the examination of additional randomly selected readings. During this process, the researcher informed the second coder about seven administrative errors the latter had made. For example, the researcher noted that they had missed an explicit



reference to the neurogenesis hypothesis of depression in a reading from *A Primer of Drug Action* (2011),

Additionally, in analyzing *Essential Evidence Based Psychopharmacology* (2012), the second coder was informed that she had incorrectly attributed a comment about the analeptic agent Modafinil's mechanism of action, to the etiology of depression. Next, in analyzing the *American Psychiatric Publishing Textbook of Psychopharmacology* (2009), the second coder missed a neurobiological hypothesis of depression that was explicitly mentioned. There were also two cases within this latter textbook and one case within Moynihan and Cassels (2006) text, *Selling Sickness*, where the second coder overlooked an explicit mention of the DSM. The researcher informed the second coder about these mistakes and treated the errors like any other disagreements when calculating kappas.

Finally, during the coding of *Anatomy of an Epidemic* (2011), the researcher informed the second coder that she had mistakenly identified the Depression and Bi-Polar Support Alliance (DBSA) as a c/s/x organization. The DBSA is not considered a c/s/x organization because it receives up to half of its funding from industry contributions and the majority of this amount has been linked to pharmaceutical companies (Mark, Levit, & Buck, 2009).

Kappa scores for inter-rater agreements within the main analysis were as follows. There was a "good" level of agreement for content on neurobiological etiology's of depression ( $\kappa = .741, p < .0005$ ), and a "moderate" level of inter-rater agreements when concurrences of no content were excluded (i.e.,  $\kappa = .555, p < .0005$ ). Inter-rater agreements for content on the DSM were "good" ( $\kappa = .801, p < .0005$ ), however, when

inter-rater agreements for no-content were omitted the kappa dropped to “moderate” ( $\kappa = .599, p < .0005$ ).

Explicit references to the manual were automatically coded within the general/applications/guidelines coding category for DSM content. Consequently, it was conceivable that agreements for this particular grouping might have been easier to achieve than for the other categories (i.e., development, benefits/merits, or criticisms/limitations/controversies—including challenges to the manual’s rigor). This may have inflated inter-rater reliability levels for DSM content. Consequently, to address this, inter-rater agreements for DSM content were recalculated subsequent to removing all instances in which both coders concurred about coding for the general/applications/guidelines category. This resulted in a “moderate” level ( $\kappa = .455, p < .0005$ ) of inter-rater agreement for DSM content.

Next, both coders agreed that Whitaker’s (2010) book *Anatomy of an Epidemic* was the only randomly selected set of readings to contain information about the c/s/x movement. Here, inter-rater agreement for c/s/x content that met the coding category requirements for this topic were in the “good” range ( $\kappa = .635, p < .0005$ ) unless agreements for no c/s/x content were excluded. In this case the kappa dropped to “moderate” ( $\kappa = .529, p < .0005$ ).

When the comparisons of critiques were based upon the same neurobiological hypotheses of depression that both raters had identified, there was a “good” level of inter-rater agreement for these critiques ( $\kappa = .625, p < .0005$ ). However, if all theories were tallied (i.e., including those where only one rater identified a particular hypothesis) and the lack of a critique for those instances was treated as a non-agreement, the level of

inter-rater reliability dropped to fair ( $\kappa = .367, p < .0005$ ). Nonetheless, consensus about the lack of a critique is arguably still an agreement regardless of whether or not the researchers concurred about the inclusion or exclusion of a particular neurobiological hypothesis of depression within that particular portion of the text.

Similarly, it made sense to include instances in which there was consensus about a critique regardless of whether both coders agreed upon the hypothesis that it represented. In this case, the researcher tallied three instances in which a critique was identified for a neurobiological hypothesis of depression that only one had coder identified. When these three disagreements were included within the analysis of critiques for hypotheses identified by both raters, the level of inter-rater reliability remained at a “good” level ( $\kappa = .719, p < .0005$ ).

Inter-rater agreement for critiques of the DSM was in the “very good” range ( $\kappa = .852, p < .0005$ ). In total, the researcher coded seven pages on which the DSM was critiqued, and these critiques were found within two of the 18 materials with content on this topic that met coding framework requirements. By comparison, the second coder found eight pages in which the DSM had been critiqued within the same two readings. The researcher and second coder agreed that the DSM’s construct validity had been critiqued on six pages of *Selling Sickness* (2006). The researcher and second coder also agreed that the inter-rater reliability of the DSM had been challenged on one page within the paper by Reeves and colleagues (2011).

In another case, both coders agreed that the DSM had been critiqued but only the researcher characterized this critique as having explicitly challenged the manual’s construct validity. Next, there was one occasion in which only the second coder identified

a critique of the DSM—this critique was not characterized as having challenged the manual's rigor.

Neither the researcher nor the second coder found critiques of the c/s/x movement within the examined materials' randomly selected readings (including the randomly selected pages of Whitaker's (201) *Anatomy of an Epidemic*. Both coders agreed on the three instances in which a c/s/x activist's work was cited within Whitaker's book. This included two interviews (one with David Oakes and another with Jim Gottstein) and a reference to John Modrow's (2003) book *How to Become a Schizophrenic*.

In terms of critics, both coders identified David Healy in two works that critiqued the neurotransmitter hypothesis of depression's etiology. Next, the researcher identified five materials (four books and one article) whereas the second coder found four materials (three books and one article), in which listed critics were cited for issues outside of the study's three topics of analysis. The researcher and second coder were in agreement about the names of these listed critics in all cases but one. In this latter instance, only the second coder identified content from Peter Breggin as being related to the c/s/x movement. Next, six reference pages were analyzed for inter-rater agreement and neither coder found any of the listed critics therein.

### **Examined Psychologist Postdoctoral Psychopharmacology Program**

The examined psychologist psychopharmacology program's 2013/2014 curriculum was comprised of 10 classes:

1. Biological Foundations of Psychopharmacological Practice I
2. Biological Foundations of Psychopharmacological Practice II
3. Neuroscience

4. Neuropharmacology
5. Clinical Pharmacology
6. Professional Issues and Practice Management
7. Treatment Issues in Psychopharmacology: Affective Disorders
8. Treatment Issues in Psychopharmacology: Psychotic Disorders
9. Anxiety Disorders
10. Other Disorders

There was a combined syllabus for the *Neuroscience* and *Neuropharmacology* classes and for the *Biological Foundations of Psychopharmacological Practice I and II* courses.

The program's syllabi identified a total of 14 required textbook readings, ten recommended books, and a semester project that had students select one of six books. The curriculum also included 57 required non-textbooks readings, 26 of which were journal articles (see Appendices E, F, G, H, I, J, K). Nine of these journal articles were primary source studies—three focused on county-level estimates of coverage by mental health professionals, one included a comparison between psychotherapy and psychopharmacology, and another focused on the efficacy of psychotherapy. Next, two of the journal articles were studies on the efficacy of antidepressants, and two investigated the effectiveness of neuroleptics.

Of the remaining 17 journal articles, three focused on safe prescribing habits, one provided a critique of the pharmaceutical industry, and two papers discussed the implications of genetic research and testing on psychopathology and diagnosis. There was also a journal article on lab values, another on Warfarin, two journal articles in support of psychologist postdoctoral psychopharmacology training, and a practice

guideline for prescribing psychologists. In addition, there were two journal articles in support of prescriptive authority, one on interpreting health statistics, two on the pharmacological management of agitated patients, and an article on the use of neuroleptics to treat schizophrenia.

Next, there were four practice parameters from the American Academy of Child and Adolescent Psychiatry (i.e., on ADHD, bipolar disorder, depressive disorders, anxiety disorders, respectively), one practice guideline on PTSD from the Department of Defense Veterans Affairs, seven prescription algorithms, five papers with safety recommendations for avoiding prescription related medical errors, two assessment templates, one template for documenting a treatment plan, a template for clinical notes, one prescription template, a measurement conversion chart, and two reports (one on mental health surveillance in the US and another on prescribing psychologists' integration of psychotherapy and psychopharmacology).

The non-textbook reading materials also included a list of resources for drug references, an unpublished "Fact Sheet for Psychologists Prescribing Psychotropic Medications," an unpublished article on Monoamine Oxidase Inhibitors, a newsletter focusing on Serotonin Syndrome, and one magazine article on prescribing. Additionally, there was a TedTalk video (its transcription was used for this study) which discussed how unreported negative findings from drug trials could limit physicians' knowledge about medications and, by proxy, negatively impact a doctor's prescribing habits. There were also a total of 56 video presentations (see Appendix M for a complete list of video topics).

For this study, the researcher analyzed ten of the 14 required Non-Semester Project (NSP) books, six of the ten recommended texts, all six of the Semester Project (SP) books and each of the 57 required non-textbook readings. Four of the required readings were omitted from the analysis because the researcher assumed that the required *Physical Examination and Health Assessment* (2012), *Clinician's Pocket Drug Reference* (2009) and *Stahl's Prescriber's Guide* (2011) were unlikely to contain information about the etiology of depression, the DSM, or the c/s/x movement. Next, a required reading on ADHD by Joseph Biederman within Lewis' (2002) textbook, *Childhood and Adolescent Psychiatry (3rd ed)* was excluded because the researcher was unable to obtain a copy of the book. Further, the researcher was informed by the examined program's Director that this particular reading had been removed from the curriculum.

In terms of the recommended readings, *Taber's Cyclopaedic Medical Dictionary* (2009) and the *Pocket Companion for Physical Examination & Health Assessment* (2011) were omitted from the analysis because their respective subject areas were outside of the study's topics of focus. Next, the *Clinical Handbook of Psychotropic Drugs for Children and Adolescents* (2007) was excluded from the examination because the researcher's analysis of the *Clinical Handbook of Psychotropic Drugs* (2011) did not yield any substantive findings pertaining to a neurobiological etiology of depression, the DSM, or the c/s/x movement. Robert Whitaker, David Healy, and Daniel Carlat, each authored a text within the list of six recommended Semester Project books.

Finally, the researcher reviewed two of the Program's 56 videos (i.e., *Biology of Affective Disorders* and *Neuropharmacology of Antidepressants and Mood Stabilizers* (see Appendix M). The two videos were 40 minutes and 15 seconds and 39 minutes and

50 seconds long, respectively. The researcher did not review the other 54 videos due to time constraints and the assumption that these presentations were unlikely to contain any substantive information about the three topics of analysis.

## **Syllabi**

**Neurobiological etiology of depression.** The combined syllabus for *Biological Foundations of Psychopharmacological Practice I & II*, noted within the course description that “The goals of these two courses are to enhance the student’s recognition of signs and symptoms of medical conditions requiring collaboration with and referral to other health professionals and to provide knowledge about the psychological, biological, and medical correlates of disease” (Psychologist Postdoctoral Psychopharmacology Program Syllabus, 2014a, p. 4). There were no specific references to depression within this syllabus.

No references to the etiology of depression were identified within the combined *Neuroscience* and *Neuropharmacology* syllabus. However, the table of contents included a Module on *Neurobiology and Pathophysiology* (Psychologist Postdoctoral Psychopharmacology Program Syllabus, 2013a). Further, the combined *Neuroscience* and *Neuropharmacology* course description stated that “Knowledge of principles of neuroanatomy, neurophysiology, neurochemistry and neuropathology will serve as the foundation for understanding of neurotransmitter systems and their role in the etiology and treatment of mental and neurodegenerative disorders” (p. 4). Next, the course objectives for this syllabus noted that students would learn to “Identify gross anatomical structures of the central and autonomic nervous systems, and surrounding structures. For



each structure, identify associated function, major dysfunction, and associated pathology” (p. 4).

The combined *Neuroscience* and *Neuropharmacology* syllabus also indicated that students would “Describe and correlate the major neurochemical systems associated with mental disorders, and psychotropic medications” (Psychologist Postdoctoral Psychopharmacology Program Syllabus, 2013a, p. 4). Next, the objectives for Module 7 (*Neuropharmacology*) stated that “Neuropeptides as neurotransmitters and/or neuromodulators” would be addressed (2013a, p. 26). In addition, the directed study questions for Module 7 queried students about the HPA-Axis and the mechanism by which Peptides A and B regulate levels of intracellular cAMP (2013a, pp. 26–27).

The *Clinical Pharmacology* syllabus outlined a “Semester Project”—an assignment that “was designed in light of the theme for this semester, which is the creation of your personal identity as a critical user of pharmacology in clinical practice” (Psychologist Postdoctoral Psychopharmacology Program Syllabus, 2013b, p. 8). For this assignment, students were instructed to “critically evaluate the case made by the author” and to conduct “A critical analysis of central arguments” (p. 8). While there were no specific references to the etiology of depression within this syllabus, the semester project contained the titles of six books (students were required to select one) indicative of works that might provide critical perspectives on neurobiological hypotheses for depression. The syllabus also noted that “[The semester] project is meant to be fun! Don’t be too concerned about grading. The intent here is to get you thinking about how you think pharmacotherapy should be integrated into psychology, where standard practice needs to be fixed, and what it means to be a prescribing psychologist” (2013b, p. 9).

Next, the syllabus for *Professional Issues and Practice Management* noted within its course description that “genetic factors in psychopathophysiology” would be addressed (Psychologist Postdoctoral Psychopharmacology Program Syllabus, 2013c, p. 3). A description for the Semester Project was also included within this syllabus. The *Treatment Issues in Psychopharmacology: Affective Disorders* syllabus indicated that the “Biological Basis for Affective Disorders” would be covered within Module 1 (2014b, p. 2). The course description noted that “Biological models of the disorders are reviewed, as are classes of medications appropriate to the treatment of mood dysfunction” (2014b, p. 3).

Course objectives for this class noted that students would learn to “Describe both current and recently popular models used to explain the etiology of affective disorders from a biological perspective” (Psychologist Postdoctoral Psychopharmacology Program Syllabus, 2014b, p. 1). The syllabus’ learning objectives for *Module 1: Biological Basis of Affective Disorders* stated that the course would “Describe the proposed theories of the biochemical basis of depression” (p. 12). In addition, Module 1 and Module 2 for this course required that students watch a video on the *Biological Basis of Affective Disorders* and the *Pharmacotherapy of Depression*, respectively (p. 4). The researcher did not find content pertaining to a neurobiological etiology of depression within the *Psychotic Disorders* (Psychologist Postdoctoral Psychopharmacology Program Syllabus, 2014c), *Anxiety Disorders* (Psychologist Psychopharmacology Program Syllabus, 2013d), or *Other Disorders* (Psychologist Postdoctoral Psychopharmacology Program Syllabus, 2013e) syllabi.

**DSM.** There were no explicit mentions of the DSM within the combined syllabus for *Biological Foundations of Psychopharmacological Practice I & II*. However, the syllabus for this course included an assessment template for documenting a client's "History and Physical Format." This document also included a section for recording "Past Psychiatric History" (Psychologist Postdoctoral Psychopharmacology Program Syllabus, 2014a, p. 12). In addition, this combined syllabus contained an assignment titled "History and Physical Evaluation", which included a section for documenting a client's psychiatric history. These documents were also included within the combined syllabi for the *Neuroscience* and *Neuropharmacology* classes (Psychologist Postdoctoral Psychopharmacology Program Syllabus, 2013a).

As noted earlier, the *Clinical Pharmacology* (Psychologist Postdoctoral Psychopharmacology Program Syllabus, 2013b) and the *Professional Issues and Practice Management* (Psychologist Postdoctoral Psychopharmacology Program Syllabus, 2013c) syllabi both contained identical descriptions of the Semester Project. While none of the Semester Project readings explicitly referred to the DSM or psychopathology, the books' titles were indicative of works that might address diagnostic issues and provide non-conventional perspectives about the manual.

A course description for the *Treatment Issues in Psychopharmacology: Affective Disorders* syllabus stated that "The course assumes a working knowledge of the Diagnostic and Statistical Manual criteria for affective disorders, as well as presentation, manifestations, and differential diagnosis of these categories" (Psychologist Postdoctoral Psychopharmacology Program Syllabus, 2014b, p. 3). The syllabus also contained a

document titled “Case Presentation Format (Mood Disorder)”, which had a section allocated for identifying “DSM Diagnosis: Axis I-III” (p. 10).

The *Treatment Issues in Psychopharmacology: Psychotic Disorders* syllabus reiterated the expectation that students already possess skills in utilizing the DSM (Psychologist Postdoctoral Psychopharmacology Program Syllabus, 2014c, p. 3). In addition, the syllabus contained the “Case Presentation Format” template mentioned within the previous paragraph (p. 11). The learning objectives for this class’ third Module (Antipsychotic Medications) noted that students would “Review symptomatology and pathophysiology of psychosis” (p. 15).

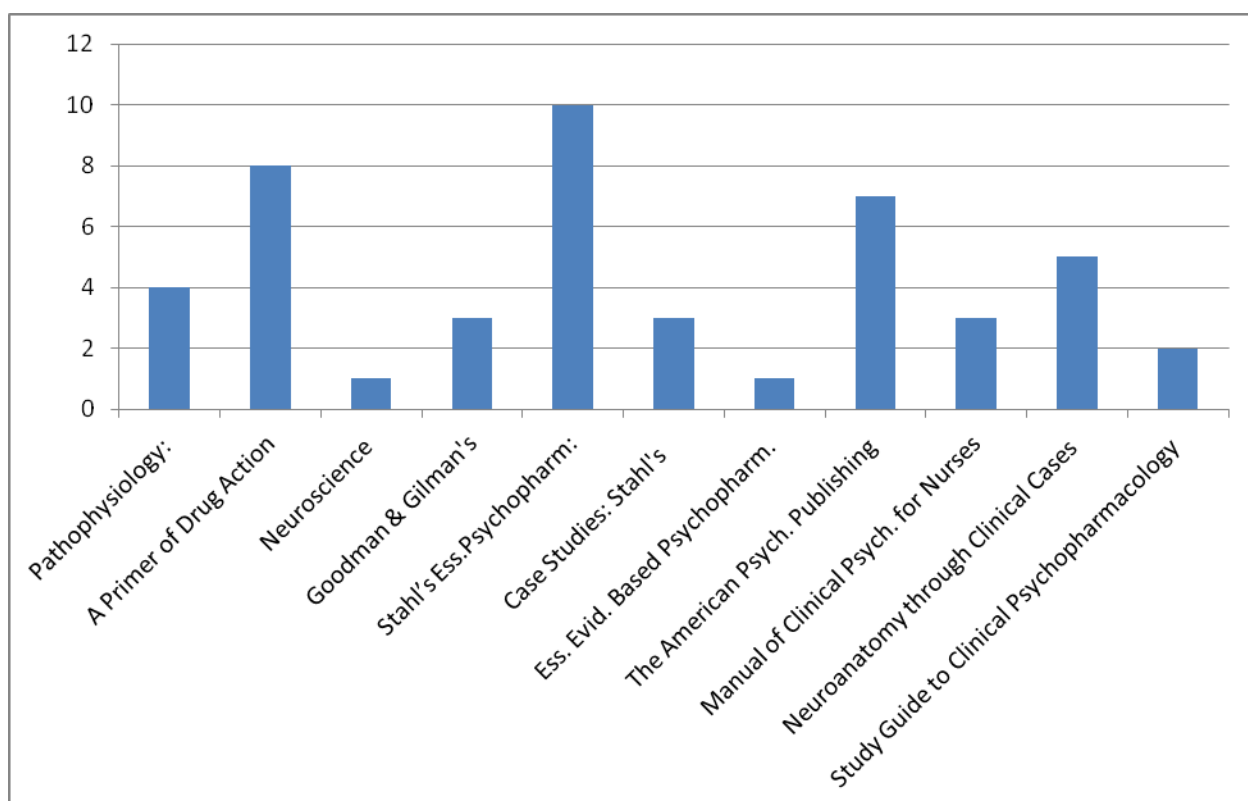
The *Anxiety Disorders* syllabus reiterated the aforementioned “Case Presentation Format” document and the stipulated expectation surrounding prior experience with using the DSM (Psychologist Postdoctoral Psychopharmacology Program Syllabus, 2013d, pp. 10–11). In addition, the learning objectives for Module 1: *Anxiety Disorders Overview* noted that students “Should fully understand and be able to discuss each of the following: . . . diagnostic classification and prevalence of anxiety disorders: Evolution of standardized classification systems (i.e., ICD and/or DSM) and implications for neurobiology” (p.13). Next, the learning objectives for this course’s third Module: *Special Populations and Genomics* stated that students would have opportunities to “Discuss population differences that can affect diagnosis and treatment of anxiety disorders” (Psychologist Postdoctoral Psychopharmacology Program Syllabus, 2013d, p. 16). In Module 4: *Confounding Drugs and Disease States*, the syllabus noted that “the physical work-up and differential diagnosis for general medical conditions and substance related confounders in anxiety disorders” would be reviewed (p. 17).

The syllabus for the *Other Disorders* class repeated the expectations that students be familiar with the DSM and the course also included the “Case Presentation Format” (previously document herein) with space for identifying DSM diagnoses (Psychologist Postdoctoral Psychopharmacology Program Syllabus, 2013e, pp. 9–10).

**C/S/X.** There were no explicit references to the c/s/x movement, its organizations, or works by c/s/x activists within the syllabi. However, it was plausible that the topic might be broached within one or more of the Semester Project’s books based on their respective titles and authors. Next, Module 5 for the *Professional Issues and Practice Management* class noted within its learning objectives that students would “Address issues of diversity as they impact on our knowledge and practice of pharmacotherapy” — a comment that could be interpreted as indicative of c/s/x content despite the fact that it did not meet the coding category frameworks for this topic (Psychologist Postdoctoral Psychopharmacology Program Syllabus, 2013c, p. 17).

### **Non-Semester Project Textbooks**

**Etiology of depression.** The 16 Non-Semester Project (NSP) textbooks collectively addressed twelve different neurobiologically based hypotheses for the etiology of depression (i.e., neurotransmitters, neuroanatomy/circuits, neuroendocrine system/neuropeptides, genetics, neurotransmitter receptors, glutamate, neurotrophins, neurogenesis, circadian rhythms, immunological-cytokines, enzymes, and GABA). For a visual representation of the number of neurobiological hypotheses addressed within each of the 16 NSP textbook’s examined readings see Figure 1 and Appendix O.



*Figure 1.* Number of neurobiological hypotheses for the etiology of depression addressed within examined non-semester project textbooks and meeting coding category requirements.

**Note:**

= Number of hypotheses presented

Pathophysiology: The Biological Basis for Disease, 6th ed. (2010) (23 chapters)

A Primer of Drug Action, 12th ed. (2011) (11 chapters)

Essential Psychopharmacology: Neuroscientific Basis & Practical Applications, 4<sup>th</sup> ed. (2013) (9 chapters)

The American Psychiatric Publishing Textbook of Psychopharmacology (2009) (16 chapters)

Neurosciences, 5th ed. (2012) (7 chapters; 8 pages from 3 other chapters; Appendix and Atlas)

Goodman & Gilman's: The Pharmacological Basis of Therapeutics, 12th ed. (2010) (38 chapters; Appendix 1)

Pharmacotherapy for Psychologists (2010) (5 chapters)

Case Studies: Essential Psychopharmacology (2011) (10 chapters)

Essential Evidence Based Psychopharmacology, 2<sup>nd</sup> ed. (2012) (3 chapters)

Clinical Handbook of Psychotropic Drugs 19<sup>th</sup> ed. (2011) (Entire Book Examined)

Manual of Clinical Psychopharmacology for Nurses (2013) (Entire Book Examined)\*

Pharmacotherapy: A Pathophysiological Approach, 8<sup>th</sup> ed. (2011) (2 chapters) \*

Seeley's Anatomy & Physiology, 9<sup>th</sup> ed. (2011) (Entire Book Examined)\*

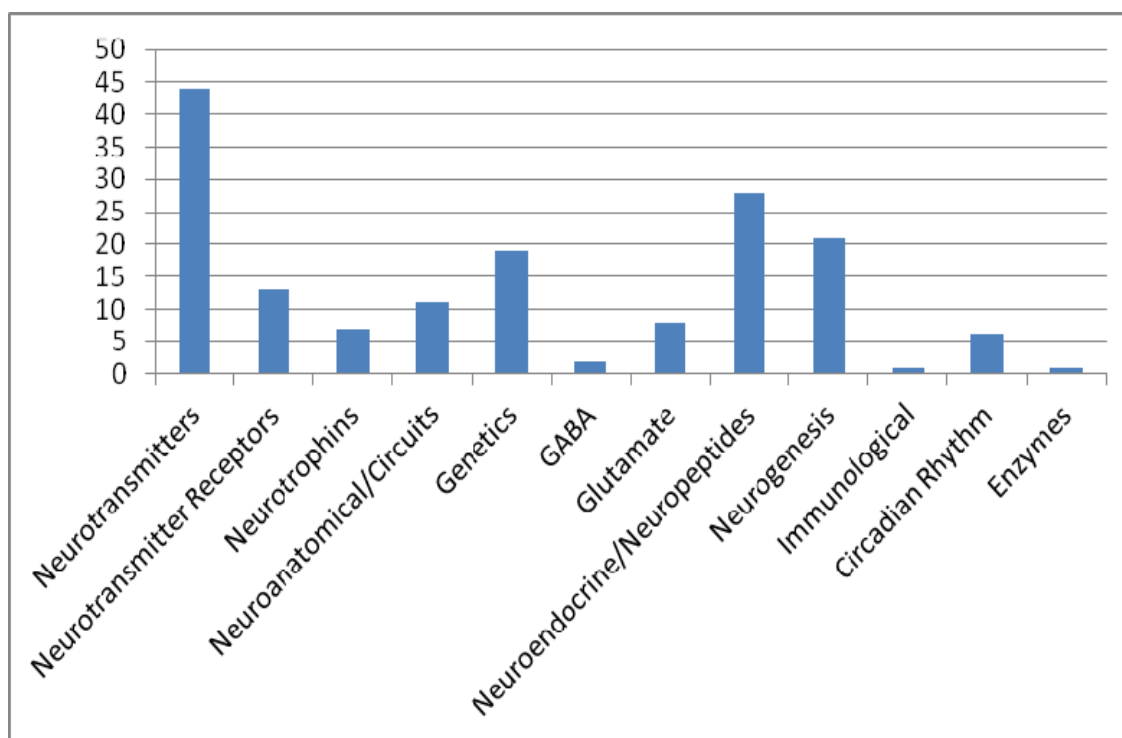
Atlas of Functional Neuroanatomy, 2<sup>nd</sup> ed. (2005). (Entire Book Examined)\*

Neuroanatomy Through clinical cases, 2nd ed. (2010). (Entire Book Examined)\*

Study guide to Clinical Psychopharmacology: A Companion (2009). (16 chapters)\*

\* = Recommended

There was some variability in the extent to which each of the neurobiological hypotheses of depression was discussed across the NSP books (see Figure 2). For example, the neurotransmitter hypothesis was collectively addressed on 44 pages of these texts in a manner that met the coding framework requirements for this topic. By contrast the neuroendocrine/neuropeptides hypothesis was discussed on 28 pages, genetics (19 pg's), neurogenesis (21 pg's), neuroanatomical/circuits (11 pg's), neurotransmitter receptors (13 pg's), neurotrophins (7 pg's), glutamate (8 pg's), circadian rhythms (6 pg's), immunological-cytokines (1 pg.), enzymes (1 pg.), and GABA hypothesis on one page of text, in a manner that met the coding framework requirements.

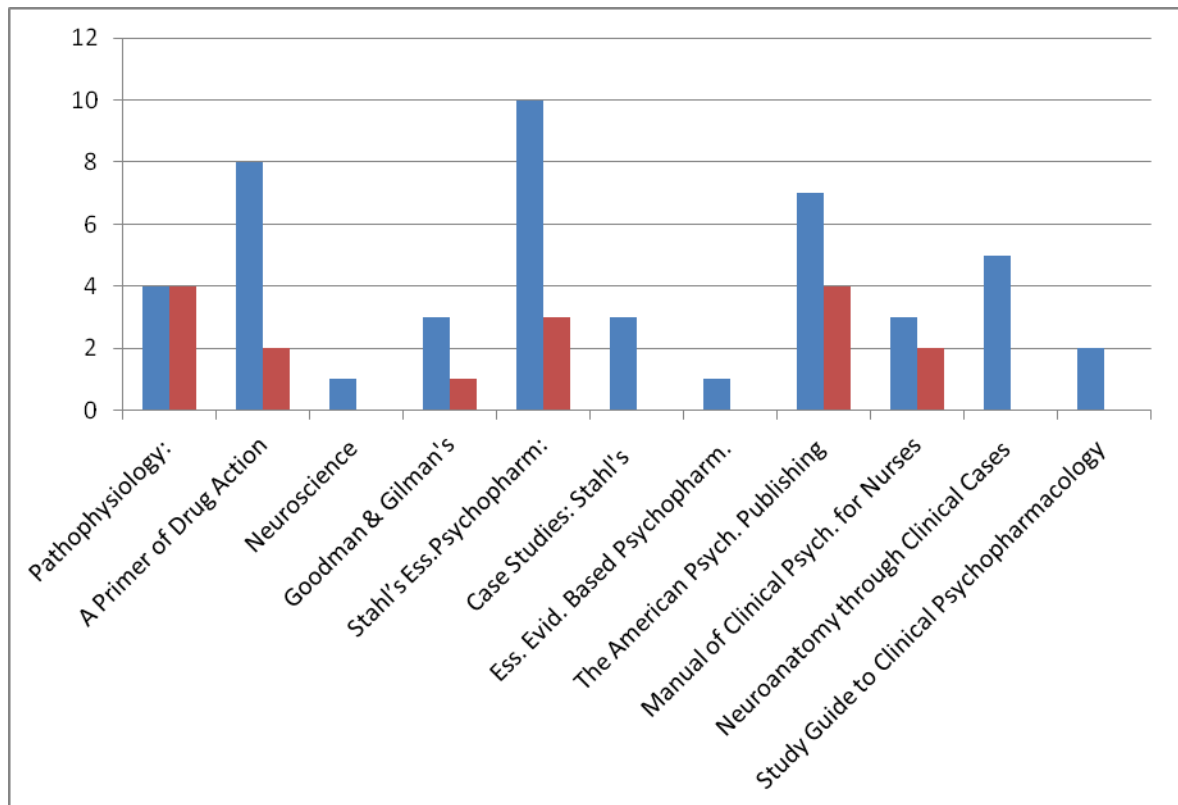


*Figure 2.* Total number of pages for neurobiological hypotheses of depression content that met coding category requirements within examined non-semester project books.

Eleven of the 16 examined NSP textbooks addressed one or more neurobiological hypothesis of depression and six (54%) of these texts provided critiques for one or more

of the neurobiological hypotheses for depression discussed. *Pathophysiology: The Biological Basis for Disease in Adults and Children* (2010) was the only NSP textbook to critique all four of the neurobiological hypotheses of depression addressed therein—these hypotheses and critiques were all focused on Pre-Menstrual Dysphoric Disorder. See Figure 3 for a visual representation of the number of hypotheses discussed and critiqued within each of the analyzed NSP books readings.





■ = Number of hypotheses presented

■ = Number of hypotheses critiqued

*Figure 3.* Number of neurobiological hypotheses for the etiology of depression addressed and critiqued within examined non-semester project books, and meeting coding category requirements.

Pathophysiology: The Biological Basis for Disease, 6th ed. (2010) (23 chapters)

A Primer of Drug Action, 12th ed. (2011) (11 chapters)

Essential Psychopharmacology: Neuroscientific Basis & Practical Applications, 4<sup>th</sup> ed. (2013) (9 chapters)

The American Psychiatric Publishing Textbook of Psychopharmacology (2009) (16 chapters)

Neurosciences, 5th ed. (2012) (7 chapters; 8 pages from 3 other chapters; Appendix and Atlas)

Goodman & Gilman's: The Pharmacological Basis of Therapeutics, 12th ed. (2010) (38 chapters; App. 1 examined)

Pharmacotherapy for Psychologists (2010) (5 chapters examined)

Case Studies: Essential Psychopharmacology (2011) (10 chapters examined)

Essential Evidence Based Psychopharmacology, 2<sup>nd</sup> ed. (2012) (3 chapters)

Clinical Handbook of Psychotropic Drugs 19<sup>th</sup> ed. (2011) (Entire Book Examined)

Manual of Clinical Psychopharmacology for Nurses (2013) (Entire Book Examined)\*

Pharmacotherapy: A Pathophysiological Approach, 8<sup>th</sup> ed. (2011) (2 chapters) \*

Seeley's Anatomy & Physiology, 9<sup>th</sup> ed. (2011) (Entire Book Examined)\*

Atlas of Functional Neuroanatomy, 2<sup>nd</sup> ed. (2005). (Entire Book Examined)\*

Neuroanatomy Through Clinical Cases, 2nd ed. (2010). (Entire Book Examined)\*

Study guide to Clinical Psychopharmacology: A Companion (2009). (16 chapters examined)\*

\* = Recommended

Examined Non-Semester Project books with analyzed required readings that discussed one or more neurobiologically based hypotheses for the etiology of depression included, *Pathophysiology: The Biological Basis for Disease, Neurosciences* (2010), *Primer of Drug Action* (2011), *Goodman & Gilman's: The Pharmacological Basis of Therapeutics* (2010), *Stahl's Essential Psychopharmacology* (2013), *Neurosciences* (2012), *Essential Evidence Based Psychopharmacology* (2012), *Case Studies: Essential Psychopharmacology* (2011), *American and the Psychiatric Publishing Textbook of Psychopharmacology* (2009). Recommended books with readings that discussed one or more neurobiologically based hypotheses for the etiology of depression included the *Manual of Clinical Psychopharmacology for Nurses* (2013), *Neuroanatomy Through Clinical Cases* (2010) and the *Study Guide for Psychopharmacology* (2009)—the former two of these three books were read in their entirety. The researcher did not find content on a neurobiological etiology of depression within the examined chapters of the required *Pharmacotherapy for Psychologists* (2011) or within *Pharmacotherapy: A Pathophysiologic Approach* (2011), *Clinical Handbook of Psychotropic Drugs* (2011), *Seeley's Anatomy and Physiology* (2011) and, the *Atlas of Neuroanatomy* (2005)—all three of these latter books were examined in their entirety.

The ratio of hypotheses to critiques for the examined chapters of the six NSP textbooks that incorporated challenges for neurobiologically based hypotheses of depression were as follows: *Pathophysiology: The Biological Basis for Disease* = 4:4, *A Primer of Drug Action* = 8:2, *Essential Psychopharmacology: Neuroscientific Basis & Practical Applications* = 10:3, *American Psychiatric Publishing Textbook of Psychopharmacology* = 7:4, *Goodman and Gillman's: The Pharmacological Basis of*

*Therapeutics* = 3:1, and the *Manual of Clinical Psychopharmacology for Nurses* = 3:2 (see Figure 3).

Heterogeneity was also reflected in the number of pages containing information and critiques for neurobiological hypotheses of depression within the analyzed NSP textbook readings (see Figure 3). For example, a neurotransmitter hypothesis of depression was absent in some texts but addressed with varying degrees of depth on up to 18 pages in other books. Further, neurobiological based hypotheses of depression were critiqued on one to three pages of the six NSP books that provided challenges for these hypotheses. The following paragraphs will discuss each of the six NSP books that contained one or more neurobiological hypothesis of depression and at least one critique of one or more of these hypotheses.

In *Pathophysiology: The Biological Basis for Disease*, four neurobiological hypotheses for premenstrual disorders were discussed (i.e., neurotransmitters, neuroendocrine, genetics, GABA) (McCance, Huether, Brashers, & Rote, 2010). The authors critiqued each of these hypotheses with one summative statement (i.e., “the mechanisms involved are not known”) (Latendresse, McCance, & Morgan, as cited in McCance et al., 2010, p. 826). In a discussion about fibromyalgia, the textbook also noted the “major role for neuroendocrine and stress response alterations” in mood disorders, and stated that “Altered circadian activity of several neuroendocrine axes and ANS dysfunction have been reported (Martinez-Lavin, 2007)” (Crowther-Radulewicz & McCance, as cited in McCance et al., 2010, p. 1607). This latter comment about a neuroendocrine-based hypothesis for depression was not critiqued.

In *A Primer of Drug Action*, Julien and colleagues (2011) discussed eight neurobiological hypotheses for depression and challenged two (i.e., the neurotransmitter and neurotransmitter receptor hypotheses). The neurotransmitter hypothesis was critiqued on two of the four pages in which it was discussed (i.e., pp. 142, 150). Here, the authors noted that the lag time in antidepressants' effects was "a weakness of this model" (p. 142), and stated that the "[chemical imbalance] interpretation is much too simplistic" (p. 150). The neurotransmitter receptor hypothesis was also critiqued via the latter sentence.

In *Essential Psychopharmacology: Neuroscientific Basis and Practical Applications* (2013), ten neurobiological hypotheses for the etiology of depression were identified. This included the neurotransmitter hypotheses of depression which were discussed to varying degrees on 18 pages—three of these pages contained a critique of this hypothesis. For example, Stahl (2013) noted that the initial conceptualization of the monoamine hypothesis was "rather simplistic" and stated that definitive evidence in support of the hypothesis was "still largely lacking" (p. 262).

In addition, Stahl (2013) noted that efforts to uncover monoamine deficiencies in depression had "yielded mixed and sometimes confusing results" (p. 262), and that there was "no clear and convincing evidence that monoamine deficiency accounts for depression—i.e., there is no 'real' monoamine deficit" (p. 266). In addition, the textbook posited that the lag time for antidepressants' results presented a "problem" for this hypothesis (p. 290).

Next, Stahl (2013) addressed the neurotransmitter receptor hypothesis of depression in seven pages and posited that direct evidence for the hypothesis "is also

generally lacking” (p. 266). In addition, the text noted inconsistencies and replication issues with neuroimaging data used to support this hypothesis (p. 266). A genetic source for depression was challenged on three of the 13 pages in which this particular premise was discussed. Each of these critiques acknowledged that a single gene for depression had not been identified. More specifically, it was noted that the specific genotype for SERT “accounts for only a small amount of the variance” of the disorder’s etiology (p. 272). The text also posited that genetics might predispose a person towards depression, particularly with the addition of environmental stressors. Additionally, Stahl discussed glutamate, neuroendocrine/neuropeptide, neurogenesis, circadian rhythm, and enzyme hypotheses for the etiology of depression, but without critiques.

In the examined sections of Schatzberg and Nemeroff’s (2009), *American Psychiatric Publishing Textbook of Psychopharmacology*, four of the seven neurobiological hypotheses of depression discussed were also critiqued (i.e., the neurotransmitter receptor, genetic, glutamate, and neurogenesis theories of depression were challenged). Critiques were integrated within one of the three pages addressing the neurotransmitter receptor hypothesis, one of four pages that discussed a genetic neurobiological etiology for depression, one of three pages which outlined the glutamate hypothesis for depression, and within two of four pages examining the neurogenesis hypothesis.

Next, a critique of the neurotransmitter receptor hypothesis stated that “In regions postsynaptic to ascending raphe neurons such as prefrontal cortex, altered levels of 5-HT1A receptor binding have been found in the prefrontal cortex of depressed suicide victims (Matsubara et al., 1991), although conflicting evidence exists (Arranz et al., 1994;

Cheetham et al., 1990)” (Robinson, Rickels, & Yocca, as cited in Schatzberg & Nemeroff, 2009, p. 488).

Genetic correlates for depression were discussed within four pages and critiqued on one page of the examined chapters from *American Psychiatric Publishing Textbook of Psychopharmacology* (2009). The critique occurred within a discussion linking the disorder to mutated gene coding for the purinergic receptor P2X7. Here, the textbook posited that “The function of the P2X7 receptors in the context of depression is not clear” (Holsboer, as cited in Schatzberg & Nemeroff, 2009, p. 521). The glutamate hypothesis for the disorder was addressed within three pages of text (i.e., pp. 25, 513, 514) and critiqued on one of these pages. In this instance, the textbook noted that “Although not always conclusive, in vivo magnetic resonance imaging (fMRI) studies have revealed altered glutamate levels in various brain areas (Auer et al., 2000; Sanacora et al., 2004) as well as in the CSF (Frye et al., 2007) of depressed patients” (p. 513).

Next, the author addressed the proposed role of neurogenesis in depression to varying degrees on four pages of the examined portions of *The American Psychiatric Publishing Textbook of Psychopharmacology* (Holsboer as cited in Schatzberg & Nemeroff, 2009) (i.e., pp. 493, 519, 520, 521). In this case, critiques for the hypothesis were provided on two pages. For example, the text posited that “altered plasma BDNF concentrations were reported in affective disorders (Machado-Vieira et al., 2007; Shimizu et al., 2003), although the source of this neurotrophin in blood remains to be elucidated” (Schatzberg & Nemeroff, 2009, p. 519). Further, in a discussion about neurogenesis, the text stated that “serum GDNF concentration is decreased in depressed

individuals, but it is not entirely clear from where this neurotrophic factor derives” (p. 521).

In the *Goodman and Gilman's: The Pharmacological Basis of Therapeutics* readings, three neurobiological hypotheses of depression were discussed (i.e., neurotransmitter, genetics, and neuroendocrine/neuropeptides) (Brunton, Chabner, & Knollman, 2010). The neuroendocrine/neuropeptides hypothesis was critiqued within one of two pages on which the hypothesis was addressed. Here, the textbook stated that “the physiological/ pathophysiological relevance of these findings is controversial, and some actions of vasopressin on memory and learned behavior may be due to visceral autonomic effects” (p. 708).

The *Manual of Clinical Psychopharmacology for Nurses* (2013) (a recommended reading examined in its entirety) addressed three hypotheses (i.e., neurotransmitters, neuroanatomical/circuits, neuroendocrine/ neuropeptides) on five, two, and two pages, respectively (Leahy & Kohler, 2013). This textbook critiqued two of the three hypotheses presented. For example, in addressing the neurotransmitter and neuroendocrine hypotheses for Pre-Menstrual Dysphoric Disorder, the textbook noted that:

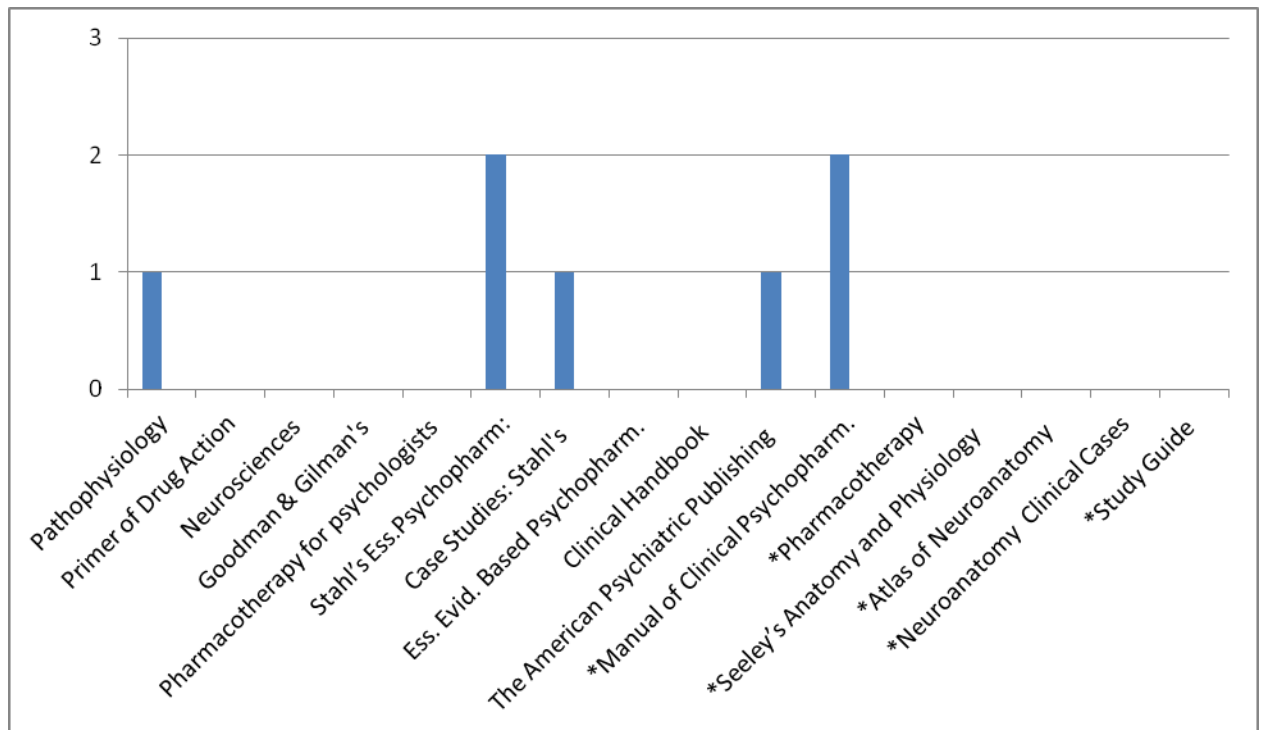
it has long been believed that PMDD is caused by a complex set of interactions between the woman's reproductive hormones (progesterone and estrogen) and the brain's neurotransmitters, especially serotonin. Because the selective serotonin reuptake inhibitor (SSRI) antidepressants have been used to treat PMDD and have shown efficacy, this would seem to hold true; however, this interaction is not yet fully understood. (Josey & Neidert, as cited in Leahy & Kohler, 2013, p. 66)

The neuroanatomy/circuits hypothesis for PMDD (i.e., abnormalities in the orbitofrontal cortex and amygdale) was not challenged.

**DSM.** Findings for the number of pages with DSM content that met coding requirements within the analyzed Non-Semester Project textbooks' examined readings are as follows (also see Appendix R). Eleven of the 16 NSP textbooks contained information about the DSM. This information was disseminated on 121 pages of text and focused primarily on discussing the manual in general terms, as well as describing its guidelines, and applications. The development of the manual was discussed to varying degrees on 26 pages and its benefits and strengths were identified on three pages.

There were 13 pages on which criticisms, limitations and/or, controversies about the manual were found (see Figure 4). Six of these pages identified problems with the manual's construct validity. The researcher found one instance in which the DSM's reliability was challenged. The researcher did not find critiques of the manual's inter-rater reliability and there were no examples of statistics pertaining to the DSM's empirical rigor. No references were made to the researcher's listed critics within the context of discussing the DSM in the examined pages of the analyzed NSP texts.





■ = Number of pages with content critiquing the DSM's construct validity (CV)

■ = Number of pages with content critiquing the DSM's inter-rater reliability (IRR)

*Figure 4.* Number of examined non-semester project textbooks' analyzed pages that contained challenges to the DSM's construct validity or inter-rater reliability, that met coding category requirements.

Pathophysiology: The Biological Basis for Disease, 6th ed. (2010) (23 chapters)

A Primer of Drug Action, 12th ed. (2011) (11 chapters)

Essential Psychopharmacology: Neuroscientific Basis & Practical Applications, 4<sup>th</sup> ed. (2013) (9 chapters)

The American Psychiatric Publishing Textbook of Psychopharmacology (2009) (16 chapters)

Neurosciences, 5th ed. (2012) (7 chapters; 8 pages from 3 other chapters; Appendix and Atlas)

Goodman & Gilman's: The Pharmacological Basis of Therapeutics, 12th ed. (2010) (38 chapters; Appendix 1)

Pharmacotherapy for Psychologists (2010) (5 chapters)

Case Studies: Essential Psychopharmacology (2011) (10 chapters)

Essential Evidence Based Psychopharmacology, 2<sup>nd</sup> ed. (2012) (3 chapters)

Clinical Handbook of Psychotropic Drugs 19<sup>th</sup> ed. (2011) (Entire Book Examined)

Manual of Clinical Psychopharmacology for Nurses (2013) (Entire Book Examined)\*

Pharmacotherapy: A Pathophysiological Approach, 8<sup>th</sup> ed. (2011) (2 chapters) \*

Seeley's Anatomy & Physiology, 9<sup>th</sup> ed. (2011) (Entire Book Examined)\*

Atlas of Functional Neuroanatomy, 2<sup>nd</sup> ed. (2005). (Entire Book Examined)\*

Neuroanatomy Through clinical cases, 2nd ed. (2010). (Entire Book Examined)\*

Study guide to Clinical Psychopharmacology: A Companion (2009). (16 chapters)\*

\* = Recommended

In examining the 23 required chapters of *Pathophysiology: The Biological Basis for Disease* (2010), the researcher found four pages with DSM content. Two of these pages addressed the DSM-IV's diagnostic criteria for Premenstrual Dysphoric Disorder (PDD) and identified problems associated with PDD's construct validity. For example, the textbook noted that "inconsistent and overlapping use of terminology and criteria are used to describe these syndromes." (Latendresse, McCance, & Morgan, as cited in McCance et al., 2010, p. 826). In addition the authors asserted, "many women with clinically relevant premenstrual syndrome/premenstrual Dysphoric disorder (PMS/PMDD) symptoms do not meet the full criteria of the DSM-IV" (p. 827). The other two pages of DSM content within this textbook focused on the diagnostic criteria for anorexia nervosa, albeit without any critiques (McCance et al., 2010).

The DSM was addressed within four pages of the eleven examined chapters from *A Primer of Drug Action* (2011). This included two pages which discussed the diagnostic criteria of a major depressive episode, and one page on which differences in the diagnostic category for anxiety prior to the DSM-III were noted. The fourth page identified the manual in a discussion about prescriptive authority, which the authors noted could add to psychologists' "armamentarium of treatment modalities for DSM-IV diagnoses" (Julien et al., 2011, p. 682). One page (i.e., p.128) within the *Neurosciences* (2012) readings referred to the DSM's definition of addiction. Next, in discussing the "Diagnosis Phase" of a "psychobiosocial" approach, *Pharmacotherapy for Psychologists* identified the DSM on two pages of text within the five chapters examined for this study (McGrath & Moore, 2010, p. 110). The manual was both critiqued and supported via the statement that the DSM and ICD "although helpful, are limited in many ways" (p. 110).

Additionally, the authors asserted that “A critical analysis of these diagnostic systems is tangential to the substance of this chapter” (p. 110).

*Stahl’s Essential Psychopharmacology: Neuroscientific Basis & Practical Applications* (2013), addressed the DSM in a manner consistent with the study’s coding framework requirements, on 12 pages of the nine chapters analyzed. For example, Stahl referred to the manual in discussions about “The Symptom Dimensions of Schizophrenia” (p. 79), “The Bipolar Spectrum” (pp. 247–249), in distinguishing unipolar depression from bipolar depression (i.e., p. 250), in identifying the “Mixed states of mania and depression” (p. 253), in proposing the “Symptoms and circuits” of depression and mania (pp. 273, 277, 278, 279) and in presenting a “Symptoms Based Algorithm for Antidepressants” (pp. 355, 356). There were two pages in which Stahl challenged the DSM’s construct validity. Here, the text discussed instances in which a patient experienced a manic or hypomanic episode while taking an antidepressant. For example, Stahl (2013) noted that:

Patients who develop a manic or hypomanic episode on antidepressants are sometimes called bipolar III. According to formal diagnostic criteria, however, when an antidepressant causes mania or hypomania, the diagnosis is not bipolar disorder, but rather, ‘substance induced mood disorder.’ Many experts disagree with this designation and feel that patients who have a hypomanic or manic response to an antidepressant do so because they have a bipolar spectrum disorder, and can be more appropriately diagnosed as bipolar III disorder. (p. 247)

Next Stahl (2013) referenced the manual within the context of discussing the Research Domain Criteria (RDoC) which the author described as the “future of psychiatry” and an improvement over the DSM which describes “categorical syndromes that mix together many symptoms” (p. 474). There were two pages within the ten examined chapters of *Case Studies: Essential Psychopharmacology* that contained

content about the DSM which met the study's coding requirements for this topic (Stahl, 2011a). The first was a case in which a pharmacological intervention was being proposed for an individual who "probably has never reached the threshold of experiencing unequivocal hypomania as defined by DSM-IV or ICD-10" (p. 72). In the second reference to the manual, Stahl questioned the DSM's construct validity with respect to its conceptualization of antidepressant induced mania. Here, the author queried whether "Despite DSM-IV is Substance Induced Mood Disorder a form of bipolar disorder that should be treated as such?" (p. 352).

Content about the DSM meeting coding framework criteria within the 17 analyzed chapters of *The American Psychiatric Publishing Textbook of Psychopharmacology*, focused on a variety of topics (Schatzberg & Nemeroff, 2009). For example, the text referred to the manual's definition of "substance (drug) dependence" (Sheehan & Raj, as cited in Schatzberg & Nemeroff, 2009, p. 474) and provided the criteria for the DSM's substance dependence disorder (i.e., p. 475). The DSM was also briefly referenced within discussions about research on psychotropic prescription drug abuse and the efficacy of benzodiazepines, antidepressants, and stimulants. Here, the DSM-II, DSM-III, DSM-III-R, and DSM-IV were cited in discussions about studies' methodologies (i.e., pp. 475, 476, 489, 1182, 1185, 1186, 1322, 1418).

For example, the text referred to the DSM-II and DSM-III-R within the context of citing research that investigated the efficacy of anxiolytic's for anxiety and depression (Schatzberg & Nemeroff, 2009, pp. 491, 496). Next, the manual was referenced in discussing OCD and its categorization as an anxiety disorder. The authors for this chapter also questioned whether the neurobiology of each DSM-IV-TR anxiety disorder was

distinct or representative of “subtle variations of a shared etiology?” (Amiel, Mathew, Garakani, Neumeister, & Charney, as cited in Schatzberg & Nemeroff, 2009, p. 979).

In chapter 49 (*Neurobiology of Substance Abuse and Addiction*), Kennedy and Kilts briefly mention the DSM-IV-TR in discussing drug addiction (as cited in Schatzberg & Nemeroff, 2009, p. 1007). Next, the manual was cited in a discussion about research on the prevalence and population estimates of individuals diagnosed with psychiatric disorders (p.1020). In chapter 51 (*Neurobiology of Personality Disorders*), Lee and Coccaro addressed the characteristics of personality disorders represented within the DSM-IV-TR and ICD-10 (as cited in Schatzberg & Nemeroff, 2009, p. 1045). In chapter 55, *Treatment of Schizophrenia*, the criteria for the disorder and some aspects of its Kraepelinian and post-Kraepelinian evolution were described (i.e., p. 1137).

The first of two critiques of the DSM (that met the study’s coding criteria) occurred within the context of discussing the psychopharmacological treatment of general anxiety disorder (Schatzberg & Nemeroff, 2009). Here, in addressing the evolution of GAD’s diagnostic criteria, Davidson, Connor, and Zhang questioned the disorder’s construct validity noting that:

A degree of uncertainty still hangs over the most appropriate way to classify GAD [General Anxiety Disorder]. Even as DSM-IV was being crafted debate centered around the extent to which GAD could be separated from mood disorders, such as dysthymia and major depression. This question has never been well resolved, and it is possible that with so much in common between GAD and depressive disorders, its classification primarily as an anxiety disorder may change in DSM-V. (as cited in Schatzberg & Nemeroff, 2009, p. 1182)

This chapter also mentioned the inclusion of Acute Stress Disorder within the context of changes to the DSM’s conceptualizations of trauma and associated nosology (p.1188).

Additionally, *The American Psychiatric Publishing Textbook of Psychopharmacology* briefly referred to the DSM-IV-TR in a discussion about incidence rates for Nicotine use (i.e., p. 1215) and in mentioning the manual's "course specifier 'on agonist therapy'" within the context of discussing the use of methadone or buprenorphine for maintenance treatment of opioid dependence (O'Brien & Dackis, as cited in Schatzberg & Nemeroff, 2009, p. 1221). The text also noted the manual's diagnostic criteria for primary insomnia (i.e., pp. 1254, 1255, 1256).

The second and last critique of the DSM within the examined readings for this textbook was located within the chapter, *Treatment of Childhood and Adolescent Disorders* (Wagner & Pliszka, as cited in Schatzberg & Nemeroff, 2009). Here it was posited that "Although DSM-IV-TR criteria are used to diagnose bipolar disorder in youths, the clinical features in children may differ from those in adolescents and adults" (Wagner & Pliszka, as cited in Schatzberg & Nemeroff, 2009, p. 1315). This chapter also referenced the DSM in discussing obsessive compulsive disorder, general anxiety disorder, social anxiety disorder, separation anxiety disorder, post-traumatic stress disorder, oppositional defiance disorder, Tourette's, schizophrenia, autistic disorders and other pervasive developmental disorders in children (i.e., pp. 1319, 1320, 1322, 1323, 1324, 1330, 1335, 1340, 1341).

The researcher found 57 pages of text within the *Manual of Clinical Psychopharmacology for Nurses* (2013) that addressed the DSM in a manner consistent with coding criteria for this topic. This book was examined in its entirety. The text's forward noted that "In particular, contents of each chapter focus on diagnostic criteria and neurobiology of relevant disorder(s)" (Leahy & Kohler, 2013, p. xxvi). The text reviewed

diagnostic criteria for anxiety disorders, depressive disorders, bipolar disorders, psychotic disorders, attention deficit/hyperactivity and autism spectrum disorders, substance use disorders, sleep wake disorders, Post-Traumatic Stress Disorder, and delirium, to varying degrees on 55 pages.

The book also discussed the DSM-IV-TR's *Outline for Cultural Formulation and Glossary of Culture-Bound Syndromes*, (i.e., pp. 390, 391, 394) (Leahy & Kohler, 2013). Next, the manual was cited in discussing psychiatric emergencies (i.e., pp. 310, 317, 320, 321). In this context, the text identified the DSM criteria for substance intoxication, withdrawal and delirium, and in defining personality disorders. The manual was also cited within instructions for the *Adult ADHD Self-Report Scale* (i.e., p. 455).

There were six instances in which content met the study's coding criteria for the DSM's "development" category (i.e., pp. 85, 130, 131, 156, 236, 237) (Leahy & Kohler, 2013). In one of these instances the text noted that "In recent years, many developments in diagnosing and treating bipolar disorder have emerged" (Knight, as cited in Leahy & Kohler, 2013, p. 85). Here, Knight acknowledged that questions remained about whether the concept of bipolar illness was best conceptualized as a discrete category or a spectrum disorder. In addition, the book's chapter on *Psychotic Disorders* posited that changes in the DSM diagnostic criteria for delusional disorders had complicated attempts to compare research results across time (i.e., p. 130). Finally, the manual's development was discussed within the context of addressing the evolving diagnostic criteria for attention deficit hyperactive disorder (i.e., p. 156), post-traumatic stress disorders (i.e., p. 236, 237), and acute stress disorder (i.e., p. 237).

The researcher found four critiques of the DSM within the *Manual of Clinical Psychopharmacology for Nurses* (2013). For example, Knight noted that the question of whether or not a categorical approach to diagnosing bipolar disorder was preferable to a dimensional method “had been the subject of considerable debate” (as cited in Leahy & Kohler, 2013, p. 89). Further, the potential benefits and pitfalls of expanding the definition to include bipolar spectrum disorder were considered.

One of the four critiques of the DSM within this text could be interpreted as a challenge to the manual’s construct validity and inter-rater reliability (Leahy & Kohler, 2013). For example, in the chapter on *Bipolar Disorders* Knight posited that “About 69% of all patients with bipolar disorder are inaccurately diagnosed” (as cited in Leahy & Kohler, 2013, p. 89). However, the text did not clarify what the source of the inaccurate diagnosis was (i.e., a problem with the manual or insufficient training of the clinician). Next, in discussing ADHD, the textbook noted that “ADHD has been a diagnosis fraught with much controversy” (Varley & Leahy, as cited in Leahy & Kohler, 2013, p. 156). At the same time, Varley and Leahy asserted that “Today we know that the symptoms related to ADHD are neurodevelopmental in origin. Despite the controversies, the core symptoms of the disorder—reduced attention span, distractibility, difficulty focusing, and hyperactivity—have remained throughout” (as cited in Leahy & Kohler, 2013, p. 156).

The researcher found three pages of text with content that met the coding criteria for the DSM within one of the two chapters examined in *Pharmacotherapy: A Pathophysiologic Approach* (2011). In the chapter, *Evaluation of Psychiatric Illness*, Schneiderhan, Nelson, and Munro provided an overview of the manual’s origins and development, and the important role it played within the context of mental health care (as



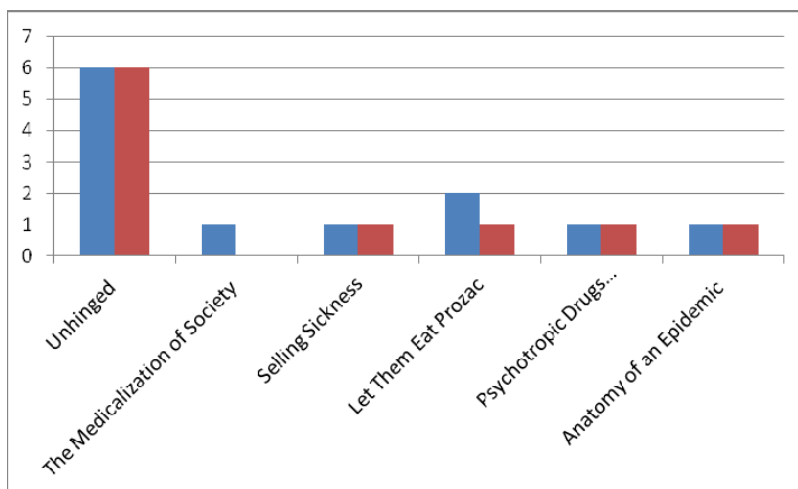
cited in Dipiro et al., 2011, p. 1077, 1078). Additionally, it was noted that “The DSM-IV-TR contains many components that provide a comprehensive understanding of specific mental illnesses and assist in making an accurate diagnosis” (Schneiderhan et al., as cited in Dipiro et al., 2011, p. 1078).

In the 17 chapters and six pages examined within the *Study guide to Clinical Psychopharmacology: A Companion*, the researcher found that the DSM-III, DSM-III-R, and DSM-IV were cited on page 350 within the context of discussing drug trials for PTSD.

**C/S/X.** The researcher did not find content about the c/s/x movement within the analyzed Program’s examined Non-Semester Project textbook readings.

### **Semester Project Textbooks**

**Etiology of depression.** One of the six Semester Project (SP) books presented six neurobiological hypotheses for depression’s etiology, while another examined two hypotheses, and four of the SP books discussed one hypothesis. There was one case in which a SP textbook did not critique a hypothesis (i.e., genetic) that was discussed (i.e., *Let Them Eat Prozac* (2003)) (see Figure 5 and Appendix P).



■ = Number of hypotheses presented  
■ = Number of hypotheses critiqued

*Figure 5.* Number of neurobiological hypotheses for the etiology of depression and critiques for these hypotheses meeting coding category requirements within semester project books.

Table 1

*Number of Pages With Content on a Neurobiological Etiology of Depression and Critiques for These Hypotheses (in brackets) Meeting Coding Category Requirements within Semester Project Books*

SP Book Title	NT	NTRE	NTROP	N/C	GEN	GABA	GLUT	NE/NP	NGEN	IMM	CR	ENZ
Unhinged	10(9)	0	1(1)	2(2)	2(2)	0	1(1)	1(1)	0	0	0	0
Medicalization of Society	0	0	0	0	1	0	0	0	0	0	0	0
Selling Sickness	5(5)	0	0	0	0	0	0	0	0	0	0	0
Let Them Eat Prozac	20(13)	0	0	0	1	0	0	0	0	0	0	0
Psychotropic RX Guide	1(1)	0	0	0	0	0	0	0	0	0	0	0
Anatomy of an Epidemic	21(15)	0	0	0	0	0	0	0	0	0	0	0

*Note.* Number of pages with critiques of each hypothesis identified within brackets ( ). NT: Neurotransmitter; NTREC: Neurotransmitter Receptor; NTROP: Neurotrophins/Proteins; N/C: Neuroanatomy/Circuits; GEN: Genetics; GABA; GLUT: Glutamate; NE/NP: Neuroendocrine/Neuropeptides; NGEN: Neurogenesis; IMM: Neuromodulators/Inflammation/Neuro-immunological; CR: Circadian Rhythm; EN: Enzymes; AD: Adrenergic-Cholinergic Balance Hypothesis; 2<sup>nd</sup> Messengers; NV: Neurovascular

The number of pages with content on a neurobiological hypothesis for depression within the Semester Project books ranged from zero to 21 (see Table 1). Critiques for these hypotheses were disseminated on between 65% (i.e., 13/20) and 100% of the pages on which they premises were addressed. The neurotransmitter hypothesis was covered by five of these books and it was also the most commonly discussed neurobiological hypothesis therein (i.e., the neurotransmitter hypothesis of depression was discussed to varying degrees on 57 pages of these books). Additionally, the chemical imbalance hypothesis was critiqued on nine of the ten pages which addressed the hypothesis within *Unhinged* (Carlat, 2010). In *Selling Sickness* (Moynihan & Cassels, 2006) all five of the pages addressing this hypothesis included a critique. Thirteen of 20 pages addressing the chemical imbalance hypothesis of depression in *Let Them Eat Prozac* also contained a critique (Healy, 2003). Next, the neurotransmitter hypothesis of depression was mentioned and challenged on one page within the *Psychotropic Drug Prescriber's Survival Guide* (Dubovsky & Dubovsky, 2007), and on 15 of 21 pages within *Anatomy of an Epidemic* (Whitaker, 2010).

Carlat's (2010) book also dealt with the neurotrophic (i.e., 1 page), neuroanatomy/circuits (i.e., 2 pages), genetics (i.e., 2 pages), glutamate (i.e., 1 page), and neuroendocrine/neuropeptides (i.e., 1 page) hypotheses of depression. In all but one case (i.e., one of the ten pages on which the neurotransmitter hypothesis was discussed) Carlat critiqued the neurobiological hypotheses for depression's etiology that were addressed.

For example, in challenging the neurotransmitter hypothesis of depression the text stated that "we are still far away from a true understanding of the biological causes of [depression]" (Carlat, 2010, p. 5) and, "no specific defect had been identified in this

regard” (p. 6). Carlat also quoted the *American Psychiatric Association Publishing Textbook of Psychopharmacology* (2009) as having asserted that the “central question of what variables drive the pathophysiology of mood disorders remains unanswered” (as cited in Carlat, 2010, p. 6). Next, in referencing the chemical imbalance hypothesis, Carlat asserted that “direct evidence of such a deficiency is lacking” (p. 7).

Similarly, Carlat (2010) challenged the veracity of neuroimaging findings typically used to support neurobiological hypotheses of depression. *Unhinged* also discussed the methodological limitations of measuring neurotransmitter levels within body fluids and the use of this data to support neurobiological hypotheses of depression. Next, Carlat posited a colleague’s supposition that longstanding maladaptive behaviors were more likely to be the source of depression than any “chemical imbalance” (p. 29).

In further critiquing the monoamine hypothesis of depression (i.e., pp. 74, 75, 76, 77, 78, 82) Carlat (2010) pointed to “increasingly elaborate theories about ‘downstream’ effects of antidepressants, in which neurotransmitters set into motion a cascade of biochemical events that eventually cause genes to be activated” (p. 78). In addition, Carlat noted that delays in antidepressants’ effects effectively undermine hypotheses that are based upon the existence of chemical imbalances.

In one of the two pages within *Unhinged* that addressed a neuroanatomical/circuits hypothesis of depression, Carlat (2010) questioned the relevance of various research findings that have been linked to the disorder (i.e., “decreased activity in the frontal lobe, a shrunken hippocampus, an oversized amygdalae, disrupted circuits around the basal ganglia, and miscellaneous abnormalities in the thalamus and the pituitary gland” (p. 6). In the second page afforded to discussing a

neuroanatomical/circuits hypothesis for the disorder, Carlat deconstructed an outdated hypothesis about the purported connection between depression and “temporal lobe epilepsy” (p. 40).

Next, genetic correlates for a neurobiological hypothesis of depression were discussed on two pages of *Unhinged*, with Carlat (2010) asserting that genes undoubtedly play a role in mental disorders. However, the text also posited that research findings linking specific genotypes to the disorder have been inconsistent (i.e., pp. 80, 81). The glutamate, neuroendocrine, neurotrophin and neurotransmitter hypotheses of depression were supported and critiqued within the summative statement that “Researchers have found evidence of abnormalities in serotonin, norepinephrine, dopamine, cortisol, thyroid, growth hormone, glutamate, and brain derived neurotrophic factor—yet no specific defect has been identified” (p. 6).

In *Selling Sickness*, Moynihan and Cassels (2005) discussed and critiqued the neurotransmitter hypothesis for depression to varying degrees on five pages of text. For example, the authors stated that:

As specialists in mental illness remind us, the idea that depression is caused by a deficiency of the brain chemical serotonin is in fact just one scientific view among many— and a simplistic and outdated one at that. But it is a theory kept very much alive by the massive marketing machinery that starts with the morning deliveries of pharmaceutical company sales representatives. (p. 23)

In the same vein, Moynihan and Cassels (2005) cited Healy in their assertion that “early theories suggesting that a serotonin imbalance cause’s depression have not been verified by later research” (p. 27). The authors also posited that pharmaceutical companies had a vested interest in disseminating this hypothesis to promote the sale of SSRIs (pp. 28, 136). Next, in discussing attention deficit disorder, the text noted “As with

depression, there is much scientific uncertainty about whether these difficulties are primarily due to biological and chemical problems in the brain, or are the result of a complex interplay of physical, social, cultural, or economic factors” (p.64).

In *Let Them Eat Prozac*, Healy (2003) referred to the neurotransmitter hypothesis for depression to varying degrees on 20 pages of text and challenged the hypothesis on 13 of these pages. For two of these critiques, Healy (2003) cited his own work (2000, 2001). In challenging the neurotransmitter hypothesis of depression Healy stated that:

This mass creation of depression affects even those who are not depressed. It is now widely assumed that our serotonin levels fall when we feel low, and that this lowering is thought to have consequences for everything from diet to criminality. But there is no evidence for any of this, nor has there ever been. The huge gap between what is scientifically demonstrable and what people believe points to a cultural fact that lies well beyond the ‘medicalization’ so worrying to sociologists and bio-ethicists. (p. 13)

In discussing the origins of the chemical imbalance hypothesis, Healy (2003) acknowledged that the premise made sense in principle, based upon SSRI’s purported mechanism of action. However, the text also reiterated that a reduction in serotonin levels among depressed individuals was unconfirmed. Further, in describing the concept of low norepinephrine levels linked to depression, Healy stated that this hypothesis “could be supported by some but not all the evidence” (p. 46).

In addition, Healy (2003) challenged the chemical imbalance hypothesis by positing how “By 1970, Ashcroft had concluded that whatever was wrong in depression, it was not lowered serotonin. More sensitive studies had shown no lowering of serotonin in depression. Indeed, no abnormality of serotonin metabolism in depression has ever been demonstrated” (2003, p. 47). The book also posited that historic research documenting the benefits of Isoniazid for depression had undermined the legitimacy of

neurotransmitter hypothesis early on since the medication has no connection to monoamines.

Further, Healy (2003) noted that reducing serotonin appeared to as effective as increasing the neurotransmitter for treating the disorder. Next, Healy (2003) posited that “If Prozac was a better serotonin reuptake inhibitor than older antidepressants and if serotonin was lowered in depression, why wasn’t Prozac bringing about recoveries in hospital depressions quicker than older drugs?” (p. 129). Moreover, Healy stated that “The mythologies underpinning Prozac cast it as an antibiotic of the mind raising lowered serotonin levels to normal, when its use is in fact more like treating mild hypertension” (p. 379).

*Let Them Eat Prozac* also referred to the “modern rhetoric of depression” and contrasted the high degree of evidence supporting the use of pharmacological interventions for bacterial infections versus the treatment of depression with antidepressants (Healy, 2003, p. 326). In addition, the book documented the role that British popular media had played in propagating this information without sufficient critiques. For example, Healy stated that “In a way unimaginable, ten years ago, popular culture takes it for granted that serotonin is low in depressed people” (p. 374). Finally, in describing the evolution of depression as a disorder, Healy briefly introduced the concept of an “endogenous depression” and noted that this “implied a genetic depression” (p. 25). However, the researcher could not find any explicit critiques within *Let Them Eat Prozac* for a genetic based neurobiological etiology of depression.

In, *Medicalization of Society*, Conrad (2007) discussed a genetic based neurobiological hypothesis of depression on one page. Here the author stated that:

Medicalization can obscure the social forces that influence well-being. For example, by focusing completely on the neurobiological features of depression, this condition is viewed increasingly as being genetic, and it is treated predominantly with antidepressants, while the social environments that frequently feed depression are not altered. (Horwitz, 2002, p. 152)

While the book did not explicitly challenge the rigor of a genetic hypothesis for depression, Conrad did note the implications of viewing the disorder exclusively through this lens. For example, the text posited that “The focus on the individual has reinforced the proclivity of treating complex societal problems with technological fixes (e.g., a medical, surgical, or pharmaceutical intervention) rather than by changing the social structure” (p. 153).

*Psychotropic Drug Prescriber’s Survival Guide* contained content on the neurotransmitter hypothesis for the etiology of depression on one page and challenged this premise by noting mixed results from early Reserpine trials. For example, the text indicated that this medication had been shown to cause as well as treat depression (Dubovsky & Dubovsky, 2007). The authors also described how the French tricyclic antidepressant tianeptine was thought to work by decreasing the amount of serotonin in the brain, thereby challenging traditional assumptions about the nature of chemical imbalances and SSRI’s mechanism of action. Next, Healy, a listed critic, was cited in critiquing the neurotransmitter hypothesis (i.e., “The rapid ascendance of a theory that had mixed empirical support emerged from a single observed action of a few medications and ‘an emerging eclipse of clinical observation by laboratory based world-views’” (Healy, 1999, as cited in Dubovsky & Dubovsky, 2007, p. 6).

In *Anatomy of an Epidemic*, Whitaker (2010) addressed one neurobiological hypothesis (neurotransmitter hypothesis) for the etiology of depression on 21 of the



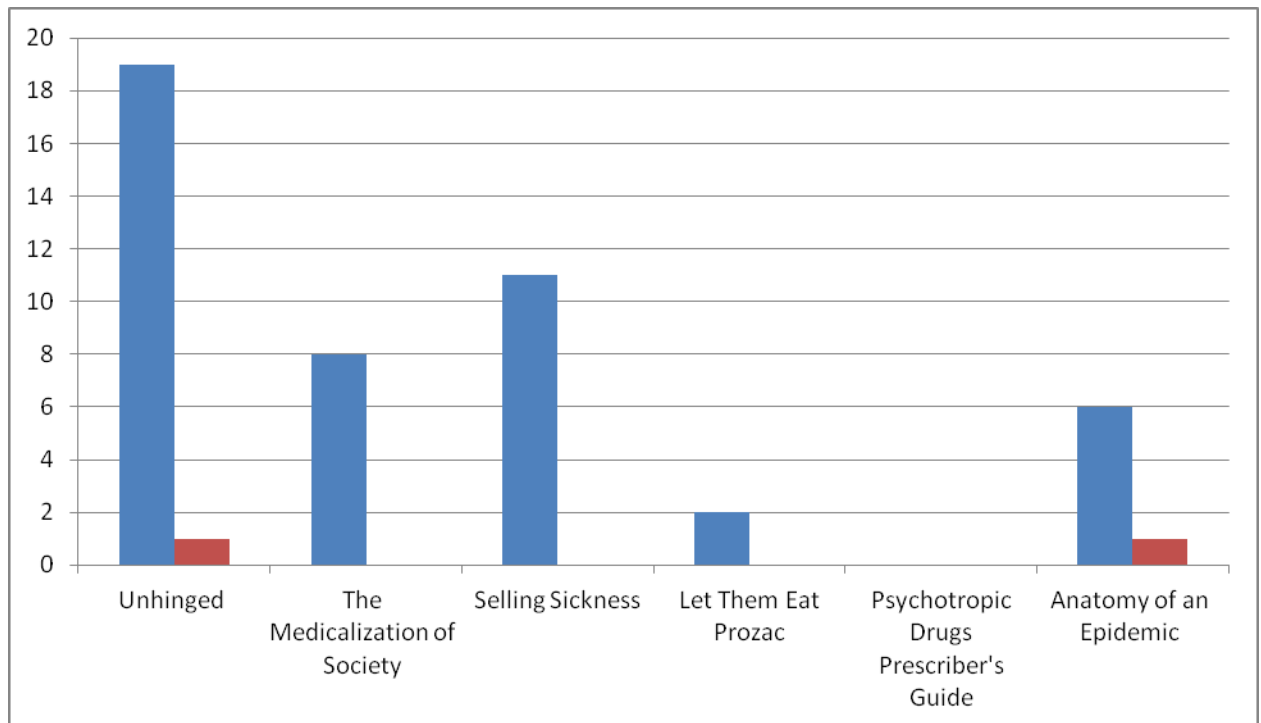
book's pages. Critiques for the hypothesis were discussed to varying degrees on 15 of these pages. Whitaker (2010) introduced the concept of a neurobiological based etiology of depression by noting the origins of the chemical imbalance hypothesis (i.e., in the 1950s with Brodie and Carlson's research on reserpine and Joseph Schildkraut's conceptualization of their work in 1965). The hypothesis was challenged with a quote by Schildkraut who asserted that the premise was "at best a reductionistic oversimplification of a very complex biological state" (as cited in Whitaker, 2010, p. 63).

Whitaker (2010) began the fifth chapter, *The Hunt for Chemical Imbalances*, with a quote by Thomas Huxley: "The great tragedy of science—the slaying of a beautiful hypothesis by an ugly fact" (Whitaker, p.77, chapter five). *Anatomy of an Epidemic* went on to discuss the complexity of the human brain and noted the limitations of research investigating neurotransmitter levels within the cerebrospinal fluid of depressed individuals. This included a description of Mendels and Frazer's work in 1974 which had reviewed and critiqued Schildkraut's hypothesis.

Here, Whitaker quoted Mendels (1974) in stating that "the depletion of brain norepinephrine, dopamine or serotonin is in itself not sufficient to account for the development of the clinical syndrome of depression" (as cited in Whitaker, 2010, p. 72). The text also described how a similar conclusion about serotonin was reached in 1984 by NIMH researchers investigating the plausibility of 5-HIAA levels being linked to amitriptyline's mechanism of action. Later in the book, works by listed critics Lacasse, Healy, and Glenmullen were cited in the process of challenging the monoamine hypothesis (Lacasse, 2005; and Healy, 2002, 2005; and Glenmullen, 2000, as cited in Whitaker, 2010). No other neurobiological hypotheses for depression were addressed.

The researcher did not find content about the neurotransmitter receptor, GABA, neurogenesis, circadian rhythm, immunological-cytokines, and enzymes/cofactors hypotheses within any of the Non-Semester Project books.

**DSM.** In total, the six Semester Project textbooks contained 107 pages on which the DSM was explicitly mentioned—this included general information about the manual, its guidelines, and applications (see Appendix S). Sixty-eight of these pages contained information about the DSM’s development, and 21 pages included content about the manual’s merits. Fifty-four of the 107 contained criticisms, limitations and/or information about controversies surrounding the manual. Forty-six pages integrated critiques surrounding the manual’s construct validity and there were two pages in which the DSM’s inter-rater reliability was critiqued (see Figure 6).



■ = Number of Pages on which the DSM's Construct Validity was Critiqued  
■ = Number of Pages on which the DSM's Inter-rater Reliability was Critiqued

*Figure 6.* Number of pages in which the DSM's construct validity and inter-rater reliability were critiqued within semester project books.

The researcher did not find statistical information about the DSM's rigor within the Semester Project books. Two critics (i.e., Peter Breggin and David Healy) from the researcher's list of critics were cited within *Anatomy of an Epidemic* with regard to challenging conventional perspectives about the DSM (Whitaker, 2010). The researcher did not find any additional instances in which a listed critic was cited for this topic within the other five SP books.

In *Unhinged* (2010), there were 37 pages containing content that met the study's coding framework criteria for the DSM. This content discussed the manual in general terms and addressed specifics surrounding its guidelines and applications. Eighteen of

these pages contained content concerning the manual's development including its origins, structure and content, its revision process including the upcoming DSM-V, and the manual's role in the evolution of psychotropic drug trials and marketing of pharmaceuticals. There were 11 pages on which the manual's merits were discussed. Here, the text noted that the DSM could be helpful with organizing information in a manner that reassured patients "who often improve markedly just by hearing that they have a condition that is well-recognized and treatable" (Carlat, 2010, pp.44, 61).

Carlat (2010) also noted how the DSM had been useful in formulating medication decisions (i.e., pp. 46, 61), establishing more precise descriptions of psychological disorders (i.e., pp. 47, 62), raising money for the American Psychiatric Association through its sales (i.e., p. 55), supporting other assessment tools for determining the existence of psychopathology (i.e., p. 57), and in accurately reflecting the debilitating effects of disorders like social phobia (i.e., p. 58) and pre-menstrual dysphoric disorder (i.e., p. 59). Further, Carlat (2010) noted that "while the diagnostic labels may sound spurious, there are plenty of people out there who are genuinely suffering from the conditions they describe" (p. 59).

*Unhinged* also posited that the pending DSM-V's diagnostic label for mild cognitive impairment might help patients "make better sense of their prognosis" (Carlat, 2010, p. 66). In addition, Carlat asserted that the DSM had been helpful in catalyzing pharmaceutical research and development (i.e., p. 67). Further, the text noted that "The writers of the DSM-IV tried to safeguard against ADHD being too easily diagnosed in adults by adding a requirement that patients had some symptoms before age seven" (p. 153).

There were 22 pages within *Unhinged* that critiqued the DSM. General criticisms of the DSM included Carlat's (2010) opinion that the manual's categorical approach to diagnosis had undermined "psychological curiosity" since the process was more focused on determining what disorder a person had versus trying to ascertain why they were suffering (p. 45). The text also suggested that the DSM had "drained the color out of the way that we understand and treat our patients" (pp. 59–60). Next, Carlat described Robert Spitzer's failed efforts to obtain copies of the minutes from the DSM-V's committee meetings and the APA's justification for their response to Spitzer's request.

Of the 19 pages critiquing the DSM's empirical rigor, all contained content which challenged the manual's construct validity and one of these pages also critiqued the DSM's inter-rater reliability (Carlat, 2010). For example, the DSM's construct validity was disseminated by the statement that "psychiatric diagnosis continues to lag far behind medicine" (Carlat, 2010, p. 14). Further, via the author's assertion that "new diagnoses are based on votes of committees of psychiatrists, rather than neurobiological testing" (p.14) Carlat went on to state that "we commonly think of diseases as collections of symptoms with clear biological origins. Psychiatric diseases are similar but different. They are indeed collections of symptoms but without any clear biological cause" (p. 44).

Next, *Unhinged* noted that the DSM had been criticized for its "collection of arbitrary labels based on shaky science" (Carlat, 2010, p. 47, also see pp. 54–55), "poor diagnostic reliability" (p. 52), pathologization of "outdated cultural mores" (p. 53), the medicalization of "normal human emotion" (p. 56, also see p. 57), and issues with comorbidity (i.e., pp. 60–61). The coverage of diagnostic reliability on page 52 included a reference to Spitzer, Forman, and Nee's (1979) article on the DSM-III field trials in

which the limits of DSM-I and II's inter-rater reliability levels were reviewed (as cited in Carlat, 2010).

In discussing critiques of the DSM-V work groups' summaries, Carlat quoted Alan France's "scathing editorial" within the New York Times which noted that the manual was being developed upon "remarkably weak methodology" (as cited in Carlat 2010, p. 64) and that the supportive evidence for new diagnoses was "slim" (p. 65). *Unhinged* also referred to Frances' concerns about new disorders such as mild cognitive impairment (MCI) leading to overdiagnosis (i.e., p. 66) and the possibility that bipolar disorder was being used as an "umbrella term" for symptoms reflecting sociological problems versus neurobiological pathology (2010, p. 146). Additionally, issues surrounding the conceptualization and diagnosis of ADHD in adults were addressed (i.e., p.150), and concerns were raised about the subjective nature of the interview process from which the diagnoses were formulated (i.e., p. 151).

Conrad's (2007) *Medicalization of Society* contained information on 27 pages that met the coding framework's criteria for DSM content (Conrad, 2007). The text discussed the manual in general terms as well as specifics surrounding its guidelines and applications. On 21 of these pages Conrad integrated information about the manual's development including: the origins and evolution of the DSM, the origins and evolution of social anxiety disorder, generalized anxiety disorder, and adult attention deficit hyperactivity disorder. Additionally, the text described the pharmaceutical industry and medical profession's involvement in the expansion of diagnostic criteria, the manual's historic references to homosexuality as a form of psychopathology, the evolution of gender identity disorder, and the increasing number of diagnoses with each successive

revision of the manual (i.e., pp. 17, 48–49, 51–52, 57–58, 68, 99–102, 104, 112–113, 118–119, 156–157, 166–167).

There were three pages of *Medicalization of Society* (2007) in which the manual's merits were discussed. Here, the text noted that diagnosis with gender identity disorder could help facilitate reimbursement for sexual reassignment surgery (i.e., pp. 103, 112). Next, it was posited that "DSM-III, the third revision, aimed for more rigorous diagnoses" (Conrad, 2007, p. 166). The DSM was critiqued to varying degrees on 12 pages of text. Four of these pages identified general controversy surrounding the medicalization of homosexuality and gender identity disorder, and eight of these pages integrated critiques of the DSM's empirical rigor. These critiques included challenges to the construct validity of social anxiety disorder "because of its loosely defined boundaries" (p. 18), and the author's assertion that the DSM "is not a scientific document" (p. 48). Conrad also noted that the DSM II included the diagnosis of ADHD despite the lack of "solid evidence of biological causation" for the disorder (p. 49).

Next, Conrad (2007) discussed problems with the DSM's construct validity in terms of its historic treatment of homosexuality as a disorder and, more recently, for gender identity disorder (i.e., pp. 99, 100, 102, 103). In addition, the book identified concerns with the increasing numbers of diagnoses that followed each revision of the manual. Within this context, Kirk (2005) was quoted in referencing a list of DSM-V disorders: "If you don't find yourself on that [DSM-V] list, don't fret, more are in the works for the next edition of the DSM" (as cited in Conrad, 2007, p. 118).

Finally, within the *Notes* section of *Medicalization of Society*, Conrad quoted the DSM- III in noting that "There are no laboratory tests that have been established as

diagnostic” for psychological disorders (American Psychiatric Association, 2000, as cited in Conrad, 2007, p. 167). The researcher did not find information about the manual’s inter-rater reliability within Conrad’s text.

In *Selling Sickness* (2005), there were 20 pages containing content that met the study’s coding framework’s criteria for the DSM. This content discussed the manual in general terms in addition to addressing specifics about its guidelines and applications. Twelve of these pages were concerned with the manual’s development, including discussions about changes to the definitions of ADHD and social phobia, the evolution of pre-menstrual dysphoric disorder, the increasing number of psychological disorders, and the DSM committee’s selection process for determining which disorders are included or omitted through the revision process. The text also discussed the development of the DSM within the context of addressing the manual’s conceptualizations of “female sexual dysfunction” (Cassels & Moynihan, 2005, p. 182).

There were five pages on which the manual’s merits were discussed. Here, the text noted that, according to the organization Children and Adults with Attention Deficit Hyperactivity Disorder (CHADD), a diagnosis with Attention Deficit Disorder “can bring special help at school, and nowadays, for adults, special help in the workplace” (as cited in Cassels & Moynihan, 2005, p. 77). Similarly, the text addressed Jean Endicott’s viewpoint that a diagnosis with PMDD could facilitate appropriate treatment (i.e., p. 100) and that the medicalization of premenstrual symptoms could legitimize menstrual related suffering for some women (i.e., p. 108). Cassels and Moynihan (2005) also presented the perspective that including PMDD within the DSM had the potential to catalyze etiology and treatment focused research (p. 109). Finally, *Selling Sickness*, posited that a diagnosis



with PMDD afforded financial coverage of associated prescription medications by insurers.

The text contained challenges to the DSM on ten pages and there were critiques of the manual's construct validity, to varying degrees, on each of these pages (Cassels & Moynihan, 2005). For example, Cassels and Moynihan raised concerns "that ordinary kids might be ending up with a medical label" (p. 78). Further, the book discussed Paula Caplan's critiques of PMDD and her perspective that "the condition has essentially been invented" (as cited in Cassels & Moynihan, 2005, p. 99). Moreover, that "using a medical label to explain away the severe distress some women experience in the lead-up to their period runs the risk of masking the underlying causes of their suffering" (Caplan, 2002, as cited in Cassels & Moynihan, 2005, p. 100).

In challenging the validity of the DSM, Cassels and Moynihan quoted Joan Crisler and Paula Caplan (2002) as having stated that "The concept of PMS is so vague and so elastic that almost every woman can see something of her own experience within it" (as cited in Cassels & Moynihan, 2005, pp. 107–108). The text also described concerns that Spitzer and other members of the DSM revision committee had expressed via their statements that "so little was known about [PMDD's] causes, or how to treat it" and, "the danger that psychiatrists were going to label aspects of ordinary life as a mental disorder" (as cited in Cassels & Moynihan, 2005, p. 109).

Additionally, the text noted that PMDD was placed within the manual's appendix because of disagreements surrounding its validity (Cassels & Moynihan, 2005, p. 110). For example, psychiatrist Sally Severino, who sat on the DSM committee tasked with defining the disorder, was quoted as having stated that "the data did not prove PMDD

existed as a valid diagnosis” (as cited in Cassels & Moynihan, 2005, p. 111). In the same vein, Caplan’s concerns were noted with regard to the potential for a PMDD diagnosis to overlook sociological factors. For example, she was quoted as having asserted that “a history of violent relationships, stressful life circumstances, poverty, or harassment— [are] problems that cannot clearly be fixed by a pill” (as cited in Cassels & Moynihan, 2005, p. 115).

In addressing international perspectives about PMDD, the text quoted a European Agency for the Evaluation of Medicinal Products panel as having stated that “PMDD is not a well established disease entity across Europe. It is not listed in the International Classification of Diseases and remains only a research diagnosis in DSM-IV” (as cited in Cassels & Moynihan, 2005, pp. 115–116). In addition, Cassels and Moynihan (2005) noted that the aforementioned panel had raised concerns about the possibility of false positive diagnoses and the inappropriate prescription of fluoxetine to treat pre-menstrual symptoms that fell below the diagnostic boundary for PMDD (i.e., p. 116). Finally, the text indicated that PMDD research efforts were confounded by disagreements surrounding how best to define and measure female sexual dysfunction (FSD) (i.e., p. 181).

Healy’s (2003), *Let Them Eat Prozac*, contained three pages with content that met the study’s coding framework’s criteria for the DSM. This included general information about the manual as well as specifics surrounding its guidelines and applications. Two of these pages addressed the manual’s development within the context of discussing the DSM-III, its operational definitions, and the manual’s authors’ decision not to recognize

“therapeutic drug dependence” (Healy, 2003, p. 42; see also p. 77). The researcher did not find content on the manual’s merits within this book.

The DSM’s construct validity was challenged to varying degrees on two pages of this text and both of these pages contained critiques of the manual’s construct validity (Healy, 2003). In the first case, Healy noted that the antipsychiatry movement had questioned “the legitimacy of psychiatric diagnoses and practices” (p. 42). In the second case—as mentioned within the preceding paragraph—Healy noted that neither the DSM—III nor the DSM-IV recognized the possibility of therapeutic drug dependence (p. 77).

One page within *The Psychotropic Drug Prescriber’s Guide* (2007) contained information that was consistent with the study’s coding framework for the DSM. This content briefly touched upon the manual’s general criteria for diagnosis (i.e., that symptoms cause distress or interfere with normal functioning), and noted that this dimension “is not considered at all in most studies” (Dubovsky & Dubovsky, 2007, p. 99). The researcher did not find content that discussed the DSM’s development or merits within this book and there were no statistics or critiques pertaining to the manual’s rigor.

In *Anatomy of an Epidemic* (2010) there were 19 pages with content that met the study’s DSM coding framework. This content discussed the manual in general terms and addressed specifics surrounding the DSM’s guidelines and applications. Fifteen of these pages dealt with the manual’s development and evolution (i.e., p. 10, 128, 177–178, 218–221, 269–272, 295, 316–317), as well as its purported impacts on societal beliefs (i.e.,

p. 10). The DSM's development was also addressed within the context of discussing its integration within the medical model (i.e., pp. 269–272, 316).

There were three pages of Whitaker's (2010) book on which the DSM's merits were identified including the manual's role in facilitating services associated with the Disabilities Act (i.e., p. 220). The book also quoted Spitzer's assertion that the inter-rater reliability of DSM-III's disorders was "so much better than we had expected (Kirk, 1992)" (as cited in Whitaker, 2010, p. 270). Next, Whitaker noted that the DSM- III had been effective in uniting psychiatrists' efforts to amalgamate the discipline within the medical model (i.e., p. 271).

The researcher found six criticisms of the DSM within *Anatomy of an Epidemic* (2010). Five of these critiques focused on the manual's construct validity and one critique challenged the DSM's reliability. In addressing the DSM's construct validity Whitaker (2010) described ADHD as a disorder with an unknown etiology whose diagnosis is primarily based on "teacher complaints" (p. 220). Further, Whitaker asserted that:

There was a long record of speculation within medicine that extremely hyperactive children suffered from brain dysfunction of some kind, which was certainly a reasonable thought, but the nature of that dysfunction was never discerned, and then, in 1980, psychiatry simply created with a stroke of its pen in DSM-III, a dramatically expanded definition of 'hyperactivity'. The fidgety seven-year old boy who might have been dubbed a 'goof-off' in 1970 was now suffering from a psychiatric disorder. (p. 221)

Also within the context of discussing the DSM-III, Whitaker noted that:

it was difficult to understand why this manual should be regarded as a great *scientific* achievement. No scientific discoveries had led to this reconfiguring of psychiatric diagnoses. The biology of mental disorders remained unknown, and the authors of the DSM-III even confessed that this was so. Most of the diagnoses, they said, 'have not yet been fully validated by data about such important correlates as clinical course, outcome, family history, and treatment response (Kutchins, 1997)'. (p. 270)

Next, in discussing the marketing of psychotropic drugs Whitaker (2010) suggested that the DSM-III was part of a “rebranding effort” designed to inform people that psychological disorders were valid diseases (p. 316). Further, in referencing the DSM-IV, the text stated that “New and expanded diagnoses invite more people into the psychiatric drugstore” including individuals diagnosed with social anxiety disorder which “in the past might have been characterized as character trait” (p. 317). Finally, in discussing the manual’s inter-rater reliability levels, the text noted that “Spitzer and others argued that such diagnostic categories [within the DSM-II] were notoriously ‘unreliable’ (Wilson, 1993)” (Whitaker, 2010, p. 269).

It is noteworthy that a considerable amount of content about the DSM fell outside of the study’s coding framework which required that the manual be explicitly mentioned on the page it was discussed. As with the NSP books, this relevant information was documented within the “comments” section. The sheer volume of these findings proved troublesome to consolidate and problematic for inter-rater reliability. While some of this content pertained to the use of DSM nomenclature (i.e., identifying various diagnoses and symptomatology), there were also a considerable number of critiques. For example, Carlat (2010) noted that:

Our diagnostic system is shallow and is based on an elaborate checklist of symptoms, leading us sometimes to overdiagnose patients with disorders of questionable validity, or conversely, to miss the underlying problems in our rush to come up with a discrete diagnostic label that will be reimbursed by the insurance company. (p. 15)

Carlat (2010) also asserted that the causes of psychiatric symptoms are “unknown” (p. 21) and “Our diagnoses are subjective and expandable, and we have few rational reasons for choosing one treatment over another” (p. 140). Similarly, in *Anatomy of an*

*Epidemic*, Whitaker (2010) questioned the controversial idea that antidepressants or stimulants could “unmask” pre-existing mania. Further, Whitaker stated that “by greatly expanding diagnostic boundaries, psychiatry is inviting an ever-greater number of children and adults into the mental illness camp” (Whitaker, 2010, p. 209).

Next, Conrad (2007) quoted Horwitz in asserting that, over time, medical diagnoses are “taken for granted as an objective natural entity” (p. 67), and noted that there “have long been pockets of resistance to medicalized disorders like ADHD” (p. 158). Additionally, Conrad (2007) posited that “the diagnosis of intermittent explosive disorder, represents a medicalization of having a ‘bad temper’—surely a problem, but is it a medical disorder?” (p. 168). In the same vein Cassels and Moynihan (2005) challenged the validity of FSD, PMDD, and ADHD. For example, the authors questioned the wisdom of “diagnosing and medicating children whose symptoms include *often fidget with hands or feet* and prescribing lifelong speed to adults who *drum their fingers*” (p. 81). For his part Healy (2003) queried “Have physicians protested the thousandfold increase in the diagnosis of depression in the psychotropic era?” (p. 377).

**C/S/X.** Two of the Semester Project textbooks disseminated content about the c/s/x movement. This included Robert Whitaker’s (2010), *Anatomy of an Epidemic*, Daniel Carlat’s (2010), *Let Them Eat Prozac* (see Figure 7). While Conrad listed the MindFreedomInternational website within his book’s reference section, the researcher could not find any citations for the organization within the text itself. In response, Conrad noted that “I don’t recognize what c/s/x movement means. I don’t recall where [www.MindFreedomInternational.org](http://www.MindFreedomInternational.org) is in the book; perhaps it was mistakenly put in the reference (it happens)” (personal communication, March 30, 2014). Similarly, while

Healy addressed a variety of critiques being levied against psychiatry by various “fringe” group but there were no explicit mentions of the c/s/x movement. Further, and in response to the researcher’s query about this observation, Healy stated that:

I have co-authored a book on the history of ECT where this is picked up in greater detail. Groups like MindFreedomInternational etc.—their origins and developments are covered. I think survivor groups certainly in 1990’s, which is the period *Let Them Eat Prozac* covers, had very little part to play in the story; unlike the ECT story for instance. I think survivor groups still have comparatively little effect within psychopharmacology. It astonishes me that they don’t see that more harm is being done by the meds than ECT etc. (D. Healy, personal communication, June 13, 2014)

Based on Conrad and Healy’s responses, the researcher did not identify any c/s/x content within either text.

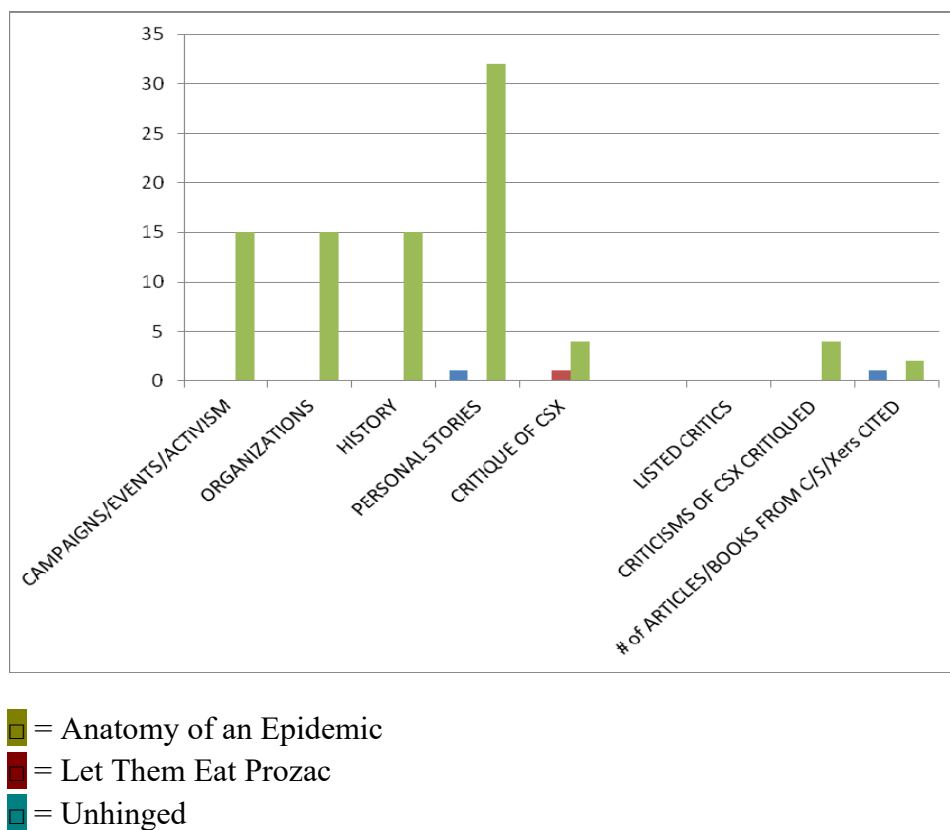


Figure 7. Number of pages with c/s/x content by topic within the three semester project books that addressed the c/s/x movement.

*Anatomy of an Epidemic* (2010) discussed c/s/x related campaigns, events, and activism to varying degrees on 15 pages of text. In addition, the book addressed c/s/x organizations and c/s/x history on 15 pages, and integrated personal stories about c/s/x activists on 32 pages of text. Whitaker (2010) identified critiques of the c/s/x movement and challenged these critiques on four pages of *Anatomy of an Epidemic*.

There were seven instances in which members of the c/s/x movement were cited. For example, in discussing the MindFreedomInternational hunger strike in 2003, Whitaker (2010) noted the letter that six c/s/x members (i.e., David Oaks, Vince Boehm,



and other allies of MindFreedomInternational) wrote to the American Psychiatric Association. Whitaker also referenced his interview with David Oaks. In addition to being a member of the MindFreedomInternational hunger strike, Oaks was one of the MFI's founders and the organization's Director until 2012. Next, Whitaker discussed his interview with John Gottstein, a c/s/x activist, human rights lawyer and president of PsychRights. In addition, John Modrow's (2003) book, *How to Become a Schizophrenic: The Case against Biological Psychiatry*, was quoted and cited.

In considering critiques levied against the c/s/x movement, the text quoted an American Psychiatric Association press release which was submitted just after the MindFreedomInternational activists broke their hunger strike (Whitaker, 2010). The press release noted that organizations like the American Psychiatric Association, and NAMI "will not be distracted by those who would deny that serious mental disorders are real medical conditions that can be diagnosed accurately and treated effectively" (Whitaker, 2011, p. 332).

Content that met the coding requirements for having challenged a critique of the c/s/x movement included Whitaker's (2011) comments about the legitimacy of MindFreedomInternational's hunger strike. For example, the author stated that "it was clear to all observers who had won this battle. The striker's had called the APA's bluff, and the APA had come up empty. It hadn't come up with a single citation that supported the 'brain disease' story told to the public" (p. 332). Further, Whitaker noted that the American Psychiatric Association's letter had directed the MindFreedomInternational activists to an introductory textbook on psychiatry. In sharing his reaction to the American Psychiatric Association's decision not to provide specific citations, Whitaker

stated that “Only the uneducated, it seemed, asked such dumb questions” (p. 332).

Carlat’s (2010) *Unhinged* contained a brief critique of the c/s/x movement on one page. Here, Juli Lawrence’s (a c/s/x member) personal experiences with electro convulsive therapy were discussed. This included a cited position statement by Lawrence and a reference to her website.

### **Non-Textbook Readings**

**Etiology of depression.** Of the 57 required non-textbook readings, five contained material that met the coding requirements for neurobiological hypotheses of depression’s etiology (see Appendix Q). Three of these articles addressed a genetic hypothesis for the disorder and two focused on a neurotransmitter hypothesis. For example, Austin’s (2013), *Genetic Testing for Psychiatric Disorders: Its Current Role in Clinical Psychiatric Practice*, discussed a genetic etiology for depression on one page. Here, the author noted that “Research data from family, twin, and adoption studies show that psychiatric disorders (such as depression, schizophrenia, anxiety, and bipolar disorder) are complex (or multi-factorial) disorders that typically arise as a result of the combined effects of genetic and environmental factors” (p. 1).

*Genetic Testing for Psychiatric Disorders* was the sole non-textbook reading to include a critique for a neurobiologically based hypothesis of depression, albeit within the context of discussing psychiatric disorders in general (Austin, 2013). Here, Austin (2013) noted that:

linkage studies were responsible for locating the genes responsible for both cystic fibrosis and Huntington disease. However, when applied to the study of psychiatric disorders, linkage studies offered only equivocal results. Attempts to replicate initial findings produced only partial or conditional success. The disappointing data drove psychiatric geneticists to consider whether the genes

that contribute to the development of psychiatric disorders were typically of an effect size that was not large enough to be detected by linkage studies. (p. 1)

The second paper to focus on genetics pertaining to a neurobiological hypothesis for depression was the *Department of Defense Veteran Affairs PTSD Guidelines* (2010) which posited that:

Family history of any psychiatric disorder or possible genetic differences in regulating pre-synaptic uptake of serotonin (or other neurobiological mechanism) can increase risk. Genetic research has shown that of the two variants of the gene regulating pre-synaptic uptake of serotonin, the long form appears to be associated with resilience and the short form with the vulnerability to stress events. Individuals who inherited the short form and were exposed to four or more stressful life events were much more likely to develop PTSD and depression and to attempt suicide (Koenen et al., 2009, pp.73–74)

A third paper, the *American Academy of Child and Adolescent Psychiatry Practice Parameter for the Assessment and Treatment of Children and Adolescents With Depressive Disorders*, discussed a genetic etiology for depression on two pages within the context of addressing risk factors for major depressive disorder (Birmaher et al., 2007, p. 1506) and in positing the importance of psycho-education as a treatment intervention (i.e., “Depression is presented as an illness, not a weakness, which is no one’s fault but has genetic and environmental contributions” (Birmaher et al., 2007, p. 1510)). The researcher did not find critiques for either of these instances in which the neurotransmitter hypothesis for depression was discussed.

Next, a paper on serotonin syndrome by Sorenson (2002) noted that:

Serotonin is one of three monoamine neurotransmitters most often associated with depression. Although serotonin is generally associated with clinical depression, there are over ten known serotonin receptor subtypes found throughout the body in many organ systems other than the CNS. (p. 1)

No critique of a neurotransmitter hypothesis for depression was provided within this paper and no other neurobiological hypotheses for depression were addressed.

**DSM.** Eleven of the 57 required non-textbook readings included DSM content that met the coding category requirements for this topic (see Appendix X). This included information about general aspects of the manual, its guidelines, and/or applications. Five of the 11 papers addressed aspects of the DSM's history and development and four papers discussed the manual's benefits/strengths. The researcher found seven papers that critiqued the manual; four of these articles focused attention on the DSM's empirical rigor. Two of the four papers critiquing the manuals' rigor challenged its reliability and all four identified problems with the DSM's construct validity.

The researcher found two pages with content on the DSM inside the *Annual Research Review: Impact of Advances in Genetics in Understanding Developmental Psychopathology* article (Addington & Rapoport, 2012). This included a statement about the development of the DSM and the limits of its construct validity within the context of genetic testing. For example, the authors noted:

Nosology is crucial for clinical practice. While there are no genetic guidelines planned for DSM-V, genetic findings have implications for diagnosis and treatment, or even prevention that are of potential clinical interest. Given the challenges in psychiatry to identify measurable laboratory tests to diagnose disorders as in other branches of medicine, one anticipated outcome from the multitude of genetics studies has been that we would elucidate more genetically homogeneous subtypes of our current diagnostic categories. As we will demonstrate throughout this review, with a few rare exceptions, this is not yet the case. (Addington & Rapoport, 2012, p. 510)

The manual was also mentioned at the end of Addington and Rapoport's (2012) article within a section titled "Key Points." In this instance, the authors asserted that "The last two decades of genetic research neither denigrates nor

informs DSM-V Psychiatric Classification” (Addington & Rapoport, 2012, p. 2015).

Further, and with regard to the strengths and benefits of the DSM, the article stated that “Clinical descriptive diagnosis remains essential for clinical prediction and treatment” (p. 2015).

The researcher’s analysis of *Mental Illness Surveillance Among Adults in the United States. Atlanta: Centers for Disease Control and Prevention* found three pages with content on the DSM that met the study’s coding requirements (Reeves et al., 2011). Two of these pages discussed the DSM’s development and efforts to co-ordinate diagnoses with the ICD (i.e., pp. 3, 4). Next, the manual’s criteria for major depressive disorder and its applications within the study’s survey were addressed. This discussion also noted the DSM’s role in a variety of other assessment tools and in establishing the prevalence of psychiatric symptomatology across populations to identify trends (i.e., pp. 2, 3, 4).

In discussing the limits of the DSM, Reeves and colleagues (2011) noted that the manual’s diagnostic categories had changed over time and stated that “the relationship among the disorders described by these different terms is often unclear” (p. 3). Potential confounds to reliability were identified with the assertion that “In practice, regardless of the diagnostic system used, diagnoses vary according to the training of the coder, local practice, availability of treatment resources, and reimbursement codes” (p. 4).

The researcher coded DSM content within *Bupropion - SR, Sertraline, Venlafaxine - XR after failure of SSRIs for Depression* (Rush et al., 2006, p. 1232) and *The Efficacy of Psychotherapy and Pharmacotherapy in Treating Depressive and Anxiety Disorders* (Cuijpers et al., 2013, p. 138). For example, both articles identified

the manual within their respective methods section and briefly described the DSM's role in selecting participants.

The *Texas Medication Algorithm Project* explicitly identified the DSM on two pages (Suehs et al., 2008). Here, the DSM was referenced in a discussion about the Quick Inventory of Depressive Symptomatology (QIDS) measure, which is based on the manual's diagnostic criteria for major depressive disorder (i.e., p. 21). The paper also referenced the manual within a Clinical Record Form located in the appendix (i.e., p. 66).

The researcher found two pages within *Effectiveness of Antipsychotic Drugs in Patients with Chronic Schizophrenia* on which the DSM was identified (Lieberman et al., 2005). Here, the manual's applications for diagnosing and selecting study participants were highlighted (i.e., pp. 1210, 1213).

In the *VA/DoD Clinical Practice Guideline for Management of Post-Traumatic Stress*, the researcher found twenty-two pages with content that met the coding requirements for this topic (The Management of Post-Traumatic Stress Working Group, 2010). This included general references to the manual within the paper's list of Tables (i.e., p. 2) and acronym list (i.e., p. 206), and in defining the DSM-IV criteria for post-traumatic stress disorder (i.e., pp. 4, 21, 80, 81, 96), and acute stress disorder (i.e., pp. 40, 41, 206). Next, comorbidities between PTSD and other DSM disorders were noted (i.e., p. 24), and the manual was mentioned in discussing triage assessments (i.e., p. 79). The DSM was also cited in discussions surrounding its research applications (e.g., epidemiological surveys) (i.e., pp. 5, 81), screening scales/checklists (i.e., pp. 18, 20, 96, 210, 211, 212), algorithms (i.e., p. 30) and for studying the efficacy of treatment

interventions (i.e., p. 137). In addition, the manual's use within primary care settings was noted (i.e., p.79).

Content within three pages of the VA/DOD guidelines challenged the DSM's rigor (Department of Veterans Affairs [VA/DoD], The Management of Post-Traumatic Stress Working Group, 2010). For example, the text questioned the construct validity of the DSM's approach to trauma and noted that "military personnel do not always respond in the same way as civilian victims of traumatic events, and the criteria for 'fear, helpless, or horror' are being reconsidered in the proposed future DSM criteria" (VA/DoD, The Management of Post-Traumatic Stress Working Group, 2010, p. 21). Next, the paper posited that "Although acute stress reaction (ASR) is not defined in the DSM-IV, there has long been recognition among mental health professionals that individuals who experience a traumatic event react in certain predictable ways" (VA/DoD, The Management of Post-Traumatic Stress Working Group, 2010, p. 28, also see p. 4).

Limits of the manual were also identified via statement that:

These diagnostic criteria and the DSM-IV classification of mental disorders reflect a consensus of current formulations of evolving knowledge in our field. They do not encompass, however, all the conditions for which people may be treated or that may be appropriate topics for research efforts. (VA/DoD, The Management of Post-Traumatic Stress Working Group, 2010, p. 81)

The development of the DSM and its merits were discussed within the context of addressing the revision process (VA/DoD, The Management of Post-Traumatic Stress Working Group, 2010). For example, the paper noted that, in an effort to prevent the pathologization of transient reactions, the DSM-IV had introduced acute stress disorder (ASD). Further, the guideline indicated that this particular diagnosis was used "to describe those acute reactions associated with an increased likelihood of developing

chronic PTSD” (VA/DoD, The Management of Post-Traumatic Stress Working Group, 2010, p. 40).

Next, the guidelines asserted that “The specified diagnostic criteria for each mental disorder are offered as guidelines for making diagnoses, because it has been demonstrated that the use of such criteria enhances agreement among clinicians and investigators” (VA/DoD, The Management of Post-Traumatic Stress Working Group, 2010, p. 80). Further, that “The purpose of the DSM-IV is to provide clear descriptions of diagnostic categories in order to enable clinicians and investigators to diagnose, communicate about, study, and treat people with various mental disorders” (p. 80).

The analysis of the *Practice Parameters for the Assessment and Treatment of Children & Adolescents (AACAP) with Bipolar Disorder*, found 11 pages with content on the DSM that met the study’s coding requirements for this topic (McClellan et al., 2007). This included details about the manual’s criteria for psychiatric disorders (i.e., pp. 108, 109, 110, 111, 112, 113, 114, 115, 116, 117, 118), information surrounding the manual’s development (i.e., pp. 108, 109), research applications (i.e., pp. 108, 109, 111, 113), and utilization as a reference tool for epidemiological findings (i.e., pp. 109, 112, 118).

There were also nine pages on which a variety of questions and concerns about the DSM’s rigor were raised (McClellan et al., 2007). For example, the practice parameters noted limitations with the DSM’s criteria for bipolar disorder. Here, the authors asserted that “there has been a shift in how the disorder is defined in juveniles. There are also similar debates about how broadly to define the disorder in adults” (McClellan et al., 2007, p. 107). Further, the paper stated that “Inasmuch, more



individuals, children and adults, are characterized as having sub-threshold or atypical cases based on periods of elated, expansive, or irritable mood” (pp. 107–108).

Next, the guidelines noted that “Although current DSM-IV-TR nosology does not distinguish age-specific criteria for bipolar disorder, the patterns of illness and symptom definition described in children often vary from the classic description of the disorder in adults” (McClellan et al., 2007, pp. 107–108). Similarly, the practice parameters asserted that “whether these presentations are bipolar disorder and/or represent the same condition classically described in adults has become an area of controversy and scientific debate” (p. 108).

Controversy surrounding the diagnosis of bipolar in children was reiterated on the following page as well (McClellan et al., 2007). Here, the diagnostic criteria being disseminated within current juvenile bipolar literature (i.e., ultrarapid cycling and ultradian cycling) was contrasted with the DSM-IV-TRs criterion for bipolar disorder (p. 109). In the same vein, the paper noted that, for children and adolescents “Changes in mood, energy levels, and behavior are often markedly labile and erratic rather than persistent. Irritability, belligerence, and mixed manic-depressive features are more common than euphoria” (p. 111). A section of the paper titled *Diagnostic Controversy* further encapsulated the question of how best to categorize age specific presentations of bipolar disorder:

The debate and controversy over juvenile bipolar disorder are not whether there are a significant number of youths who are explosive, dysregulated, and emotionally labile or whether these youths suffer significant impairment or are at risk for a variety of adverse outcomes, including substance abuse. These difficulties and concerns are commonplace, especially in community mental health settings and systems of care that deal with at-risk youths (e.g., juvenile justice and foster care). The debate is whether these problems in youths are best

characterized as bipolar disorder and, more important, whether juvenile mania is the same illness as that classically described in adults. (McClellan et al., 2007, pp. 111–112)

Next, the paper described how overlapping symptomatology could complicate the diagnostic picture:

Mood dysregulation in children and adolescents is often associated with features of borderline personality disorder. This raises questions of diagnostic specificity and the overlap between mood and personality disorders, while also generating concerns regarding the validity of personality disorder diagnoses in youths. A related debate occurs in the adult literature, in which bipolarity overlaps with a broad array of mood and anxiety problems, including difficulties attributed to personality disorders or substance abuse. (McClellan et al., 2007, p. 112)

Questions surrounding diagnostic accuracy based on contradictory epidemiological findings in adults and heritability studies were also raised (McClellan et al., 2007, p. 112).

Similarly, in discussing the DSM's construct validity and reliability the practice parameter asserted that

The lack of a gold standard, independent of diagnostic criteria, for confirming a diagnosis remains the major challenge. This is a problem for all psychiatric research because ultimately the application of diagnostic criteria is dependent on the clinician's or investigator's views as to what constitutes a symptom. (McClellan et al., 2007, p. 113)

Further, the paper posited that “The validity of diagnosing bipolar disorder in preschool children has not been established” (McClellan et al., 2007, p. 113) and “There are no biological tests, including imaging or genetic studies that are helpful in making the diagnosis of a bipolar disorder” (p. 114)—a sentiment reiterated on page 116. This practice parameter also challenged the DSM's characterization of an antidepressant precipitated manic episode as being substance induced versus the unmasking of preexisting bipolar disorder (McClellan et al., 2007, p. 117).

The analysis of *Practice Parameters for the Assessment and Treatment of Children & Adolescents with ADHD*, found DSM content meeting criteria for the topic's coding categories on seven of the paper's 28 pages (Pliszka, 2007). This practice parameter addressed aspects of the DSM-IV-TR's diagnostic criteria for ADHD on five pages (Pliszka, 2007) (i.e., pp. 895, 898, 899, 901, 902). Next, ADHD and its comorbidity with other DSM disorders was discussed (i.e., pp. 896, 899, 901, 902), as were the manual's applications in ADHD rating scales (i.e., p. 899).

In terms of critiques, the paper questioned the construct validity of using DSM-IV-TR criteria for diagnosing ADHD in adults (Pliszka, 2007). For example, the practice parameter asserted that "an adult may suffer significant impairment even though he or she suffers from fewer than six of nine symptoms in these areas" (Pliszka, 2007, p. 895). Next, the guideline stated that "the prevalence of mood disorders in patients with ADHD is more controversial" (p. 896) and the authors posited that neuroimaging was not useful in diagnosing the disorder "unless there is strong evidence for such factors in the medical history" (pp. 897–898). A third critique of the manual's construct validity pertained to the number of environments in which problematic symptoms are required for diagnostic criteria to be met. For instance the paper asserted that "DSM-IV requires impairment in at least two settings (home, school, or job) to meet criteria for the disorder, but clinical consensus agrees that severe impairment in one setting warrants treatment" (Pliszka, 2007, p. 898).

The researcher's analysis of the *Practice Parameters for the Assessment and Treatment of Children & Adolescents with Anxiety Disorders*, found DSM content meeting criteria for the study's coding framework in six of the paper's 17 pages

(Connolly et al., 2007). There was one page on which the DSM's rigor was challenged. Here, the practice parameters questioned the manual's capacity to discern clinical levels of anxiety symptomatology. For example, the authors posited that there is "evidence that disability can be associated with subthreshold anxiety symptoms that may not meet full criteria for a DSM-IV diagnosis" (p. 269).

*The American Academy of Child and Adolescent Psychiatry Practice Parameter for the Assessment and Treatment of Children and Adolescents with Depressive Disorders*, was coded for DSM content on five of the document's 24 pages (Birmaher et al., 2007). There was one case in which a potential problem with using the DSM was raised. Here, the paper posited that:

DD [Dysthymic Disorder] consists of a persistent, long-term change in mood that generally is less intense but more chronic than in MDD. As a consequence, DD is often overlooked or misdiagnosed. Although the symptoms of dysthymia are not as severe as in MDD, they cause as much or more psychosocial impairment. (Birmaher et al., 2007, p. 1504)

The paper did not extrapolate on the extent of this problem or clarify whether these concerns pertained to specific limitations with the manual's reliability and/or construct validity (Birmaher et al., 2007, p. 1505). Next, the practice parameter asserted that diagnosing children and adolescents "can be challenging because it is difficult to differentiate whether their depression is part of unipolar major depression or the depressive phase of bipolar disorder" (p. 1505). It was also posited that "not all children who become activated or hypomanic while receiving antidepressants have bipolar disorder" (p. 1505). In discussing the use of neuroimaging for diagnostic purposes the paper asserted that "At present, no biological or imaging tests are clinically available for the diagnosis of depression" (Birmaher et al., 2007, p. 1508).

Additionally, the manual was referenced in a section defining depression and in identifying the DSM-IV-TR criteria for mood disorders (i.e., pp. 1504–1505, 1507) (Birmaher et al., 2007). Next, the manual’s applications in screening and in measuring treatment responses were noted (i.e., pp. 1507, 1520).

### **C/S/X**

The researcher did not find content about the c/s/x movement within the Non-Textbook readings.

### **Videos**

***Etiology of depression.*** The researcher examined the *Biological Basis of Depression*, and *Pharmacotherapy for Depressive Disorders* videos from the *Treatment Issues in Psychopharmacology: Affective Disorders* class (Psychologist Postdoctoral Psychopharmacology Program Syllabus, 2014b). Videos for this particular course that were not analyzed for the study included *Pharmacotherapy of Bipolar Disorders* and *Treatment Guidelines and Considerations for Bipolar Disorder*.

The *Biological Basis of Depression* video identified the following learning objectives: “describe the various biological models used to explain the etiology of affective disorders, describe the biochemical basis of depression and bi-polar disorder and describe the proposed neural circuitry associated with depression and mania” (Anonymous, n.d.) (i.e., from 01:00 to 01:31 in the video). Neurobiological hypotheses of depression addressed within this video included the chemical imbalance hypothesis (neurotransmitter hypothesis), as well as the neurotransmitter receptor, neuroanatomical/circuits, genetic, neuroendocrine, and neurotrophin hypotheses.

The majority of this presentation focused on the purported relationships between genes, proteins, neurocircuitry and depression (Anonymous, n.d.) (i.e., from 01:42 to 40:03 in the video). For example, during the introduction, the presenter (a psychiatrist) noted that:

I call this genes, proteins, neurocircuits, symptoms, and syndromes, and that's the progression. Essentially we inherit genes from our parents, these genes will code for different proteins, these proteins can be receptors, enzymes, etc.; they code for a variety of things. If you have abnormal genes you'll have abnormal proteins, and this is what we call mutations. If you get enough of these abnormal proteins together, and if they're in a certain part of the brain, in a certain set of neurocircuitry, those circuits may malfunction and malfunctioning means to me that the circuits are too hot or too cold, which means that it's too hyperactive or too underactive or hypoactive. (i.e., from 01:42 to 02:26 in the video)

Next, the presenter suggested that when a sufficient number of these proteins accumulate, “malfunctioning” of the brain’s neurocircuitry could occur, leading to psychological disorders like depression (Anonymous, n.d.) (i.e., from 03:03 to 03:46 in the video). The presenter subsequently provided a disclaimer (i.e., “Even though I’m giving a fairly neuroscientific, biological type talk, there are clearly gene and environment interactions. There’s a psyche there’s a heart and there’s a soul. But again my job is to teach the more biological underpinnings”) (i.e., from 03:34 to 03:46 in the video). The *Biological Basis of Depression* video also tied specific symptoms of depression (e.g., fatigue) to “incorrect proteins” and “neurocircuitry problems” (i.e., from 05:54 to 05:56 in the video).

During this discussion the presenter asserted that no single gene codes for a particular DSM disorder (Anonymous, n.d.). Rather, the presenter posited that a gene codes for a symptom implicated in a variety of different disorders. Next, in the process of linking this brain region to depressive symptomatology, the presenter referred to a

PowerPoint slide with a drawing of a “hyperactive amygdala” (i.e., at 06:18 in the video).

The presenter asserted that it was currently infeasible to diagnose depression using neuroimaging findings. Similarly, it was posited that genetic testing was still in its infancy in terms of its capacity to determine risk for a psychiatric illness.

The chemical imbalance hypothesis was introduced within this video as one of the “leading theories” for depressive disorder (Anonymous, n.d.) (i.e., from 09:24 to 15:30 in the video). The presenter stated that, “One idea is you’re low on a certain chemical and depression will ensue” (i.e., from 09:30 to 09:36 in the video). Next, links between dopamine, norepinephrine, and attentiveness were discussed and connections between serotonin and emotional experiences such as sadness, guilt or suicidality were posited. In addition, the presenter stated that:

In theory, if someone is sitting across from you talking about their typical major depressive disorder symptoms, if they tell you they’re a bit more anxious, sad, weepy, and a lot more suicidal, I would start thinking in my practice that their brain is probably a bit lower, underactive in serotonin. If that different patient is sitting across from me telling me that they’re tired fatigued, amotivated, melancholic, I start thinking they’re probably a bit more low, so to speak, in norepinephrine and/or dopamine. (i.e., from 12:20 to 12:50 in the video)

The presenter qualified a subsequent discussion on antidepressants’ mechanism of action, by linking neurotransmitters to specific symptoms of depression (Anonymous, n.d.). Here, it was posited that:

Again this is theoretical, there are some human lab studies, there are some pharmaceutical studies, you know you look at drugs that are more norepinephrine or more serotonin based and you try to decide if they’re better. I’m not sure that we’ve proven this although many psychopharmacologists feel this way in practice. (i.e., from 12:59 to 13:21 in the video)

A PowerPoint was used to reiterate links between increased negative affect with serotonin and norepinephrine and reduced positive affect with norepinephrine and

dopamine (Anonymous, n.d.) (i.e., from 13:17 to 14:27 in the video). The presenter also posited that “The idea is that you can try to start guessing which transmitter, what neurocircuitry system is faulty” (i.e., from 14:20 to 14:27 in the video). By linking neuroimages of “hyper” and underactive neurocircuitry to depressive symptoms, the presenter asserted that “We’re trying to delineate which specific part of the brain serves which function” (i.e., from 14:54 to 14:58 in the video).

The presenter returned to the monoamine hypothesis in discussing the monoamine receptor hypothesis of depression (Anonymous, n.d.). Here, he asserted that “It’s also possible that you have depression because of the first theory. You have less serotonin because maybe you make less serotonin” (i.e., from 16:20 to 16:28 in the video). The chemical imbalance hypothesis was also mentioned briefly within a discussion about brain atrophy. In this case the presenter stated:

So having enough BDNF is a good thing. But what if that gene is turned off and now you won’t have enough? You might actually turn on more mechanisms to make more serotonin so you have your chemistry; your chemical imbalance doesn’t happen because you make enough. What if that gene doesn’t work? Now you’re going to be low on the chemical. (i.e., from 22:27 to 22:47 in the video)

Next, in discussing comorbidity of depression with Parkinson’s disease, the presenter stated:

That’s more in the limbic system. That’s in the deep part of the brain and what we would notice is that they have less dopamine. So down here you would notice normal functioning of dopamine and here’s the Parkinson’s patient and notice that they have less red, less active dopamine, they are more likely to become depressed. That may be your chemical imbalance theory at work. (Anonymous, n.d.) (i.e., from 31:05 to 31:30 in the video)

During the latter part of the video within the context of differentiating bipolar disorder from depression, the presenter asserted that “What about the dopamine



hypothesis of mania? Again, opposite of depression—low dopamine is depression, high dopamine might be mania” (Anonymous, n.d.) (i.e., from 35:55 to 36:00 in the video). In addition, the video’s presenter asserted that “We talk about neurotransmitter being too high in mania, maybe too low in depression” (i.e., from 38:38 to 38:43 in the video).

In the process of introducing the monoamine receptor hypothesis the presenter asserted that “So it does become a chicken or the egg phenomenon. But it’s theoretically possible that somebody has a depression based on low monoamine levels, and it’s also possible that the next person has depression based on having too many monoamine receptors” (Anonymous, n.d.) (i.e., from 16:34 to 16:48 in the video). The video ultimately conceptualized the monoamine receptor and neurotransmitter imbalance hypotheses of depression as corollaries for dysfunctional circuitry (i.e., from 15:28 to 19:50 in the video). For example, it was noted that “What happens is [when] you have a bad number of receptors and a bad amount of neurotransmitters, the ratio is off, and what happens is that causes that specific part of the brain or that neurocircuit to fire too much or too little” (i.e., from 17:17 to 17:23 in the video). Further:

So we have studies, we have models, you know if you were to look at some of those things, here are some of those brain scan pictures of those things that are too cold or too hot. And, so again, if you look at those parts of the brain, this is normal sadness. We all get sad, and you see this bright hot area of the brain and that may be indicative of normal sadness. Now people who are depressed in general will have this as well. It’s just stuck that way, more permanently. It’s like the brain can’t turn that section off. (i.e., from 17:30 to 18:04 in the video)

The video also used neuroimaging to discuss fluoxetine’s mechanism of action. Here, it was posited that the drug and, in some cases, a placebo could purportedly “turn that hot area of the brain cold” (Anonymous, n.d.) (i.e., from 18:10 to 18:17 in the video). Next, the presenter stated that “So these circuits are real. I mean these are theories—too

many receptors, too little transmitters. But when you look at the parts of the brain being hyper or hypoactive these are real findings in real patients” (i.e., from 19:15 to 19:29 in the video).

In another reference to the receptor hypothesis of depression, the presenter noted that:

My residents, what they’re taught to answer on exams is that the number one theory of depression right now is that we have too many receptors and we treat them by downregulating them. We give a lot of antidepressant which raises a lot of neurotransmitter and those receptors shrink and downregulate. They disappear and then you restore your balance between the number of receptors and the number of transmitter molecules around. (i.e., from 33:15 to 33:40 in the video)

Later in the *Biological Basis of Depression* video, genetics were linked to research which observed decreased brain volumes among some depressed subjects (Anonymous, n.d.) (i.e., from 19:51 to 21:13 in the video). The presenter also discussed correlations between depression, genetics and proteins like brain derived neurotrophic factor (BDNF) (i.e., from 21:13 to 23:33 in the video). In addition, the video presentation noted that environmental factors play an important role in major depression and that irreparable damage could ostensibly occur in the brain from multiple depressive episodes. However, the neurogenesis hypothesis was not explicitly mentioned within this context.

The video subsequently revisited a genetic etiology for depression and in doing so included the caveat that “These are findings that have been replicated by a few labs. There have also been some negative studies so this is still theoretical” (Anonymous, n.d.) (i.e., from 26:07 to 26:14 in the video). Within this context the presenter discussed the Serotonin Transporter Gene, and noted that inheriting the S- allele could lead to serotonin transporter pump abnormalities and, by proxy, increased depressive symptomatology

(i.e., suicidal ideation and suicide)( i.e., from 26:04 to 27:17 in the video). Abnormal genes for dopamine and glutamate were also linked to an increased risk of depression (i.e., from 27:18 to 27:35 in the video).

Next, the endocrine system's role in depression's etiology was discussed (Anonymous, n.d.) (i.e., from 27:37 to 29:10 in the video). Here, the negative impact of glucocorticoids on BDNF levels was correlated with apoptosis and depressive symptoms. The thyroid's role in depression was also noted, albeit not explicitly as a neurobiological source for the disorder. Additionally, the presenter posited how cerebral asymmetry from strokes supported a neuroanatomical etiology of depression and that the pathophysiology of Parkinson's (i.e., decreased dopamine) could be viewed as evidence for the chemical imbalance hypothesis of major depressive disorder (i.e., from 30:30 to 31:30 in the video).

In concluding the neurobiology of depression portion of this video, the presenter stated that "There are many potential neurochemical causes of major depressive disorder. There are hormonal ones, there are neurodegenerative ideas. And again it may come down to what genes you have, what proteins you have, what circuits are hot and cold" (Anonymous, n.d.) (i.e., from 31:35 to 31:44 in the video).

On introducing bipolar disorder the presenter stated, "Not sure there's as much information or replicated information and the theories may not be as solid for mania as for depression" (Anonymous, n.d.) (i.e., from 34:06 to 34:15 in the video). The presenter also reiterated that there was no single gene for depression (i.e., from 34:34 to 34:54 in the video). Next, the idea of low norepinephrine and depressive symptomatology was noted (i.e., chemical imbalance hypothesis) (i.e., from 34:58 to 35:04 in the video).

Additionally, the presenter stated that “So again there’s theory and we’re starting to develop these neuroimages that really show that brains operate differently when you have a psychiatric illness versus when you don’t” (i.e., from 39:09 to 39:19 in the video).

Towards the end of the video, the presenter repeated that it was unlikely for a single gene to be responsible for depression or mania, and that environmental factors played a role in depression’s etiology. In addition, the presenter noted that degenerative changes and inter-relationships between malfunctioning genes, proteins, and circuits, might precipitate depressive symptomatology (Anonymous, n.d.) (i.e., from 39:44 to 40:02 in the video). Finally, none of the researcher’s listed critics were cited or referenced within the video.

In the *Pharmacotherapy for Depressive Disorder* video, the presenter stated:

Let’s take a look at the neurochemistry of depression. So biochemically what occurs in depression? While there’s still a lot that’s unknown it appears that the neurotransmitters serotonin and norepinephrine play an essential role in depression. To simplify a great deal, the depletion of these transmitters is indirectly correlated with depression just as potentiating these neurotransmitters is beneficial with many cases of depression. Dopamine, on the other hand, appears to be only secondarily associated with depression. And an increase in dopamine also appears to aid in some cases of recovery in depression. (i.e., from 01:23 to 02:20 in the video)

The video subsequently discussed the biochemistry of these neurotransmitters (Anonymous, n.d.) (i.e., from 02:21 to 06:30 in the video). Next, in discussing the neuropharmacology of depressive disorders, the presenter noted that “Based on the serotonin deficiency hypothesis serotonin seems particularly implicated in depression just as 5-HT increase is particularly implicated in the treatment of depression. Nonetheless, the exception to this is 5-HT 2a, which is associated with agitation and psychosis (i.e.,

from 15:58 to 16:20 in the video). This latter caveat was reiterated in a discussion about atypical antidepressants, Mirtazapine, Trazodone, and Bupropion.

Next, in discussing bipolar disorder, the presenter asserted:

Bipolar disorder has forced us to look beyond the immediate neurotransmitters and to look inside of the cell. In fact neuroscience is finding that all conditions from depression to anxiety to psychosis are characterized by gene expressions that modulate initial superficial actions at the synapses level. (i.e., from 24:02 to 24:43 in the video)

Further, in the process of differentiating between the neurochemistry of bipolar disorder and depression, the presenter asserted that:

The inositol hypothesis states that transmission of certain monoamines, norepinephrine and dopamine during an initially adaptive stress response leads to transduction through G-protein receptors into a perseverating increase on inositol. Inositol as an intracellular messenger may play a role in increased glutamate activity. Glutamate is an excitatory neurotransmitter emitted by glutamatergic inter-neurons found throughout the brain. It is also a neurotoxin in high sustained quantities. The inositol theory, but one among many, states that bipolar disorder is the result of the nervous system's inability to shut off the arousal phase of the stress response maintaining high activity in the glutamatergic pathways which creates manic acting out and, finally, neuronal death. (Anonymous, n.d.) (i.e., from 24:25 to 25:50 in the video)

**DSM.** In the *Biological Basis of Affective Disorders* video, within the context of discussing the etiology of depression, the presenter asserted:

If you develop enough of these gene mutations, abnormal proteins, malfunctioning circuits, you now have multiple symptoms, and if those coalesce you start to get these categories, DSM like categories or diagnoses. For example, schizophrenia, [and] major depressive disorder. (from 02:58 to 03:17 in the video)

The presenter also posited that “The categorical diagnosis used by the DSM probably isn’t the way that the brain truly functions” (i.e., from 05:00 to 05:07 in the video). Next, the presentation discussed depressive symptoms and the diagnosis of depression within the context of dysfunctional brain circuitry. For example, the presenter

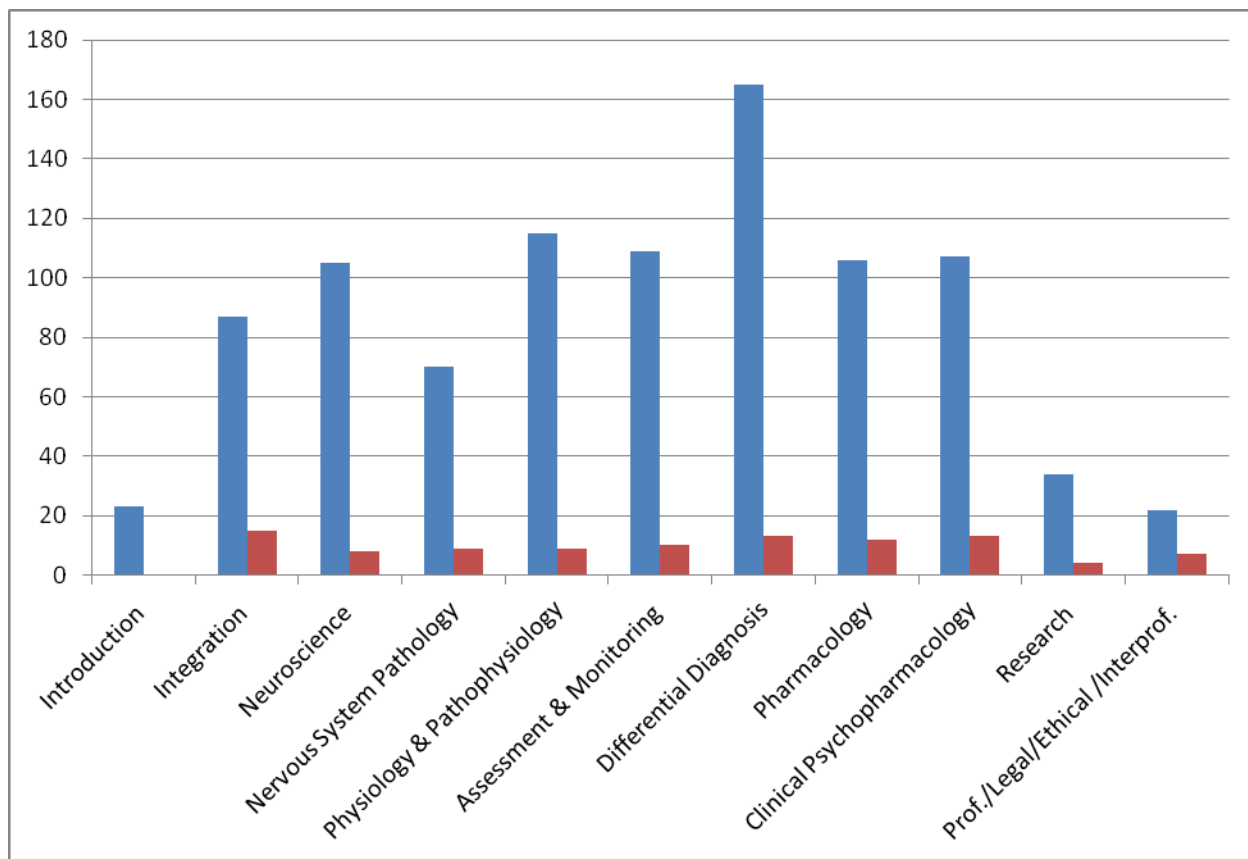
stated that “If you develop at least five out of nine DSM depressive symptoms because you have five out of nine brain areas that are abnormally active than maybe you have a diagnosis” (from 15:05 to 15:15 in the video).

The researcher did not find content which met the study’s coding framework requirements for this topic, within the *Pharmacotherapy of Depression* video (Anonymous, n.d.). However, the presenter for this video did identify depressive symptomatology (e.g., low mood, concentration difficulties, psychomotor retardation or agitation, guilt, suicidal ideation, appetite changes, motivation, and suicidal ideation), in discussing psychopharmacological treatments for the disorder. Next, the presenter referred to a number of DSM disorders (e.g., bipolar disorder, depression, ADHD, and schizophrenia). Further, manic behavior was mentioned on a number of occasions in which psychopharmacological treatments for bipolar disorder were discussed.

**C/S/X.** The researcher did not find information about the c/s/x movement within either of the examined psychologist postdoctoral psychopharmacology program’s analyzed videos.

### **Division 55 PEP Review DVD**

The Division 55 PEP Review DVD was comprised of ten learning Modules in addition to a short introductory PowerPoint which outlined the training materials in broad strokes. All of the learning Modules used PowerPoint presentations to disseminate content and the majority of these slides contained an audio clip. The audio clips were predominantly verbatim readings of the slides’ written content. For a list of the learning modules including the numbers of slides for each module and the percentage of questions on the PEP pertaining to each of the Modules, see Figure 8.



■ = Number of Power Point slides per topic  
■ = Percentage of questions for each topic within PEP Exam

*Figure 8.* Number of division 55 pep review dvd power point slides afforded per topic and percentage of questions within psychopharmacology examination per topic.

**Neurobiological etiology of depression.** Five slides from the Division 55 PEP Review DVD's ten learning Modules addressed neurobiologically based hypotheses of depression's etiology. For example, the audio clip for one slide (#64) within the *Nervous System Pathology Module* noted a genetic hypothesis for the etiology of depression, and the audio clips from two slides (i.e., #16, #69) within this Module implicated neurotransmitters and the Neuroendocrine system in the disorder's etiology.

Next, the *Pharmacology Module* pointed to a neurotransmitter based etiology of depression in slide #68 and noted “Knowledge of theoretical relationships thought to exist between neurotransmitter systems and psychopathological conditions based on known mechanisms of action and clinical observation. Examples: roles of serotonin in depression.” In addition, slide # 43 from the *Integration Module* described what could be interpreted as an anatomical hypothesis for the etiology of depression. For example, the slide stated that “[The] amygdala doesn’t turn off in some depressed patients.”

In the *Nervous System Pathology Module*, various disorders of the nervous system with depressive symptomatology were addressed. Slide #6 from the *Research Module* provided a link to the Carlat Report (2015). Carlat was one of the study’s listed critics and while his report was not analyzed for this study, it does contain challenges of conventional viewpoints about psychopharmacology.

The *Neuroscience Module* (i.e., audio clip for slide #4), recommended that students use a neuroanatomy text in conjunction with the presentation. Similarly, slide #35 associated a number of brain areas with psychopathology and suggested that students familiarize themselves with these regions. Next, the audio clip for slide #61 referred students to chapters six and seven from *Stahl’s Essentials of Psychopharmacology* (2008), in order to increase familiarity with brain regions and tracts implicated within conditions commonly treated via prescription psychotropics.

An analysis of both chapters for content on neurobiological etiologies of depression found a genetic hypothesis for the disorder was mentioned in chapter six with the caveat that “depression is moderately biologically determined in many individuals—not enough to manifest without environmental input but vulnerable to breakdown in the



presence of major stressors” (Stahl, 2008, p. 192). The text also noted that “Major Depressive Disorder may be less robustly biologically determined than schizophrenia” (p. 193). In chapter seven, the researcher did not find content about a neurobiological etiology of depression that met the coding categories for this topic (Stahl, 2008).

The Division 55 PEP Review DVD’s computer file of “supplemental materials” was comprised of two papers on medication dosages and applications, an Excel template for documenting drug references, a list of pregnancy related medication safety classifications, the *PEP Candidate Guide*, the *Mental Health: Culture, Race, and Ethnicity: A Supplement to Mental Health: A Report of the Surgeon General* (Rockville, 2001), and the *National Healthcare Disparities Report* (U.S. Department of Health and Human Services, 2006). An analysis of these materials yielded one case of content pertaining to a neurobiological hypothesis of depression.

For example, the Surgeon General’s report noted the potential role that genetics play in depression’s etiology. The report also challenged the veracity of this claim positing “less heritability for depression than for bi-polar disorder and schizophrenia” (Rockville, 2001, chapter 1, p. 26). The *PEP Candidate Guide and Application Materials* noted connections between depression and the endocrine, hematological, and immunological systems and commented on the existence of depressive symptoms within some medical disorders (American Psychological Association College of Professional Psychology, 2006).

The Division 55 PEP Review DVD also provided links to a variety of different organizations disseminating information about psychopharmacology (Division 55 & Hoover, n.d.). The majority of these links were defunct. Websites in operation included

Epocrates, the National Institute of Health, the sites at Harvard University and Stanford University, and the Psychiatric Times. The Division 55 PEP Review DVD's supplemental resource list also included a link to the aforementioned Carlat Report—a blog written by Daniel Carlat (one of the researcher's listed critics) which addresses a variety of nonconventional perspectives about psychopharmacology.

In addition, students were encouraged to read *Patient Centered Method and Self-Directed Behavior Change*, by Eivind Meland (1995) and, *The Cost effectiveness of Anxiety Nervosa Treatment*, by Crow and Nyman (2003). The researcher did not find content on neurobiological hypotheses of depression within either of these papers.

**DSM.** The researcher did not find DSM content that met criteria for this topic's coding framework within the Division 55 PEP Review DVD's learning modules (Division 55 & Hoover, n.d.). However, the audio clip for slide #61 within the *Neurosciences Module* referred students to chapters six and seven from *Stahl's Essentials of Psychopharmacology* (2008) to increase familiarity with brain regions and tracts implicated within conditions typically treated with prescription psychotropics (Division 55 & Hoover, n.d.). The DSM was explicitly referenced on two pages in chapter six of this text (Stahl, 2008). One of these pages included content on the limits of the DSM including a critique of its construct validity. For example, Stahl noted that the manual was based on “consensus statements” and that “mental illnesses are not diseases” (p. 178). Stahl also noted that the manual was useful as a tool that aided communications about the symptomatology of mental illness (i.e., p. 178). In addition, Stahl referenced the manual in a discussion about “symptom endophenotypes” (p. 184). The researcher

did not code for any DSM content within chapter seven. Similarly, the researcher did not locate any DSM content within the PEP Practice Questions provided by Dr. Hoover.

The researcher found content that fit the coding framework for the DSM within one of the Division 55 PEP Review DVD's (n.d.) Supplemental Articles (i.e., the Surgeon General's supplemental report, *Mental Health: Culture, Race, and Ethnicity* (Rockville, 2001)). In total, there were 27 pages on which the DSM was addressed in general terms therein, including discussions about its applications and guidelines. Eleven of the 27 pages contained information about the manual's development and ten pages integrated critiques, limitations, or criticisms of the DSM. In addition, aspects of the DSM's strengths and merits were identified on six pages of text.

Critique wise, the DSM's content validity was challenged on five pages of the report and there was one page on which the manual's inter-rater reliability was critiqued (Rockville, 2001). The researcher did not find statistics about the DSM's rigor within this report. References to the manual's merits focused on Rockville's assertions that the manual's authors had a concerted effort to consider the impact of culture, race, and ethnicity on symptom presentation and diagnostic considerations.

There was no DSM content meeting the study's coding frameworks for this topic within the articles by Meland (1995) or Crow and Nyman (2003). Finally, the researcher did not find content on the DSM within the PEP Practice Questions provided by Hoover.

**C/S/X.** The researcher did not locate any c/s/x content that met the study's coding requirements for this topic, within the Division 55 PEP Review DVD's (n.d.) ten PowerPoint learning Modules or chapters six and seven from *Stahl's Essentials of Psychopharmacology Neuroscientific Basis and Practical Applications* (2008). There

was one example of c/s/x content within the Division 55 PEP Review DVD (n.d.) Supplemental Materials that met the criteria for the study's c/s/x coding category framework for this topic. This was located within a footnote of the first chapter of the Surgeon General's report which noted that consumers, survivors, and ex-patients were terms that "identify people who use or have used mental health services" (Rockville, 2001, p. 16). There was also some content within the report falling outside of the coding categories that warrants mentioning—in chapter two of Rockville's report it was noted that anti-psychiatry groups had exploited pre-existing African-American mistrust of mental health professionals (p. 29). However, it was unclear whether the authors were considering c/s/x groups within this context.

The report also noted the importance of a "voluntary support network," but no further clarification was provided in this regard (Rockville, 2001, p. 33). In addition, page 166, in chapter seven noted the benefit of including "representatives from the community being served in the design, planning, and implementation of services." Again, there was no explicit mention of the c/s/x movement within this context.

A comment within Eivind Meland's (1995) article bears mentioning as well: "As society changes the paternalistic role must change in accordance with the lay consumer and patient movement" (p. 1.). Again, it was unclear whether the author was referring to the c/s/x movement in this regard. Similarly, an assertion within the *National Disparities Report* (U.S. Department of Health and Human Services, 2006) asserted that patient centered care involved "Providing care that is respectful of and responsive to individual patient preferences, needs, and values and ensuring that patient values guide all clinical decisions" (p. 33). While this statement is congruent with c/s/x values there was no clear

reference to the movement in the article and for this reason none of the aforementioned content was coded as such. The researcher did not find c/s/x content in the article by Crow and Nyman or within the PEP Practice Questions provided by Hoover.

### **Listed Critics**

In terms of the examined psychologist postdoctoral psychopharmacology program none of the Non-Semester Project (NSP) books or non-textbook readings cited the study's listed critics within discussions about the DSM or neurobiological hypotheses of depression. The c/s/x movement was not addressed therein.

Three of the Semester Project (SP) books were authored by a critic (i.e., Carlat, Healy, and Whitaker). Four of the SP books cited one or more of the listed critics in addressing the etiology of depression. In *Selling Sickness*, for instance, Moynihan and Cassels (2005) cited David Healy on three occasions in which a neurotransmitter hypothesis for depression was discussed. Two of these citations were for Healy's (2003), *Let Them Eat Prozac*, and the third was for an interview with Healy within the documentary *Selling Sickness* (Moynihan & Cassels, 2005).

In *Let Them Eat Prozac*, Healy (2003) cited two of his own works (2000, 2001) in the process of critiquing the chemical imbalance hypothesis of depression. Similarly, in *Psychotropic Drug Prescriber's Survival Guide*, Dubovsky and Dubovsky (2007) cited a 1999 article by Healy in their critique of the monoamine hypothesis. In addition, in *Anatomy of an Epidemic*, Whitaker (2010) cited three of the researcher's listed critics in challenging the neurotransmitter premise for depression. This included three citations for a 2005 article by Jeffery Lacasse about pharmaceutical marketing and the portrayal of serotonin's role in depression (Whitaker, 2010). Next, Whitaker (2010) referenced a 2005

paper by Healy on antidepressant advertising. Whitaker (2010) also noted that Healy “has written a number of books on the history of psychiatry” (p. 74). Finally, in referring to the monoamine hypothesis for depression, Whitaker (2010) quoted Joseph Glenmullen from his book 2000 *Prozac Backlash*, and the latter’s assertion that “in every instance where such an imbalance was thought to be found it was later proved to be false” (Whitaker, 2010, p. 78).

*Anatomy of an Epidemic* also included two citations for one listed critic within critiques of the DSM’s rigor that met coding requirements for this topic (Whitaker, 2010). For example, Peter Breggin’s (2001) book, *Talking Back to Ritalin*, was referenced in a discussion about the development of the DSM-III and the purported lack of evidence to support a neurobiological basis for ADHD (as cited in Whitaker, 2010, p. 221). Whitaker (2010) also referenced Breggin’s 1991 book, *Toxic Psychiatry*, in discussing the economic benefits that the DSM-III’s publication and subsequent sales brought the American Psychiatric Association (p. 272).

In addition, a number of the researcher’s listed critics were also cited within discussions that fell outside of the study’s three topics of analysis. Three of the examined NSP textbook chapters, all of the SP books, and five of the non-textbook readings cited one or more of the researcher’s listed critics within this context. For example, in *A Primer of Drug Action*, Daniel Carlat (2010) was cited within a discussion about prescriptive authority for psychologists (as cited in Julien et al., 2013, p. 682). Next, in *Pharmacotherapy for Psychologists: Prescribing and Collaborative Roles*, two works by David Antonuccio were cited in addressing the benefits of psychosocial interventions

(Antonuccio, Danton, DeNelsky, Greenberg, & Gordon, 1999; Brown, Antonuccio, DuPaul, Fristad, King, Leslie, et al., 2008, as cited in McGrath & Moore, 2010, p. 98).

In chapter six of *Pharmacotherapy for Psychologists: Prescribing and Collaborative Roles*, LeVine and Foster, referenced a paper by Carlat on the kindling hypothesis and its implications to psychiatry with regard to establishing rapport with clients (2008, as cited in LeVine and Foster, as cited in McGrath & Moore, 2010, p. 125).

Next, chapter seven of McGrath and Moore's text included a section on research methodology in which the last-observation-carried-forward (LOCF) procedure was examined and a citation for Irvine Kirsch integrated therein (as cited in McGrath & Moore, 2010). In the same chapter, Joanna Moncrieff's research on medication side effects and the purported issue of unblinding within placebo controlled antidepressant drug trials was noted (Moncrieff, Wessely, and Hardy, 1998, as cited in McGrath & Moore, 2010, p. 136).

Finally, in *Essential Evidence Based Psychopharmacology*, a paper co-authored by Kirsh and Deacon was cited in examining the implications that study participants' baseline symptoms could have on the results of antidepressant drug trials (Kirsch, Deacon, Huedo-Medina, Scorbina, Moore, and Johnson, 2008, as cited in Stein et al., 2012) Another page referenced a meta-analysis co-authored by Healy on suicide risk and antidepressant drug trials (Fergusson, Doucette, Glass, Shapiro, Healy, Hebert, et al., 2005, as cited in Stein et al., 2012).

In terms of non-textbook readings, Antonuccio was cited on three occasions within *Practice Guidelines Regarding Psychologists' Involvement in Pharmacological Issues* (i.e., pp. 835, 841, 843) (American Psychological Association, 2011). For

example, one of these papers was cited within a discussion about “professional challenges” conceivably faced by prescribing psychologists (Antonuccio, Danton, & McClanahan, 2003, as cited in American Psychological Association, 2011, p. 835). Concerns about “iatrogenic medication effects” and the comparable level of efficacy between treating depression with psychotherapy or psychopharmacology were also noted (Antonuccio, Burns, & Danton, 2002, as cited in American Psychological Association, 2011, p. 841; Antonuccio et al., 1999, as cited in American Psychological Association, 2011).

The article, *Antidepressant Drug Effects and Depression Severity: A Patient-Level Meta-Analysis*, referenced a paper by Kirsch et al., on the potential for baseline symptom severity to impact drug trial results (2008, as cited in Fournier et al., 2010). Next, another of Kirsch’s papers, this one questioning the efficacy of antidepressants, was cited in Smith’s article, “Research Shows that all too Often, Americans are Taking Prescriptions that may not Work or May be Inappropriate for their Mental Health Problems” (2008, as cited in Smith, 2012).

Smith’s (2012) article also referenced Carlat’s (2010) book, *Unhinged*, in discussing the financial incentives for psychiatrists who prescribe medications instead of making provisions for psychotherapy. Next, Carlat was cited in a discussion about the purportedly unrealistic expectations that some patients have surrounding the effectiveness of psychotropic medications (2010, as cited in Smith, 2012).

The examined study by Cuijpers and colleagues on the effectiveness of medications for treating anxiety and depression referenced a meta-analysis coauthored by Jeffery Spielmans which concluded that certain psychotherapies were as effective as



antidepressants (Spielmanns, Berman, & Usitalo, 2011, as cited in Cuijpers et al., 2013). Next, the *Department of Defense/Veterans Affairs PTSD Guidelines* referenced four articles on hypnosis by Kirsch in discussions about the treatment of trauma symptoms via adjunct interventions (1994, 1995, 1996, 1999, as cited in VA/DoD, The Management of Post-Traumatic Stress Working Group, 2010).

There were also a number of instances within the Semester Project books in which listed critics were cited for content that fell outside of the study's three topics of analysis. For example, in chapter four of *Unhinged*, Carlat quoted Healy in a discussion about concerns surrounding the over-prescription of psychotropic medications (1997, as cited in Carlat, 2010). Healy was also cited along with Joseph Glenmullen, regarding risks of medication side effects (Healy, 2004, and Glenmullen, 2001, as cited in Carlat, 2010). Next, Joanna Moncrieff was referenced within the context of critiquing the dopamine hypothesis for schizophrenia (2009, as cited in Carlat, 2010). Also in chapter four, an article by Lacasse and Leo was referenced within a discussion about Eli Lilly's use of the neurotransmitter deficiency hypothesis to market Cymbalta (2005, as cited in Carlat, 2010).

In chapter five of *Unhinged*, Carlat cited Marcia Angel's book, *The Truth About the Drug Companies*, as being an "invaluable source" of information about the "deceptive marketing strategies of the pharmaceutical industry" (2005, as cited in Carlat, 2010, p. 231). Next, Healy and Cattell's article outlining concerns about ghost writing, was referenced in a section of *Unhinged* that addressed the pharmaceutical industry's purported manipulation of medical publications (2003, as cited in Carlat, 2010). In

addition, a work by Healy outlining the history of ECT was referenced in chapter eight (2007, as cited in Carlat, 2010).

Carlat (2010) cited a number of his own works within chapters five, six, seven, eight, and ten for topics ranging from the author's experiences as a Wyeth speaker, as well as his concerns about ghost writing, bipolar and ADHD diagnoses in children, the validity of ADHD testing, and unethical pharmaceutical marketing (2006a, 2006b, 2007a, 2007b, 2008, as cited in Carlat, 2010).

In *The Medicalization of Society*, Conrad (2007) cited three of the researcher's listed critics within content that fell outside of the study's three topics of analysis. Here, Angel was cited in a discussion about the growing influence of the pharmaceutical industry, while Breggin was referenced in relation to his critiques surrounding the construct validity of ADHD and the use of Ritalin to treat the disorder (Angel, 2003, and Breggin, 1998, as cited in Conrad, 2007). In chapter seven, Healy and Angel were both cited within a section on the history and development of pharmaceutical marketing (Healy, 1997, Relman & Angel, 2002, as cited in Conrad, 2007).

Similarly, in *Selling Sickness*, Moynihan and Cassels referenced Angel's article, *Is Academic Medicine for Sale*, in discussing the relationship between psychiatry and the pharmaceutical industry. In addition, Healy was cited on eight occasions within the context of discussing antidepressants, iatrogenic suicide risk, pharmaceutical marketing, and the medicalization of human experience (Angel, 2000, 2003, and Healy, 2003, 2004a, 2004b, as cited in Moynihan & Cassels, 2006). Next, Antonuccio was referenced twice for papers which discussed the effectiveness of non-pharmacological interventions and in

questioning the efficacy of antidepressants (1995, 1999, as cited in Moynihan & Cassels, 2006).

In chapter four, Moncrieff was cited on three occasions. For example, in discussing the prevalence of ADHD in children, questioning the validity of the ADHD diagnosis, and in raising concerns about the implications of pharmaceutical company's efforts to partner with parents of children diagnosed with ADHD (Moncrieff, 2003, 2007, and Timimi, Moncrieff, & Jureidini, 2004, as cited in Moynihan & Cassels, 2006). Moynihan and Cassels also referenced Healy in a discussion about the ways in which pharmaceutical marketing reportedly shapes societal conceptualizations of mental distress (2003, as cited in Moynihan & Cassels, 2006).

There were also cases in *Let Them Eat Prozac* (2003), where critics were listed for topics that fell outside of the study's analysis. Within this context, Healy (2003) cited 44 articles, two replies to articles, and six books he had authored, coauthored, edited, or co-edited. In addition, Healy referenced documents from three trials he had taken part in (i.e., a testimony, expert witness statement, and three depositions). Healy's critical analysis of Prozac also addressed concerns surrounding the drug's development process (i.e., research, publications, FDA process), marketing (i.e., fraudulent claims), safety (i.e., increased risk of harm to self and others), and efficacy (i.e., compared to other antidepressants and psychotherapy).

In addition, *Let Them Eat Prozac* (2003) referenced Glenmullen's (2001) book *Prozac Backlash*, five times across chapters two, four, nine, ten, and twelve. Here, Glenmullen was cited in discussing the antidepressant poop-out effect, the Joseph Wesbecker case, and issues with Prozac and dependency (2001, as cited in Healy, 2003).

Healy also discussed the criticism Glenmullen faced after publishing *Prozac Backlash*. Next, Breggin was cited on nine occasions across chapters three, four, five, six, and seven, within the context of discussing the 1991 Food and Drug Association's advisory committee on Prozac, the effectiveness of psychotherapy, and Breggin's role as an expert in lawsuits against pharmaceutical companies (1991, 1994, as cited in Healy, 2003).

Breggin's (2001) book, *Toxic Psychiatry*, was also referenced by Healy. For example, in discussing negative reviews of that text including Eli Lilly's purported efforts to portray Breggin in a negative light (as cited in Healy, 2003). In addition, Healy cited Breggin and Whitaker in addressing concerns surrounding the use of pharmaceutical company donations to support patient organizations like CHADD (Breggin, 1991, and Whitaker, 2002, as cited in Healy, 2003). Next, Marcia Angell's book, *Science on Fire*, was referenced in discussing breast implants and associated law suits (1997, as cited in Healy, 2003). Healy (2003) noted that these litigations brought "corporations to their knees using medico-legal experts who were all but charlatans" (p. 335).

In *Drug Prescriber Survival Guide*, Dubovsky and Dubovsky (2007) cited Angel's book, *The Truth about Drug Companies*, on twelve pages of text for content that fell outside of the study's three topics of analysis (2005, as cited in Dubovsky & Dubovsky, 2007). Here Angel was cited within content that focused on the pharmaceutical industry's purportedly negative impacts on the field of medicine. For example, partnerships between drug companies and universities, industry sponsored CME programs, manipulations of physician's prescribing habits, problems with the FDA approval process and the methodology of drug trials, and cases in which pharmaceutical

advertising claims were inconsistent with research findings (Angel, 2005, as cited in Dubovsky & Dubovsky, 2007).

Similarly, four of Healy's works were referenced on eleven pages of *Drug Prescriber Survival Guide* in addressing the pharmaceutical industry's impacts on the profession of psychiatry, the history of antidepressants, concerns about the increased prevalence of depression, the extent of industry sponsored research, and links between the pharmaceutical industry, academia, and physicians' prescribing habits (Healy, 1999, Healy & Cattell, 2003, Healy & Thase, 2003, as cited in Dubovsky & Dubovsky, 2007). Further, Healy was referenced in the text's discussions about the pathologization of human behaviours, biases in the reporting of research findings, the limits of data from industry sponsored trials, and issues surrounding the side effects of SSRIs (1999, Healy & Cattell, 2003, Healy & Thase, 2003, 2006, as cited in Dubovsky & Dubovsky, 2007).

In addition to the listed critics cited within discussions about the DSM and the chemical imbalance hypothesis of depression, *Anatomy of an Epidemic* included references to works by Breggin, Kirsch, Moncrieff, Healy, Carlat, Leo, and Glenmullen in addressing other topics related to psychopharmacology (Whitaker, 2010). For example, seven works by Breggin were cited on 17 pages across chapters six, eleven, thirteen, and fourteen (Breggin, 1991, 1993, 1997, 2001a, 2001b, 2008a, 2008b, as cited in Whitaker, 2010). This content focused on iatrogenic effects of antipsychotics, challenges to the validity of a neurobiological hypothesis for ADHD, concerns about the methodology of ADHD research, and questions surrounding the safety and efficacy of prescribing Ritalin for the disorder.

Whitaker also referenced Breggin in discussing the efficacy and side effects of SSRI's (i.e., suicide risk, apathy syndrome, and cognitive decline), the development of the DSM, and concerns about partnerships between the American Psychiatric Association and the pharmaceutical industry (Whitaker, 2010). Finally, Whitaker (2010) cited a number of Breggin's works in the process of challenging claims about neurobiological theories of mental distress, and in discussing the marketing of xanax and the purportedly negative impacts that challenging pharmaceutical companies and the American Psychiatric Association had on Breggin's career.

Additionally, a meta-analysis by Kirsch and colleagues was cited in chapter eight of *Anatomy of an Epidemic* within the context of questioning the efficacy of antidepressants (Kirsch et al., 2008, as cited in Whitaker, 2010). Next, Joanna Moncrieff's works were cited in chapters eight and nine, in the process of discussing British sickness benefits and trends therefore. Further, her book, *The Myth of a Chemical Cure*, which outlined potential risks associated with long term lithium therapy, was referenced (2000, 2008, 2009, as cited in Whitaker, 2010). Next, the article by Jonathon Leo, *SSRI Trials in Children*, was cited in a discussion about methodological concerns pertaining to SSRI drug studies (i.e., manipulation of placebo responders) (2006, as cited in Whitaker, 2010).

Whitaker also referenced works by Angel and Carlat in discussing pharmaceutical company's relationships with physicians and psychiatrists who are hired to speak at industry sponsored drug information sessions (Angel, 2000, and Carlat, 2007, as cited in Whitaker, 2010, p. 278). In addition, Glenmullen's *Prozac Backlash*, was referenced by Whitaker in discussing how "Eli Lilly mounted a campaign to discredit [Glenmullen]"

(2000, as cited in Whitaker, 2010, p. 307). Healy was cited on a total of 13 pages within chapters nine, eleven, fourteen, and sixteen, which included references to eight of his works (2000, 2001a, 2001b, 2004, 2005, 2008, 2009a, 2009b, as cited in Whitaker, 2010). More specifically, Healy was cited within content about the history of manic depression, suicide risk associated with SSRIs, Eli Lilly's "fraudulent" marketing of Prozac, and the marketing of pharmaceuticals via collaborations between the National Institute of Mental Health and Eli Lilly (Whitaker, 2010, p. 284). Healy was also referenced within Whitaker's content about the history of asylums, psychiatric bed utilization, the dangers of over-prescribing, and the negative impact that speaking about the suicide risks associated with SSRIs had on the former's career (as cited in Whitaker, 2010).

The researcher did not find any of the study's listed critics within the PEP Review DVD materials.

### **Relevant Content Falling Outside of the Coding Framework Requirements**

The researcher found a number of instances in which there was relevant content that fell outside of the study's coding categories for information about a neurobiologically based etiological hypothesis of depression (within the examined materials from the analyzed psychologist postdoctoral psychopharmacology program). For the Non-Semester Project books, this included comments about the apparent role that genetics, neuroanatomy, neurocircuitry, receptors, and neurotransmitters play in the neurobiological etiology of psychiatric disorders in general. There were also some instances within these texts where broad critiques of neurobiological hypotheses for the etiology of mental disorders were noted. For example, in the *Manual of Clinical*

*Psychopharmacology for Nurses*, Leahy and Kohler (2013) asserted that “Although the field of psychopharmacology has experienced a revolution over the past two decades, much remains unknown about the human brain, neuropathology, and neurotransmitters as they relate to an individual’s thoughts, moods, and behaviours” (p. xxviii). Similarly, McGrath and Moore (2010) stated that “The psychobiosocial model does not circumscribe the nature of human suffering or problems with adaptation as a ‘chemical imbalance’” (p. 113). In the same vein, Blumenfeld (2010) stated in his neuroanatomy textbook that “Emotions and drives are almost as difficult to explain as consciousness itself” (2010, p. 976).

The Semester Project books also included a number of critiques within the scope of relevant content falling outside of the coding frameworks for this topic. For example, Carlat, Healy, Moynihan and Cassells, and Whitaker all made broad assertions that the neurobiology of psychiatric disorders was unknown. Further, Conrad’s book on medicalization noted skepticism about the validity of ADHD. There was one Non-Textbook reading in which the researcher found relevant content that fell outside of the coding framework requirements for a neurobiological hypothesis of depression’s etiology. In this instance, the authors of *Psychotherapy and Psychopharmacology: Different Universes or an Integrated Future?* noted that some patients requested medications for the explicit purpose of treating chemical imbalances (Winston, Been, & Serby, 2005). However, the theory was not challenged and there was no further discussion about how best to address the limitations of this hypothesis with patients.

In terms of the videos, relevant content falling outside of the coding framework for depression’s etiology was limited to an assertion, within *Psychopharmacology of*



*Affective Disorders*, about neurochemistry's role in every cognitive-emotional experience (i.e., from 38:59 to 38:57 in the video) and the comment that:

The chemistry involved in complex biopsychosocial operations is well beyond our present day ability to calculate let alone measure and control. Key to appreciating this complexity nevertheless is the acknowledgement of interplay and multiple feedback systems at the molecular as well as global levels. For the time being it takes deductive reasoning and an act of faith to understand the complex role of neurochemistry and even the simplest of mental operations. (n.d., from 39:11 to 39:44 in the video)

The PEP Training DVD's supplemental materials also included relevant content about a neurobiological etiology of depression that fell outside of the coding categories. In this regard, the examined chapters of Stahl (2008) and Rockville's (2001) paper made a number of general references to a neurobiological etiology of mental distress involving neurotransmitters, neurocircuitry, neuroanatomy, and genetics. Next, in terms of critiques, Stahl (2008) discussed the complexity involved in trying to determine links between genetics and mental distress. Finally, Rockville (2001) asserted that "The precise causes of most mental disorders are not known: the broad forces that shape them are genetic, psychological, social, and cultural, which interact in ways not yet fully understood" (p. 7).

In summary, there were a few critiques surrounding the validity of a genetic based neurobiological etiology for mental distress, and an assertion about the lack of neurobiological evidence for explaining casual factors pertaining to psychiatric disorders as a whole. However, there were no specific references to depression or specific hypotheses for the disorder's etiology within the context of relevant content not captured by the coding categories for this topic.

There was a substantial amount of information about the DSM falling outside of the coding framework's requirement that the manual be explicitly mentioned on the page it was discussed. This content was documented within the "comments" section of the data collection spreadsheet. The volume of these findings was challenging to consolidate and also proved to be problematic for inter-rater reliability. This latter issue will be discussed in more detail within the limitations section. The majority of this content included references to DSM nosology, although there were also some cases in which the manual's construct validity was briefly challenged. For example, in *Primer of Drug Action*, Julien and colleagues (2011) asserted that the diagnosis of bipolar disorder in childhood or adolescence was "difficult and it is still being debated" (p. 196). In addition, the text posited that it was unclear whether "ADHD is a medical disorder, a behavioral problem mainly manifest in schools, or a disorder of human adaptation (Mayes et al., 2009)" (Julien et al., 2011, p. 610).

Similarly, Stahl (2013) asserted that the diagnostic criteria for schizophrenia and bipolar disorder were not sufficiently sensitive to capture some relevant cases that would be overlooked by the DSM-IV-TR's criterion. In addition, Stahl noted that the manual's interpretation of antidepressant induced mania as a substance induced disorder ignored the possibility of a pre-existing disposition for bipolar. The researcher did not find any critiques of the DSM's inter-rater reliability within this subset of relevant, albeit uncoded, content. Thus, overall, a comprehensive analysis of the DSM that addressed issues surrounding race, gender, socioeconomic status, and well established problems with the manual's inter-rater reliability and construct validity appeared to be missing within the

examined materials regardless of whether the relevant content falling outside of the coding categories was included.

### **Interview With Examined Psychologist Postdoctoral Psychopharmacology Training Program's Director**

The examined psychologist postdoctoral psychopharmacology program's Director was interviewed for roughly an hour on April 10, 2015. However, on April 12, 2015, the Director requested that the interview responses be excluded from the study. For a list of the interview questions see Appendix U.

## Chapter IV: Discussion

The discussion addresses considerations surrounding the extent to which the analyzed curriculum materials from the examined psychologist postdoctoral psychopharmacology program's curriculum and the PEP Review DVD materials (including the PEP Practice Questions), integrated critiques of neurobiologically based hypotheses of depression, identified challenges to the DSM's rigor, and discussed the c/s/x population and movement. This will be followed by an Integrative Discussion.

### **Examined Psychologist Postdoctoral Psychopharmacology Training Program**

**Syllabi.** The brevity of the syllabi's format conceivably limited the extent to which the three analyzed topics could be addressed therein. Nonetheless, the study's analysis of the examined program's syllabi indicated that some efforts had been made to address nonconventional perspectives about psychopharmacology. For example, the Semester Project (SP) required students to read one book that critiqued various aspects of psychopharmacology and write a paper critiquing the author's main arguments. However, it could be argued that challenging a critique of conventional perspectives about psychopharmacology leads the student back to conventional thinking—unless students are required to critique conventional viewpoints as well. It also warrants mentioning that students were required to read two articles and watch a TedTalk video that challenged conventional perspectives about pharmacological interventions.

Next, the analysis of the syllabi clarified that neurobiologically based hypotheses for the etiology of depression were being addressed within the examined iteration of the analyzed psychologist postdoctoral psychopharmacology program. It also warrants mentioning that the syllabus recommended students read the Carlat Report, an online

source which critically analyzes a variety of psychopharmacology topics (www.thecarlatreport.com). While this indicated that students were encouraged to be critical of conventional perspectives there was no way to confirm whether these materials were being accessed or, if they were, which articles students were choosing to read. Further, there is evidence that compliance rates for required readings are low, particularly for graduate students with work and family responsibilities. Additionally these readings were recommended and non-specific which also raises questions about the likelihood of compliance.

Further, in considering *c/s/x* content it is noteworthy that a search of the Carlat Report's website did not yield any information for the following search terms: "survivor," "MindFreedom," "Icarus Project," "MadPride," "*c/s/x*," "ex-patient" (www.thecarlatreport.com). Additionally, a search of the website using the term "consumer" yielded links to six documents—one which focused on depression and how to be a more "informed consumer of medical research," another that examined which Electronic Health Records consumers should purchase, and a third paper which explored issues surrounding psychiatrists' management of their web presence (i.e., nothing about the *c/s/x* movement). Similarly, the fourth paper addressed the FDA's order for consumer medication guides for stimulant medications, the fifth paper covered the use of Adderal by college students, and the sixth article explored the complexities of prescribing generic drugs to patients. Consequently, while the Carlat Report likely provides some critiques of hypotheses for neurobiological etiologies of depression and the DMS's rigor, it seems unlikely that the resource provided any substantial examinations of the *c/s/x* movement.

## Non-Semester Project Books

**Etiology of depression.** It was notable that the majority of the examined Non-Semester Project books did not critique all of the neurobiological hypotheses of depression disseminated. Further, only one of the examined NSP readings made an effort to outline a variety of different problems with a particular hypothesis. For example, Stahl's (2013) critique of the monoamine hypothesis for depression provided a relatively detailed critique that outlined the origins, support, and shortcomings of this hypothesis. In other cases, challenges to neurobiological hypotheses of depression being disseminated within the examined NSP texts' readings provided only cursory critiques. Here, the readings noted that research findings were "inconsistent," "disparate," "controversial" or "discordant" or, in some cases, the existence of "conflicting evidence" was identified (Brunton et al., 2010, p. 708; Schatzberg & Nemeroff, 2009, pp. 488, 911, 913, 928). In rare occurrences, the limits of a particular study's methodology were briefly mentioned (i.e., the "use and overuse" of brain scans) (McCance et al., 2010, p. 646).

In this regard, it would have been useful for the text to offer at least a brief summary surrounding the limits of psychopathology based neuroscientific research as a means to identify important caveats for the findings being presented (i.e., by reviewing the limits of neuroimaging technology, identifying shortcomings of animal research, acknowledging problems with using the DSM as a diagnostic tool for research, and discussing potential biases in the reporting and publishing of some research findings).

Next, it warrants mentioning that the only examined NSP textbook reading (i.e., *Pathophysiology: The Biological Basis of Disease*, 2009), to critique all of the neurobiological hypotheses for depression discussed within its examined pages focused

these challenges exclusively on PMDD - a condition that lay outside of the DSM-IV-TR's established disorders. Similarly, it was notable that the brief critique of the monoamine hypothesis in the recommended *Manual of Clinical Psychopharmacology for Nurses* (2013) also occurred within the context of disseminating the symptoms of PMDD. It is unclear how the omission of consistent and/or thorough critiques of neurobiological factors for the etiology of depression impacts a new prescriber's understanding about the causes of mental distress, the rigor of the DSM, and the implications this could have on acquiring informed consent. These latter considerations will be addressed in greater detail during the study's Integrated Discussion.

It was also notable that four neurobiological based hypotheses for depression disseminated across the body of examined NSP readings were not critiqued at all and that four of the examined readings' discussed the monoamine hypothesis of depression without any provisions for critiques therefore. The chemical imbalance hypothesis has not received sufficient support to warrant its consideration without a thorough deconstruction of the evidence used to support it (Stahl, 2008, 2013; Whitaker, 2010).

Next, the researcher queried the author of one NSP book that did not integrate critiques for a number of neurobiological hypotheses for depression's etiology. This author, who wished to remain anonymous, reported having relied on support from colleagues with expertise in affective disorders (anonymous, personal communication, March 7, 2014). His response raises questions about the responsibility of a primary author or editor when it comes to integrating critical analyses of content provided by other experts.

That none of the 16 examined chapters from *The American Psychiatric Publishing Textbook of Psychopharmacology* (2009) challenged the monoamine or neuroendocrine hypotheses of depression, despite having addressed these hypotheses in some detail, also warrants revisiting. Particularly since students were required to read roughly a quarter of this textbook. This raises questions surrounding the level of importance that the examined program's curriculum developers placed upon critical examinations of conventional viewpoints.

Next, *Neuroanatomy through Clinical Cases* (2010)—which was read in its entirety—identified five neurobiological hypotheses for the etiology of depression, without critiquing any of them. While the text was not designed to focus on the neurobiology of affective disorders, it is concerning that no critiques were integrated therein. Again, this raises questions about the implicit message that omitting critiques might convey to students (i.e., that these critiques may not be essential to consider when it comes to safe and effective prescription practices and obtaining informed consent).

Additionally, the researcher observed some notable differences in the ways NSP textbooks conceptualized neurobiological causes of depression. For example, some neurobiological hypotheses of depression were ascribed to the disorder's etiology while other authors conceptualized the same hypotheses within the context of disease pathophysiology. These heterogeneities in conceptualizing depression likely stem from ongoing uncertainties about depression's neurobiological causes and symptomatology. Further, some degree of overlap might be expected between both characterizations of the disease process. For example, it is plausible that apoptosis may represent a cause and consequence of depression.



Another observation was that Stahl (2013) referred to the “monoamine hypothesis of depression” and also called it a “classic theory about the biological etiology of depression” (p. 262). Whitaker (2010) referred to the “chemical imbalance theory” (p. 10) as did the presenters of both examined videos from the analyzed psychologist postdoctoral psychopharmacology training program. Carlat (2011) stated that “Officially this [chemical imbalance] theory of depression is known as the ‘monoamine hypothesis’.” Next, Julien and colleagues (2011) described the “neurogenic theory of depression” (p. 143)—a premise that has also been identified as the neurogenic hypothesis and neurogenesis hypothesis of depression (Eisch & Petric, 2012; Sapolsky, 2004). The reason for these discrepancies is unclear although differences in opinion about the strength of the respective hypothesis or theory or loose adherence to scientific principles in this context, seem like plausible explanations.

Another observation with regard to the examination of NSP book findings was the complete absence of citations within *Stahl’s Essential Psychopharmacology: Neuropsychological Basis and Practical Applications* (2013). This omission made it difficult to determine which studies were being used to support the author’s assertions. In justifying the decision to omit citations, Stahl asserted “We have a large number of references at the end of the book, for each chapter. Not every statement is referenced in the body of the text as this is a not a review, but a textbook” (S. Stahl, personal communication, November 12, 2014).

By comparison, Julien and colleague’s (2011) book incorporated 52 citations (the majority were primary source studies) for the first three chapters, while Stahl’s book included references to five suggested readings for the first three chapters of his text—all

from secondary sources and arguably not indicative of a “large number of references” (S. Stahl, personal communication, November 12, 2014). Similarly, while Stahl provided 32 citations exclusively from primary sources for the chapters on mood disorders (chapter six), antidepressants (chapter seven), and mood stabilizers (chapter eight), Julien et al. (2011) cited 139 sources in their text’s chapter on antidepressants.

Stahl’s justification for omitting citations in favor of recommended readings is inconsistent with the book’s title which specified that the text would address the “Neuroscientific Basis” of psychopharmacology. According to the Merriam Webster dictionary, the term *basis* is defined as, “something on which something else is established or based” and the Oxford Dictionary’s definition states that *basis* is “the underlying support or foundation for an idea, argument, or process” and “the justification for or reasoning behind something.”

The relevant content that fell outside of the study’s coding categories also requires discussing. In retrospect, the provision of an additional category to capture “broad critiques” that challenged conventional viewpoints about the etiology of mental distress may have been more effective than documenting this information within a catch-all “comments” section. For example, in the *Manual of Clinical Psychopharmacology for Nurses*, Leahy noted that “Although the field of psychopharmacology has experienced a revolution over the past two decades, much remains unknown about the human brain, neuropathology, and neurotransmitters as they relate to an individual’s thoughts, moods, and behaviors” (Leahy & Kohler, 2013, p. xxviii). Similarly, in *Neuroanatomy Through Clinical Cases*, Blumenfeld (2010) posited that “many processes of the mind, particularly certain aspects of consciousness and emotions, remain difficult to explain fully in

neurophysiological terms” and, “Emotions and drives are almost as difficult to explain as consciousness itself” (pp. 974, 976). Further, in *Psychopharmacology for Psychologists: Prescribing and Collaborative Roles*, McGrath and Moore (2010) asserted that “The psychobiosocial model does not circumscribe the nature of human suffering or problems with adaptation as a ‘chemical imbalance’” (p. 113). Next Blumenfeld (2010) asserted that “Emotions and drives are almost as difficult to explain as consciousness itself” (p. 976). In the same vein, within the context of discussing the pharmacological treatment of depression, Bresee, Gotto, and Rapaport acknowledged “disparate biological findings” with regard to the disorder’s “biological underpinnings” (as cited in Schatzberg & Nemeroff, 2009, p. 1090). While these comments acknowledge that the body of evidence for neurobiological etiologies of mental distress is limited, they do not address the particular shortcomings of each theory.

Given that the majority of NSP books were not read in their entirety it warrants considering whether relevant content surrounding a neurobiological etiology of depression may have been missed within the unexamined chapters of the partially analyzed books. For example, in reviewing the *American Psychiatric Publishing Textbook of Psychopharmacology’s* (2009) chapter on the *Neurobiology of Mood Disorders*—which was not a required reading—the researcher found that seven of the ten neurobiologically based etiological hypotheses of depression being presented were critiqued. This indicates that the topic was being addressed elsewhere in the book, albeit with inconsistent critiques.

It was notable that the study’s analysis of *Stahl’s Essential Psychopharmacology* (2013) did include the chapter on mood disorders, and the chapter on antidepressants. In

addition, the study examined the book's chapter on chemical neurotransmission, and another on transporters, receptors, and enzymes as targets of psychotropic drug action. All of these chapters cover topics related to the etiology of depression which suggests that this topic would have been covered within the required pages of this text. Similarly, while the analysis of *A Primer of Drug Action* (2011) did not examine nine of the book's 20 chapters, the study's analysis did include the text's required chapter on pharmacodynamics, another on neurotransmission, and a third on antidepressant drugs. Further, there was no chapter which explicitly noted depression or mood disorders within this book. Consequently, the three aforementioned chapters would ostensibly have been the most likely sources of information about a neurobiological etiology of depression within this textbook.

Next, in reviewing a chapter (not a required reading) in *Pathophysiology: The Biological Basis for Disease in Adults and Children* (2010) focusing on mood disorders, the researcher found a similar dearth of critiques. For example, five neurobiological hypotheses of depression's etiology were addressed on 17 pages of text and there was one brief critique for a neurotransmitter hypothesis of depression's etiology—this was located within the fine print below a figure and stated that “In depressed individuals, neurotransmitter levels are hypothesized to be reduced. The mechanisms responsible for this reduction are not understood” (Takahashi, as cited in McCance et al., 2010, p. 655).

Additionally, in examining the index of *Pharmacotherapy for Psychologists* (2010), the researcher found that there were seven pages with content on “depression” and “major depressive disorder” and no listings for “mood disorders” or “affective disorders.” Neither of the two pages which had not been examined within the required

readings focused attention on the etiology of depression. In *Essential Evidence-Based Psychopharmacology* (2012), the book's index listed a total of 21 pages with content on "depression," and "major depressive disorder"—there were no listings under "mood disorders" or "affective disorders." Five of these pages had not been examined as part of the study's analysis and none of these pages contained content about the etiology of depression. It is also notable that the study's analysis of this text did include the required chapter on *Evidence-Based Pharmacotherapy of Major Depressive Disorder*—ostensibly the most likely source within that text for any content addressing a neurobiological etiology of depression.

Next, in reviewing the index for *Neurosciences* (2012), another text that was not analyzed in its entirety, the researcher found that a total of four pages were listed under "depression," "major depressive disorder," "mood disorders," and "affective disorders," and all within one of the unexamined chapters. These pages were part of the chapter on *Emotions* wherein Purves and colleagues (2012) noted that "Despite evidence for a genetic predisposition and an increasing understanding of the brain areas involved [in affective disorders], the cause of these conditions remains unknown" (p. 660). It also warrants reiterating here that students were required to read this textbook's chapter on *Neurotransmitters and Their Receptors* which contained a full page "Box" discussion about "Biogenic Amine Neurotransmitters and Psychiatric Disorders" (p. 126), and none of the content therein critiqued the chemical imbalance hypothesis of mental distress.

An analysis of the index from *Goodman and Gilman's: The Pharmacological Basis of Therapeutics* (2011) found 19 pages of text listed under primary headings for "depression," and "major depressive disorder"—there were no listings under "mood

disorders” or “affective disorders.” Of these 19 pages, there was only one page that had not been analyzed within the examined psychologist postdoctoral psychopharmacology program curriculum’s required readings. This particular page was part of the chapter entitled *5-Hydroxytryptamine (Serotonin) and Dopamine*, and included content which focused on research into animal models of depression. For example, Sanders-Bush and Hazelwood asserted that “Mutant mice lacking the 5-HT transporter display anxiety and a ‘depressive-like’ phenotype (Fox et al., 2007)” (as cited in Brunton et al., 2011, p. 343). These authors also noted that 5-HT receptors have been “implicated in the animal models of depressions, such as learned helplessness” (p. 343). In what might be seen as a critique for the neurotransmitter hypothesis of depression, Sanders-Bush and Hazelwood described how manipulated reductions in the amount of serotonin in the brain could abruptly reverse the efficacy of SSRI’s for depression. Here, the authors posited that “This clinical finding adds credence to somewhat less convincing neurochemical findings that suggest a role for 5-HT in the pathogenesis of depression” (as cited in Brunton et al., 2011, p. 343).

In reviewing the index of *Pharmacotherapy: A Pathophysiologic Approach* (2011), the researcher found a combined total of 18 pages with content on depression identified via primary headings for “depressive disorder” and “mood disorder”—there were no primary headings for “affective disorders” or “major depressive disorder” (Teter, Kando, & Wells, as cited in Dipiro et al., 2011). Sixteen of these pages were contained within the book’s chapter, *Major Depressive Disorder*. An analysis of this chapter found that three neurobiologically based hypotheses for the etiology of depression were

addressed (i.e., neurotransmitter, neurotrophin, and neuroendocrine hypotheses) and critiqued.

For example, Teter and colleagues asserted that “It is apparent that no single neurotransmitter theory of depression is adequate” and noted that the involvement of neurotrophins within the etiology of depression “is a relatively recent theory, which has not been firmly established” (as cited in Dipiro et al., 2011, p. 1174). Next, the authors of this chapter acknowledged the “high rate of false positive and false negative results associated with neuroendocrine abnormalities in depressed patients” (p. 1175).

The two additional pages with information on depression as identified within the text’s index were both located within the chapter on *Pregnancy and Lactation: Therapeutic Considerations* (Dipiro et al., 2011). This content focused on depression within the context of discussing mental health conditions during pregnancy; more specifically, epidemiological findings for the disorder and considerations surrounding the use of antidepressants during the neonatal period (i.e., p. 1369). This chapter also identified considerations pertaining to the psychotropic treatment of post-partum-depression.

**DSM.** Variability in DSM content across and within the Non-Semester Project books was anticipated. For example, a number of texts dealt with topics largely unrelated to the manual (i.e., *Seeley’s Anatomy and Physiology*, 2011; *The Atlas of Functional Neuroanatomy*, 2005; and *Neurosciences*, 2012). In addition, as noted elsewhere in this analysis, the examined program did not require its students to read all of the curriculum’s books in their entirety. Thus, it is possible that partially examined books contained critiques of the DSM within omitted chapters.

At the same time, the absence of any explicit mentions of the DSM within *Neuroanatomy through Clinical Cases* (Blumenfeld, 2010), warrants mentioning. For example, despite having focused on medical conditions, there was a section within the textbook's chapter on the *Anatomical and Neuropharmacological Basis of Psychiatric Disorders* which discussed the symptomatology of schizophrenia, obsessive compulsive disorder, anxiety disorders, depression, and mania. The symptoms of major depressive disorder and, to a lesser extent mania and anxiety disorders, were also discussed in chapter three.

Given the amount of content directly related to the DSM (i.e., its disorders and respective symptomatology) it was surprising that the manual was never explicitly referenced within Blumenfeld's (2011) textbook. The use of DSM nomenclature within the context of discussing neuroanatomical considerations for psychopathology, without explicitly identifying or addressing the manual's shortcomings could be interpreted as an implicit message to readers that the limits of the DSM's diagnostic validity and reliability are secondary to the very findings that the manual is used to conceptualize. This might lead students to conclude that these considerations are mutually exclusive, reiterating concerns about how prescribing psychologists are being trained when it comes to addressing the empirical limits of the DSM as a necessary component of acquiring informed consent from clients and prescribing medications with potentially serious health risks.

Similarly, the absence of explicit references to the DSM within the *Clinical Handbook of Psychotropic Drugs* (2011) was notable because the medications addressed therein are used to treat DSM disorders. Further, problems with the manual's construct



validity and inter-rater reliability have implications for the pharmaceutical interventions discussed within the handbook (i.e., the issue of informed consent and the ramifications of false positive or false negative diagnoses when it comes to psychopharmacological interventions).

DSM content within the *Manual of Clinical Psychopharmacology for Nurses* (2013) warrants further mention because of the extent to which this text integrated the manual within its coverage of mental disorders and psychotropic interventions. For example, the text noted within its introduction that “In particular, contents of each chapter focus on diagnostic criteria and neurobiology of the relevant disorder(s), pharmacological choices, and recommendations on monitoring, side effects to consider, and treatments with special populations and those with medical illnesses” (Leahy & Kohler, 2013, p. xxvi).

Further, despite discussing the manual to varying degrees within 55 pages of text, there was only one instance in which the DSM’s empirical rigor was critiqued in a manner that met coding framework requirements for this topic (Leahy & Kohler, 2013). The omission of any thorough critiques of the DSM therein gives the impression that the manual is a safe and effective tool for empirically diagnosing patients and prescribing psychotropic drugs based on these prescriptions.

Similarly, despite having briefly identified limitations with the manual’s classification of general anxiety disorder and acknowledged that the clinical features of bipolar disorder may differ between adolescents and youths, there were no thorough critiques of the DSM’s empirical rigor with the 17 examined chapters of the *American Psychiatric Publishing Textbook of Psychiatry* (2009).

In *Stahl's Essential Psychopharmacology* (2013), the author explicitly noted on a few occasions that the reader should obtain an additional reference source for a comprehensive description of diagnostic considerations including “ongoing debates” about diagnostic criteria (p. 573). Thus, while the reader was not provided with any information about where to find these critiques, Stahl had made an effort to highlight the reader’s responsibility in augmenting their readings of his text to address these considerations

In considering the possibility that DSM content was addressed within the unexamined chapters of the Non-Semester Project book readings, the researcher reviewed their indices for listings under the primary headings “DSM,” “Diagnostic and Statistical Manual of Mental Disorders” and “Diagnosis.” In doing so, the researcher found two additional pages with critiques of the DSM within portions of the required texts that students did not have to read—both occurred in Stahl (2013). For example one page within the preface of *Stahl's Essential Psychopharmacology* (2013) identified the manual within the context of discussing its limitations relative to the Research Domain Criteria (RDoC). Here, Stahl (2013) noted that many of the manual’s diagnoses:

‘travel’ transdiagnostically without respecting the DSM (*Diagnostic and Statistical Manual*) of the American Psychiatric Association or the ICD (*International Classification of Diseases*). This is the future of psychiatry—the matching of symptom endophenotypes to hypothetically malfunctioning brain circuits, regulated by genes, the environment and neurotransmitters. (p. x)

The second of the aforementioned pages from *Stahl's Essential Psychopharmacology* (2013) mentioned the RDoC developer’s efforts to correlate diagnostic schemes with neuroimaging and genetic findings. In this regard, the content seemed to infer that the manual’s categorical system was insufficient in terms of its

capacity to discern between symptom domains that cut across a variety of different disorders.

**C/S/X.** The absence of any information about the c/s/x movement within the examined Non-Semester Project books' analyzed chapters was not unexpected given that the primary focus of these texts pertained to psychopharmacology and neuroscience. However, it was surprising that McGrath and Moore's (2010) book, *Pharmacotherapy for Psychologists: Prescribing and Collaborative Roles* did not address the movement within any of its chapters (required or otherwise), given the extent to which Sammons et al. (2003) had addressed the c/s/x movement within their seminal text, *Prescriptive Authority for Psychologists: A History and Guide*.

It warrants considering that there may have been content on the c/s/x movement within unexamined sections of the other partially analyzed texts. In an effort to address this, the researcher reviewed the indices of these texts using the terms "consumers", "survivors," "ex-patients," and "c/s/x." However, no additional content on the c/s/x movement was located via this analysis of the indices.

### **Semester Project Books**

**Neurobiological etiology of depression.** There were some important differences among the Semester Project books with regard to the topics covered and the depth of their respective analyses. For example, Healy's (2003) book focused on Prozac and the purported increase in suicidal ideation and completed suicides linked to SSRIs. By contrast, Carlat's (2010) *Unhinged* provided a critical examination of psychiatry as a profession and made recommendations about improving client care (i.e., longer visits involving empathic listening and cognitive-behavioral therapy).

Next, Moynihan and Cassels (2006) centered their analysis on the pharmaceutical industry and the negative impacts of marketing strategies on consumers. By comparison, in *The Medicalization of Society*, Conrad (2007) focused more attention on the processes by which human conditions are transformed into medical disorders. In Dubovsky and Dubovsky's (2007), *Psychotropic Drug Prescriber's Guide*, examination of the pharmaceutical industry, relatively more attention was focused on the issue of bias within academic publications relative to the other SP books. In addition, this text provided specific recommendations for how to critically interpret drug research. Robert Whitaker's (2010), *Anatomy of an Epidemic*, sought to address the question of why increasing numbers of Americans are disabled by mental illness.

Whitaker's (2010), Carlat's (2010), and Healy's (2003) books provided considerably more content on individual cases and stories relative to the other SP books. These heterogeneities meant that students' exposure to certain issues was largely dependent on which SP book they had selected.

It was noteworthy that half of the Semester Project books focused solely on the monoamine hypothesis of depression without addressing any other neurobiological hypotheses for the etiology of mood disorders. The potential reasons for this are worth considering. For instance, Prozac's mechanism of action has traditionally been conceptualized as inhibiting the reuptake of serotonin which is consistent with the chemical imbalance hypothesis for depression. Consequently, it is not surprising that Healy (2003) would focus attention on deconstructing this particular hypothesis. In addition, Healy concentrated on sociopolitical factors surrounding antidepressants, including the FDA approval process, and the marketing of Prozac. This meant that there

was less space for reviewing other neurobiological hypotheses of depression.

Nonetheless, the absence of content pertaining to additional hypotheses for the disorder limited the scope of Healy's analysis.

In discussing *Anatomy of an Epidemic*, Whitaker (2010) reported having focused his attention on the monoamine hypothesis because:

a) this chemical imbalance theory was the prevailing theory that has been told to the public about why the drugs are effective. And so, if you are going to put that public story under scrutiny, you need to review whether it is so; b) second, the research into the chemical imbalance hypothesis led researchers to come to an understanding of how the drugs affect the brain over the longer term, and that is they induce compensatory changes the opposite of what was originally intended. Thus, by focusing on the chemical imbalance theory, the research provides a fairly clear picture of how the drugs act on the brain, short term and long term. Thus, you can then frame the question: how does this drug action, which includes compensatory changes, affect people over the long term? And the point here is this: I am not really focused on hypotheses for the disorder—that would be a different book. I am focused on how the drugs affect the brain, and then what science tells us about their long-term effects. (Personal communication, July 19, 2015)

In considering potential differences between these books it is also worth noting that Healy and Carlat's prescription practices may be more liberal than the ones Whitaker would subscribe to. For example, Healy (2003) described himself as:

someone committed to both pharmacotherapy and ECT. In my opinion the idea of getting by without physical treatments, hoping psychotherapy alone will do the job is a romantic notion. Romance is nice but doesn't make people well. Drug therapies have done a great deal to improve things, to the point where critics of psychiatry are taken seriously when they argue there is no such thing as mental illness. Sadly, the history of psychiatry does not bear out arguments that all of this is just a matter of social control, or that the problems for which physical treatments are given would respond to psychotherapies if sufficient time and skilled therapists were available. (2003, pp. 17–18)

Similarly, Carlat (2010) described psychopharmacological interventions as being “remarkably helpful to patients”, and noted that he regularly prescribed antidepressants

(p. 16). Conversely, while Whitaker (2010) agreed that psychotropic prescription drugs might be helpful in some circumstances, he raised relatively more concerns than Healy (2003) and Carlat (2010), about the long-term safety risks and questionable efficacy of psychotropic medications.

In describing his assessment of *Anatomy of an Epidemic*, Carlat (2011) asserted that “My overall take is that Whitaker has his basic facts right, and that he communicates them in a compelling style that I envy. But I disagree with his interpretation of the facts” (para. 5). Further, Carlat noted that “Whitaker does a great job documenting an astonishing rise in psychiatric disability, but he erroneously blames the drugs, when the actual causes are more nuanced and multifactorial” (Carlat, 2011, Cause #3, para. 2).

It was notable that Dubovsky and Dubovsky (2007) chapter on *How to Identify and Deal With Marketing* did not discuss how the chemical imbalance hypothesis had been used to advertise, sell, and prescribe psychotropic medications—an arguably pertinent issue within the context of this particular discussion. If a student from the examined iteration of the psychologist postdoctoral psychopharmacology training program had selected the Dubovskys’ (2007) or Conrad’s (2007) book for the Semester Project, their exposure to critiques of this hypothesis within the context of this particular assignment would have been extremely limited in the first case and non-existent in the second. There was also a strong possibility that students would not be exposed to critical analyses of other neurobiological hypotheses of depression during this assignment because only one of the three SP texts addressing alternatives to the monoamine hypotheses for depression provided any critiques for these ideas.

Differences in the authors' training and professional experiences are also noteworthy. For example, Steven Dubovsky, Daniel Carlat and David Healy are psychiatrists, and Amelia Dubovsky was a medical student when the book she co-authored with her father was published. Robert Whitaker and Ray Moynihan are journalists, Peter Conrad a sociologist, and Alan Cassels is a pharmaceutical policy researcher. Diversity in the backgrounds of these authors has the potential to expose students to a variety of perspectives—but only if they are required to read more than one book for this assignment.

**DSM.** It is conceivable that students completing the examined iteration of the analyzed psychologist postdoctoral psychopharmacology program, within the context of the Semester Project and its six recommended books (of which students were required to select one book), could have missed being exposed to more critical perspectives about the DSM. For example, the Dubovskys' (2007) text only briefly mentioned the manual on one page and without the provision of a critique. Next, despite noting that changes in diagnostic nosology made it difficult to compare research over time (via content that did not meet coding category framework requirements), the rest of the book used DSM nomenclature without examining the manual's rigor. Further, four of the six Semester Project texts did not address the manual's inter-rater reliability at all, meaning that students' exposure to this particular issue could also have been missed within the context of their book selection for the Semester Project.

Apparent differences between the SP books' assessments of the DSM's utility also warrants discussing. For instance, while Carlat's (2010) text identified a number of

the manual's limitations, he was less critical in this regard than Whitaker. For example, Carlat (2010) stated that:

Over the years, the various versions of the DSM have been criticized and ridiculed. The book has been called a tool of the pharmaceutical industry and a collection of arbitrary labels based on shaky science. But with all its imperfections, it actually evolved out of a crying need in the profession for more precise descriptions of disorders, and it has done a great service by providing them. DSM actually represents the culmination of a profession's noble struggle to categorize the inner anarchy that is psychiatric illness. (pp. 47–48)

In contrast, Whitaker (2010) focused most of his analysis on the DSM's shortfalls. For example, while Whitaker noted that a DSM diagnosis could facilitate the provision of special services for children with ADHD, there was little indication that the author considered this to be a positive recourse given the potentially harmful pharmaceutical intervention(s) that would likely follow.

**C/S/X.** The dearth of c/s/x information within all but two of the Semester Project books suggests that students were unlikely to be introduced to the c/s/x movement unless they read Whitaker's (2010) or Carlat's book. Further, while *Unhinged* provided a brief discussion of Julie Lawrence's personal reservations about the use of electroconvulsive therapy, no other activists from the c/s/x movement were mentioned (Carlat, 2010). Nor did Carlat explicitly focus on other aspects of the c/s/x movement (i.e., concerns about forced drugging, forced hospitalization, ECT, and the limited involvement of consumers/survivors/ex-patients within the mental health care system).

Whitaker's (2010) book stood out in this regard because it addressed the c/s/x movement's history, activism, campaigns, events, advocacy efforts, and organizations within 40 pages of text. Further, Whitaker made a concerted effort to integrate the personal stories of c/s/x activists and reference a number of their works.



It also warrants mentioning Healy's and Carlat's references to "fringe groups" and organizations linked to the Church of Scientology which have been involved in critiques of psychiatry. Regardless of their respective intentions, the use of the term "fringe," without clarifying its parameters more effectively could be interpreted as discrediting consumer/survivor/ex-patient organizations and their initiatives to critique the discipline of psychiatry and center mental health practices. For example, c/s/x activist David Oaks (2006) noted that some academics "Align the history of our movement with the 'radical left' to a great extent, ignoring decades of outstanding work by conservatives and libertarians in fighting psychiatric abuse" (p. 1212).

### **Non-Textbook Readings**

**Neurobiological etiology of depression.** There was limited content about hypotheses for a neurobiological etiology of depression, within the non-textbook readings. This was not particularly surprising given that the majority of these readings dealt with topics indirectly related to depression's etiology. It could be argued that more space should have been afforded to the provision of caveats challenging the disorder's neurobiological etiology—particularly since four of the five papers which did identify a hypothesis for depression did not integrate critiques therefore (e.g., the *American Academy of Child and Adolescent Psychiatry's Guidelines for the Assessment and Treatment of Children and Adolescents with Depressive Disorders*, 2007, which specifically focused on depression and its psychopharmacological treatment).

**DSM.** It was anticipated that only a minority of the non-textbook readings would contain information about the DSM. Many of these articles addressed issues related to medications and fell outside of diagnostic considerations pertaining to psychiatric

disorders (i.e., avoiding medication errors, surveys of mental health coverage, lab values, and dangerous abbreviation lists). Nonetheless, the researcher was surprised at the number of non-textbook readings which discussed DSM diagnoses without actually referencing the manual. For example, the Harvard Medical School Algorithms for Bipolar Depression (Ansari & Osser, 2010a), Bipolar Mania (Ansari & Osser, 2010b), Post Traumatic Stress Disorder (Bajor, Ticlea, & Osser, 2011), Social Anxiety Disorder (Osser & Dunlop, 2010), Psychotic Depression (Tang & Osser, 2012), and Schizophrenia (Osser, Roudsari, & Manschreck, 2013) made no mention of the manual or its limitations. Similarly, one might assume that research papers on the effectiveness of antidepressants and antipsychotic drugs would reference the manual and briefly review controversies surrounding its applications within the contexts of drug trials and clinical practice. By not examining issues with the manual's construct validity and inter-rater reliability, an important methodological limitation and potential confound to these studies' data was overlooked. Further, there is an implicit message to readers that these considerations are not particularly relevant to the topic.

It was also notable that 55 of the 57 non-textbook readings (i.e., 96.5%) did not address the issues surrounding the DSM's inter-rater reliability levels and only one of the five papers dealing with the manual's construct validity did so in any detail (i.e., less than 1% of the non-textbook readings). Next, this latter paper focused more on developmental considerations in diagnosing bipolar disorder among children than in critiquing the manual itself. It is concerning that the researcher could not find any thorough examinations of the DSM's limitations across the entire body of the examined psychologist postdoctoral psychopharmacology program's Non-Textbook readings. The

lack of any findings that comprehensively addressed problems with the manual's construct validity and inter-rater reliability within this subset of materials raises questions about the importance being afforded to this issue.

**C/S/X.** The absence of information about the c/s/x movement within the Non-Textbook readings was not particularly surprising given these papers' respective foci.

## **Videos**

**Neurobiological etiology of depression.** The finding that the *Biological Basis of Depression* video focused considerable attention on the etiology of depression and critiqued one of the six neurobiological theories for depression disseminated is notable because it sends a message to students that the information can be interpreted without attendance to its shortcomings. Further, the aforementioned critique was limited by its brevity (i.e., in noting that there were also some negative studies). While this presentation focused on the purported relationships between genes, proteins, neurocircuitry and depression, it was particularly notable that the chemical imbalance hypothesis was introduced as one of the leading theories for depression and referenced on a variety of occasions during the video—without the provision of any critiques acknowledging the lack of empirical support for the hypothesis.

Another concern was the extent to which neuroimaging research was simplified in order to elucidate neurobiological hypotheses for the etiology of depression. For example, in asserting that “we’re starting to develop these neuroimages that really show that brains operate differently when you have a psychiatric illness versus when you don’t” (i.e., from 39:09 to 39:19 in the video). The same argument could be made of the video’s conclusion that “There are many potential neurochemical causes of major

depressive disorder. There are hormonal ones, there are neurodegenerative ideas. And again it may come down to what genes you have, what proteins you have, what circuits are hot and cold” (Anonymous, n.d.) (i.e., from 31:35 to 31:44 in the video).

Despite the absence of any thorough critiques of neurobiological theories for the etiology of depression, the second presenter’s concluding statement within the *Pharmacotherapy for Depressive Disorder* video broadly challenged the extent literature surrounding the neurochemistry of mental disorders. For example, he noted that,

The chemistry involved in complex biopsychosocial operations is well beyond our present day ability to calculate let alone measure and control. Key to appreciating this complexity nevertheless is the acknowledgement of interplay and multiple feedback systems at the molecular as well as global levels. For the time being it takes deductive reasoning and an act of faith to understand the complex role of neurochemistry and even the simplest of mental operations (Anonymous, n.d.) (i.e., from 39:11 to 39:44 in the video)

At the same time, the lack of more consistent and detailed critiques of hypotheses for depression’s neurobiological etiology raises concerns about the impact that this video may have had on new prescribers and their respective levels of certainty afforded to the presented evidence on this topic.

**DSM.** In the *Biological Basis of Affective Disorders* video, the DSM was primarily referenced within the context of linking the symptomatology of depression to hypotheses for its neurobiological etiology. While the presenter noted that the manual’s categorical approach was unlikely to match the brain’s functioning, no further critique of the DSM was provided. There was a similar dearth of information within the *Pharmacotherapy of Depression* video. The absence of a thorough critique for the DSM suggests that the manual’s empirical shortcomings were considered to be outside of the respective presentations’ scopes of analysis. It is unclear whether this was based on an

assumption that students were already well versed in this information. It could be argued that pharmacological interventions should not be considered without acknowledging problems with the construct validity and inter-rater reliability of depression and bipolar disorder within the context of obtaining informed consent and prescribing psychotropic medications.

**C/S/X.** The examined videos focused on the neurobiology and psychotropic treatment of depressive disorders. Again, it was not expected that this content would yield information about the c/s/x movement. However, it is noteworthy that none of the other 54 video titles were indicative of c/s/x content either.

#### **Division 55 PEP Review DVD**

**Neurobiological etiology of depression.** The limited extent to which neurobiological hypotheses of depression's etiology were addressed and the absence of critiques for these hypotheses within the Division 55 PEP Review DVD is noteworthy because these materials represent the breadth of knowledge required to safely prescribe. A definitive neurobiological etiology for the disorder has not been established. Further, antidepressants' mechanisms of action remain uncertain. Moreover, depression is one of the most commonly diagnosed mental disorders in the United States (National Institute of Mental Health, 2015). In addition, the Centre for Disease Control and Prevention found that 11% of Americans aged 12 years and over were using an antidepressant (Pratt, Debra, & Qiuping, 2011). While the DVD recommended within its *Research Module* and resource list that students read the Carlat Report, student' compliance in this regard is unclear.

**DSM.** The absence of any explicit references to the DSM within the Division 55 PEP Review DVD's learning module's slide presentations is worthy of mention for the same reasons identified above. On the one hand, the DVD did encourage students to read the *Surgeon General's Report*—a document that contained critiques of the DSM's construct validity on a number of pages and also acknowledged problems with the manual's inter-rater reliability (Rockville, 2001). Conversely, no statistical analyses of the DSM were provided within these critiques. However, it is possible that students may have encountered this information with the recommended Carlat Report.

**C/S/X.** The absence of information about the c/s/x movement within the Division 55 PEP Review DVD and PEP Practice Questions—save one brief mention of the phenomenon's nomenclature within a footnote of the *Surgeon General's Report*, strongly indicates that this topic was completely overlooked within the training DVD. Reasoning behind the absence of this content and the implications of this will be discussed in further detail within the integrated discussion.

### **PEP Practice Questions**

Possible explanations for the absence of any PEP Practice Questions addressing the limits of the DSM's empirical rigor, critiques of neurobiological hypotheses for depression's etiology, and information about the c/s/x movement, will be provided within the integrated discussion.

### **Listed Critics**

The fact that three of the Semester Project books from the examined psychologist postdoctoral psychopharmacology program were authored by members of the study's listed critics confirms that critical discourse was valued within this curriculum. However,

there are shortcomings associated with having students select only one of these books which will be discussed further within the Integrated Discussion. The absence of any listed critics within the two analyzed videos and the PEP Review DVD suggest that the perspectives of the study's listed critics were not considered as pertinent to the content being addressed therein.

It is not surprising that the study's listed critics were well represented with the Semester Project Books which cited these experts' to support their own critiques of conventional perspectives about psychopharmacology. While one might not have anticipated finding works by the study's critics within the examined program's Non-Textbook readings given their focus on prescription related information, it is noteworthy that the curriculum did not consider assigning an article by one or more of these authors—particularly since that would have been a more efficient approach to integrating a broader range of critical perspectives than the Semester Project.

### **Content Falling Outside of the Coding Category Frameworks**

The content falling outside of the coding category frameworks largely mirrored the coded data and did not appear to change the overall findings. However, there are some important implications for the study's validity and inter-reliability in this regard, particularly for relevant uncoded content pertaining to the DSM. This will be addressed in greater detail within the Limitations and Delimitations.

### **Integrated Discussion and Implications**

Findings from the examined body of psychologist postdoctoral psychopharmacology training materials indicated that critiques of neurobiological hypotheses for the etiology of depression were not consistently integrated therein.

Further, more detailed analyses of etiological hypotheses of depression focused solely on the monoamine hypothesis which is no longer considered a viable explanation for the disorder. Of particular note here was the examined program's video presentation on the neurobiology of depression which highlighted but never deconstructed the chemical imbalance hypothesis or any of the other hypotheses that were discussed therein.

Next, critiques of the DSM primarily dealt with its construct validity versus problems with the manual's inter-rater reliability. Additionally, the researcher only found one example within the entire body of examined materials where a critique of the DSM integrated statistical data (i.e., one sentence within the recommended book *Unhinged*). This was surprising given the well documented empirical problems with the manual and the implications of using DSM to inform decisions about psychotropic drug interventions that have the potential to cause serious health problems, particularly for already marginalized groups like the c/s/x population.

Additionally, there was only one resource (i.e., the recommended book *Anatomy of an Epidemic*) with substantial content about the c/s/x movement. It is unclear why a well-established movement and cultural phenomenon representing some of the most marginalized members of our population and alleging human rights violations from coercive psychiatric practices would not be a topic of focus within the training that psychologists receive in order to prescribe.

These conclusions about the extent of critical discourse on a neurobiologically based hypothesis for depression's etiology and challenges to the DSM are consistent with comments made by Marlin Hoover (M. Hoover, personal communications, November 23 and December 10, 2014), a prescribing psychologist who developed the *American*



*Psychological Association's Division 55 PEP Review DVD*, and Sean Ransom—another psychologist with prescriptive authority. For example, in describing his own psychologist postdoctoral psychopharmacology training experience, Ransom noted that critiques of neurobiological hypotheses of depression and challenges to the DSM's rigor were almost entirely limited to Carlat's (2010) book, *Unhinged*. Ransom indicated that the curriculum he completed primarily focused on treatment approaches, drugs' mechanisms of action, and other information pertaining to the safe and effective prescription of psychotropic medications (S. Ransom, personal communications, November 7, 15, and 17, 2015).

When queried about his knowledge surrounding the c/s/x movement, Ransom responded that he was aware of groups funded by the Church of Scientology that had critiqued psychiatry and the discipline of psychopharmacology (S. Ransom, personal communications, November 7 and 15, 2014). Further, he was aware that psychiatric patients had spoken out against the discipline of psychiatry. However, Ransom indicated that he was unfamiliar with the consumer/survivor/ex-patient movement per se. Next, Ransom asserted that neurobiologically based etiological hypotheses of depression and challenges to the DSM's rigor should be addressed within graduate programs for psychologists versus psychologist postdoctoral psychopharmacology training. In this regard, Ransom reported having been sufficiently exposed to critiques of psychopharmacology during his own graduate training (S. Ransom, personal communications, November 7 and 15, 2014).

Hoover shared Ransom's belief that psychologists should be well versed in the limits of neurobiological hypotheses for mental disorders as well as challenges to the

empirical rigor of the DSM, prior to starting psychologist postdoctoral psychopharmacology training (M. Hoover, personal communications, November 23 and December 10, 2014). This argument notwithstanding, Hoover asserted “I think doctoral clinical psychologists SHOULD be well versed in all of the literature, including the critical literature, about psychopharmacology (and all treatment options, by the way.) I would not assert that they ARE” (M. Hoover, personal communications, November 23 and December 10, 2014).

When queried about the seemingly limited extent to which the DSM’s shortcomings were being addressed in the examined curriculum and within the Division 55 PEP Review DVD, Hoover stated “It’s about treating symptoms not diagnoses. This may make it appear that I don’t think the limits of the DSM are important—they are. But psychopharmacologists treat symptoms not diagnoses.” Hoover also asserted that “The review class may have some acknowledgement of the DSM, [but] it does not play heavily in course work and the review process” (M. Hoover, personal communications, November 23 and December 10, 2014).

In addition, Hoover noted that critical perspectives about psychopharmacology should be addressed within the “regular clinical coursework and introductory coursework” that psychologists complete during their graduate studies (M. Hoover, personal communication, December 10, 2014). Similarly, Hoover asserted that:

all clinical psychologists need to be introduced to criticism of ALL treatment options . . . and when an already practicing licensed clinical psychologist is electing to take training in psychopharmacology that education must be devoted to safe and effective psychopharmacology practice that meets the standard of care. (M. Hoover, personal communication, December 10, 2014)

The researcher could not find any data confirming the integration of critiques for conventional perspectives about psychopharmacology within psychology's graduate or undergraduate training programs' curricula. However, the *American Psychological Association's Guiding Principles for Accreditation of Programs in Professional Psychology* noted within its section on *Science and Practice* that:

all programs should enable their students to understand the value of science for the practice of psychology and the value of practice for the science of psychology, recognizing that the value of science for the practice of psychology requires attention to the empirical basis for all methods involved in psychological practice (2013b, p. 3)

It also warrants mentioning that doctoral training programs in psychology are not homogeneous. For example, according to the American Psychological Association, PhD programs typically take longer to complete than PsyD training and focus relatively more attention on research—although findings by Morgan and Cohen (2008) suggested more similarities than differences in their study which compared psychology graduate programs via a survey developed by the researchers. Dissimilarities between the disciplines of clinical psychology and counseling psychology also exist (e.g., more attention on testing versus psychotherapy, respectively). In addition, Association of Psychology Postdoctoral and Internship Centers (APPIC) accredited programs meet curriculum requirements that non-APPIC accredited programs may not be adhering to. Finally, even if one assumes that students across the diverse spectrum of doctoral level training programs in psychology are being exposed to critical perspectives about psychopharmacology, it is unclear whether sufficient attention is being paid to the implications that these critical viewpoints might have on prescribing psychotropic drugs as a psychologist with prescriptive authority.

There are important health related implications when it comes to limiting or ignoring critical discourse about the neurobiological etiology of depression and the rigor of the DSM, within the materials used to train psychologists for prescriptive authority. For example, gaps within the body of evidence used to support diagnostic and psychotropic interventions mean that treatment errors are possible. Further, these treatment mistakes can negatively impact the health of patients based on their potential for consequent iatrogenic drug effects and the negative impacts of diagnoses. If psychologists are not being trained in the limits of diagnostic assessment for psychopathology and shortcomings in the evidence supporting neurobiological hypotheses used to justify medications, it is conceivable that they may over diagnose and over prescribe. However, one could also argue that an overly conservative approach to diagnosing and prescribing psychotropic drugs could prevent some patients from receiving pharmacological treatments that would help them. In this regard, a balanced approach is needed—one that considers a variety of perspectives surrounding psychiatric practices and the evidence upon which these interventions are based. Training for prescriptive authority which integrates the critiques of the DSM's rigor and shortcomings surrounding neurobiological hypotheses for the etiology of depression is integral to this effort.

There are also ethical and socio-cultural implications for basing psychiatric interventions on inconclusive evidence—particularly when it comes to considering their impacts on marginalized groups like the c/s/x population and the importance of obtaining informed consent from all patients. The *American Psychological Association's Ethical Principles of Psychologists and Code of Conduct* (American Psychological Association,

2010) and *Practice Guidelines for Psychologists' Involvement in Psychopharmacological Issues* (American Psychological Association, 2011) highlight the importance of cultural sensitivity, informed consent, beneficence, and non-maleficence. Non-adherence to these principles of practices could ostensibly result in a breach of ethical principles. Of interest here is Paula Caplan's (2012) formal complaint to the American Psychiatric Association's Ethics Committee in an effort to have the organization explicitly recognize that psychiatric diagnoses resulted in harm (i.e., due to job loss, child custody issues, loss of health insurance, and revocation of the right to make decisions about one's own medical and legal affairs). In this regard, it is worth noting that the American Counseling Association (2014) recently revised its *Code of Ethics* to permit counselors to refrain from making and/or reporting a diagnosis if the clinician believes that the diagnosis would be harmful to the client or others.

In terms of the legal implications of obtaining informed consent, Tenenbaum (2012) noted "Patients are also significantly less likely to bring malpractice actions if they are included in the treatment process and there is effective communication between the physician and patient" (p. 8). Being clear and upfront with patients about the limits of the DSM's rigor in diagnosing psychological distress and shortcomings in the etiological evidence upon which psychotropic interventions are based would presumably help to protect prescribing psychologists against ethical complaints and malpractice suits stemming from the informed consent process.

The apparent dearth of information about the c/s/x movement within this body of examined training materials has implications for advocacy efforts. While the *Practice Guidelines for Psychologists' Involvement in Psychopharmacological Issues* note that

psychologist may play a role in forced drugging and forced confinement, it is unclear whether increased knowledge about the c/s/x population's history and stance against coercive psychiatric practices might impact this. The limited amount of information about the c/s/x population within the body of examined psychologist postdoctoral psychopharmacology training materials suggests that current advocacy efforts between psychologists and the c/s/x movement are limited at best. A search of the MindFreedomInternational website did not locate any information about prescribing psychologists and there was no indication from the c/s/x activists that the researcher has spoken to about advocacy efforts between the c/s/x population and prescribing psychologists. If this is true it would be interesting to know what happened to the recommendations of the psychologist prescriptive authority movement's early founders who made such a concerted effort to outline the importance of advocating for the c/s/x population within the seminal text by Sammons et al. (2003).

In *Talking Back to Psychiatry*, Morrison (2005) concluded that:

The challenge of gaining support from the general public and of joining with other movements is of strategic importance for the future of this movement. The everyday realities of its members are driven by their own internal identities as activists who resist the power of psychiatry to define their lives, in the various ways they do their advocacy work. The [c/s/x] movement will continue, as it has for thirty years, shaped by their choices, their alliances, and the responses of psychiatry, policymakers, and the public. (p. 174)

Similarly, Crossley and Crossley (2001) asserted that "the 'voices' of mental patients or users constitute a social, historical, and political construct" and posited that:

[the] formulation of voice remains dependant on specific schemas of habitus which shape it in various ways. Simultaneously, however, it depends on the existence of audiences and relations of symbolic power which allow it to be heard. And both of these factors are, in turn, related to the growth of social movements within the mental health field; their relationship to social movement

activity outside of that field; and changes within the structure of the health field itself. (p. 1488)

### **Limitations and Delimitations**

The results of this study were based on a partial analysis of one psychologist postdoctoral psychopharmacology program's curriculum, the Division 55 PEP Review DVD, and 160 PEP Practice Questions. In addition, not all of the textbooks were analyzed in their entirety. According to the examined program's website, there were also online resources, chats and discussion boards, as well as case formulations and presentations. Consequently, it is possible that these other learning materials and formats may have provided additional examples of critiques for neurobiological hypotheses of depression, challenges to the DSM's rigor, and information about the c/s/x movement. As such, this study's findings should not be interpreted as an exhaustive analysis of the examined program's curriculum, of any other psychologist postdoctoral psychopharmacology program's curriculum. Further, curricula are dynamic and more current syllabi from the examined program likely differ from the iteration examined by this researcher. Similarly, some of the information within the Division 55 PEP Review DVD may be outdated.

It also warrants mentioning that content analysis and the process of determining coding frameworks and categories are inherently subjective processes. For example, another researcher might have conceptualized this study's topics using different categories or elected to measure the existence of critiques in different ways. Indeed, they may have chosen to focus attention on different topics altogether. Similarly, the

researcher's list of critics was not exhaustive and other researchers might have added or removed certain experts from this particular list.

A number of important limitations surrounding inter-rater reliability levels also require further consideration. For example, four of the *American Academy of Child and Adolescent Psychiatry Practice Parameters* were mistakenly excluded from the random selection of required non-textbook reading materials for the second coder and this may have impacted kappa scores. In addition, inter-rater reliability levels were not established for the examined psychologist post-doctoral psychopharmacology program's syllabi, the examined videos, or for the Division 55 PEP Review DVD—meaning that the reliability of the researcher's findings for these materials was unconfirmed.

Additionally, difficulties in establishing inter-rater agreement for content that fell outside of the coding categories, particularly with regard to the DSM, raises questions about the coding framework's reliability and validity. For example, it is possible that the researcher missed or misinterpreted DSM content, including critiques of the manual's empirical rigor, within the examined materials.

In terms of delimitations, it should be noted that the study's collective findings from the mass of examined materials was consistent with feedback from two prescribing psychologists—one of whom developed the Division 55 PEP Review DVD. Next, while the researcher omitted certain materials, the study did analyze all of the examined program's syllabi, non-textbook readings, and Semester Project books. Further, examined recommended books were read in their entirety. Also, the researcher only omitted textbooks that were unlikely to address the etiology of depression, the DSM, or the c/s/x movement (i.e., textbooks that focused on physical examination, drug references, and a



prescriber's guide). Additionally, the researcher was informed by the examined program's Director that the omitted article on ADHD from *Childhood and Adolescent Psychiatry* (2002) had been excluded from the curriculum. Finally, the examined syllabi's course titles and objectives indicated that the two examined video presentations were the best exemplars of content on neurobiological theories of depression (see Appendix M).

The likelihood that the examined psychologist postdoctoral psychopharmacology program's omitted training materials contained additional information about the three topics of analysis warrants further consideration. While it is possible that relevant information was missed in this analysis, the titles and topics of the omitted readings strongly indicated that these materials would not have focused on any of the study's three topics of analysis. Next, there was some indication that the omitted chapters from the partially examined Non-Semester Project books (i.e., chapters that were not identified within the curricula) contained a limited amount of relevant content pertaining to the DSM and etiology of depression. For example, as noted in reviewing the indices of partially examined texts the researcher found one instance in which a brief critique of the DSM's construct validity was included. However, no examples in which the manual's inter-rater reliability was challenged were located in this regard.

The examination of these texts' indices also found seven pages across four of the Non-Semester Project books in which a hypothesis for a neurobiological etiology for depression was discussed within an omitted chapter. Critiques of these four hypotheses were provided in all cases—although three were located in one text meaning that this

content may still have been missed despite student's efforts to conceivably read some but not all of the additional chapters .

Based upon the assertions within the examined syllabi that psychologists be well versed in using the DSM prior to starting the program, it seems unlikely that any of the aforementioned videos would have discussed the manual in depth (i.e., via a comprehensive analysis of the manual's empirical limits). Further, as noted, the apparent dearth of information about the DSM was consistent with findings from the analysis of the Division 55 PEP Review DVD, the PEP Practice Questions, and feedback from Hoover and Ransom about psychologist postdoctoral psychopharmacology program curricula content.

Next, the two analyzed videos which focused on the neurobiology and psychopharmacological treatment of affective disorders provided a critique for one (i.e., genetics) of the six neurobiological theories for the etiology of depression that were discussed. In this case the critique lacked specificity and was disseminated within a single sentence. While it is possible that additional critiques of these and other hypotheses for a neurobiological etiology of depression were contained within one of the other videos, the probability seems low given that the video on affective disorders was ostensibly the primary resource in terms of presentations, for this information.

Finally, based on the analysis of the syllabi, the examined psychologist postdoctoral psychopharmacology program's analyzed readings, the Division 55 PEP Review DVD, the PEP Practice Questions, and feedback from Hoover and Ransom, there is little evidence to suggest that the *c/s/x* movement was addressed within any of the unexamined training materials.

## **Future Research**

There are many possibilities for additional research in this area. For example, an analysis of other psychologist postdoctoral psychopharmacology programs is warranted to ascertain how their curriculums compare with this study's findings. In examining the syllabi from another psychologist postdoctoral psychopharmacology program the researcher did not find any textbooks by well-known critics of conventional perspectives (i.e., like the books within the examined psychologist postdoctoral psychopharmacology program's Semester Project) and the program's syllabi did not require students to read any primary source research articles.

Similarly, another psychologist postdoctoral psychopharmacology training program—which was unwilling to provide a complete set of syllabi for the study—did send five documents in response to the researcher's request for materials pertaining to the three topics of analysis. In this case, the Director of the program sent one syllabus for the “Psychopharmacology” class. The course description noted that the class would provide an “overview of neurochemistry” and indicated that students would learn to “Identify clinical signs and symptoms of neurobiologically based psychopathology.” The Director of this program also sent a study guide on research issues associated with pharmacotherapeutics. This paper identified internal and external validity as topics for review. There were no explicit references to the DSM within this document.

Next, the forwarded materials included a second “study guide” also focusing on research issues pertaining to prescription psychotropics. This document identified a number of research terms, and listed a variety of “Research Factors” including confounds to validity (i.e., experimenter bias and unblinding). Next, this paper provided a list of

research designs and a chart outlining the Federal Drug Administration's medication approval process. In addition, the Director sent an exam review document for the treatment of depression, which identified "Diagnostic Issues" (i.e., "depression versus grief" and "psychotic depressions"), "Neuroprotection and the role of BDNF," and noted "The impact of increased serotonin on dopamine neurons in both the frontal lobes and basal ganglia."

Finally, the package contained a PowerPoint on *Psychopharmacology*, comprised of 85 slides focusing on professional, legal, ethical, and interpersonal considerations. This presentation referenced a number of articles and books that critiqued the disciplines of psychiatry and psychopharmacology including, *The Americanization of Mental Illness* (Watters, 2010), *Head Case: Can Psychiatry be a Science* (Menand, 2010), *Battling the Growing Influence of the Pharmaceutical Industry* (Johnson, 2008), *Against Therapy* (Masson, 1994), *The Myth of Mental Illness* (Szasz, 1960), *The Dark Side of Psychiatric Drugs* (Bibeau, 1994), and *Exposing the Myth Makers: How Soft Sell has Replaced Hard Science* (Duncan, Miller, & Sparks, 2000). A paper by Carey (2008) titled *Change Urged in Antipsychotics Given to Kids* was also referenced but the exact source was not identified and the researcher could not locate the document. Next, the presentation noted that "inaccurate diagnosis" was a "potential shortcoming with practice guidelines."

A thorough analysis of all psychologist postdoctoral psychopharmacology training programs would help to clarify the extent to which these curriculums are integrating critical content that challenges conventional perspectives about psychopharmacology. Additionally, it would be worth investigating how psychologist postdoctoral psychopharmacology training programs' attendance to critical content

compares with the psychopharmacy training that physicians, psychiatrists, pharmacists and nurse practitioners receive. For example, are the concerns identified herein limited to one discipline or are they reflective of the training that other prescribers of psychotropic drugs receive?

Further, in addition to the topics examined by this study, it would be useful to know the extent to which psychologist postdoctoral psychopharmacology training programs and the prescription training programs of other disciplines, address critiques of neurobiological theories for a wider spectrum of disorders (i.e., schizophrenia, bi-polar disorder, and AD/HD). Additionally, research could focus on how these programs and their respective readings and videos conceptualize the history of psychopharmacology. For example, do these materials make a concerted effort to identify alternative perspectives which challenge conventional narratives about the history of psychopharmacology (i.e., the conventional belief that psychopharmacological interventions were responsible for emptying the asylums)?

It would also be of interest to learn how the acquisition of prescription privileges impacts a prescribing psychologist's continuing education choices. For example, in Illinois psychologists with prescriptive authority must complete "24 required hours of instruction relevant to prescriptive authority during the 24 months prior to application for renewal" (98th General Assembly State of Illinois 2013 and 2014 HB3704, n.d., p. 6). It is unclear whether the need to maintain psychopharmacological knowledge impacts prescribing psychologists' continuing education surrounding non-pharmaceutical interventions? Further, what are the implications of continuing education choices on prescribing psychologists' clinical practices and the papers they publish?

Other analyses might focus on the extent to which graduate training programs in psychology are integrating critical perspectives within psychopharmacology classes. Particularly since concerns have been raised about whether psychopharmacology is being addressed at all within some doctoral psychology programs. For example, Dunivan and Southwell (2000) noted “it is estimated that less than half of the APA-accredited doctoral programs and internships mention psychopharmacology training in self-studies” (S. Zlotlow, personal communication, February 8, 2000, as cited in Dunivan & Southwell, 2000, p. 610). Further, in proposing a psychopharmacology curriculum for pre-doctoral internships, Dunivan and Southwell (2010) posited that “For a considerable number of psychology interns, this seminar might represent their first formal coursework in psychopharmacology” (p. 613).

More recently, Julien (2011), a vocal supporter of prescriptive authority for psychologists, asserted “Psychopharmacology deficiencies both in psychology training programs and in post-graduate continuing education have long been noted” (p. 446).

Further, Julien stated that,

no matter one’s position on this contentious issue, clinical psychologists in general suffer a critical deficiency in pharmacology knowledge, which impedes their ability to evaluate their patient’s cognitive abilities (at a minimum), to evaluate medication side effects, and to recommend modification of existing treatments. Worst of all it impedes optimal and comprehensive care for their patients. (p. 446)

To address this, Julien recommended that the discipline make a concerted effort to offer “more comprehensive [psychopharmacology] training” to graduate and post-graduate psychology students (p. 446).

Similarly, an article by Jaffe (2010) cited a *Psychological Science in the Public Interest Report* asserting that the doctoral psychology program accreditation system had failed to ensure that graduates were sufficiently versed in scientifically validated interventions. In the same vein, Julien and colleagues (2011) faulted many training programs in clinical psychology for not upholding high academic admission standards and not emphasizing science in the curricula.

Tavris (2004) extended the critique beyond the discipline of psychology in asserting that failure to sufficiently address core principles of critical and scientific thinking was “widespread in graduate clinical psychology programs and psychiatric residencies, where students can earn a PhD or an MD without ever having considered the basic epistemological assumptions and methods of their profession” (p. xi). In addition, Tavris cited findings from ethnographic research, and asserted that “rarely do [psychiatric residents] learn to be skeptical about questions, analyze research, or consider alternative explanations or treatments” (p. xi). Thus, the researcher is not alone in suggesting that an analysis of curricula content is warranted in order to ascertain whether mental health practitioner training programs are making sufficient efforts to challenge conventional viewpoints about psychopharmacology.

The Psychological Clinical Science Accreditation System (PCSAS) has been recommended as a means to critically examine psychology curricula and determine whether or not programs are sufficiently science based (Jaffe, 2010; Julien et al., 2011). According to the organization’s website:

PCSAS is an independent, non-profit body incorporated in December 2007 to provide rigorous, objective, and empirically based accreditation of Ph.D. programs in psychological clinical science (the terms *psychological clinical*

*science* and *scientific clinical psychology* are used interchangeably). PCSAS was created to promote superior science-centered education and training in clinical psychology, to increase the quality and quantity of clinical scientists contributing to the advancement of public health, and to enhance the scientific knowledge base for mental and behavioral health care. (<http://www.pcsas.org/> )

In conversation with the researcher, the PCSAS’s executive Director stated that “The PCSAS Review Committee looks at the full curriculum of each applicant program and judges the scientific foundations of the courses being taught in the program by examining the scientific rigor reflected in the syllabi for the courses” (R. McFall, personal communication, December 5, 2014). In addition, McFall asserted:

I can say with confidence that no position is barred from legitimate criticism in a scientific investigation, whether it be questions of etiology or assessment or intervention . . . The issue is not whether a given program presents a particular perspective on this or any other issue, but whether the perspective is treated in a scholarly, empirically supported, and critical way that encourages the students to help advance the science. (R. McFall, personal communication, December 5, 2014)

In this regard, and considering the findings from this study, it is notable that the PCSAS’s primary sources of information are syllabi. This study found that the examination of required and recommended readings, are integral to establishing how content is being disseminated (i.e., the extent to which mainstream readings and course videos integrate critiques of popular hypotheses and whether additional readings by critics who challenge conventional perspectives are being included). It is also noteworthy that the “PCSAS accredits only doctoral training programs that grant Ph.D. degrees and are housed in non-profit, research-intensive universities” and not PsyD degrees (<http://www.pscas.org/>).



## Recommendations

If psychologist postdoctoral psychopharmacology training materials and curricula are not sufficiently integrating thorough critiques of both the DSM and theories for a neurobiological etiology of depression, this should be rectified. Further, if this information was excluded based upon the assumption that it is being addressed within graduate level psychology training, there needs to be empirical evidence to support this.

Additionally, this study's findings suggest that a number of mainstream psychopharmacology textbooks are not providing thorough and consistent critiques of the DSM and challenges to neurobiological hypotheses for the etiology of depression. Further, if psychologist prescriptive authority training programs elect to include critics' works as a means to compensate for this, the heterogeneity of the critics' perspectives must be considered—the complexity and breadth of the critical literature on psychopharmacology should not be overlooked.

Ideally, if the examined program's reading project is being used to integrate critiques of conventional perspectives, the assignment should include a variety of different "critical" readings. Additionally, requiring that students compare and contrast these viewpoints with those found within the mainstream textbooks would ostensibly foster a more critical examination of the extent literature and research. It is also recommended that the examined psychologist postdoctoral psychopharmacology program's video on the neurobiological underpinnings of affective disorders incorporate thorough critiques of the hypotheses being disseminated—including a deconstruction of the chemical imbalance hypothesis for depression that notes, as Stahl and others have asserted, that this hypothesis is indefensible based on current evidence.

This particular video ostensibly had some impact on students' communications with patients about the neurobiological etiology of depression—particularly since the rationale behind prescribing an antidepressant is commonly based upon these etiological hypotheses. This has important implications for informed consent and whether or not clinicians are attending to this responsibility in a thorough manner. In their article about the chemical imbalance hypothesis, France, Lysaker, and Robison (2007) asked “how much information should be provided to clients concerning the validity (or potential lack thereof) of chemical imbalance explanations of depression? Is it unethical to gloss over the complexities of this and related (i.e., antidepressant vs. placebo efficacy) issues?” (p. 418). Presumably, this statement also applies to other hypotheses about the etiology of depression and other forms of mental distress.

The study's integrated analysis also raises questions about program evaluations that focus solely on the examination of syllabi (e.g., the Psychological Clinical Science Accreditation System). While analyses of syllabi provide important information about the duration of classes, credits, assignments, course objectives, and the types of required and recommended training materials (i.e., books, articles, and videos), they do not provide any substantial details about the content of these readings and videos. Consequently, important information about the ways in which textbooks and articles are disseminating topics (i.e., with regard to thoroughness, and the integration of critiques and citations) might be missed.

Incorporating content analyses of textbooks into curricula research adds important information to these examinations and, in the case of psychologist postdoctoral psychopharmacology training, would ostensibly support professors in developing a larger

list of textbooks and articles that adequately critique conventional perspectives about psychopharmacy. While a definition or standard of what constitutes a balanced approach to textbook writing has yet to be established, the provision of citations within psychopharmacology textbooks is a feasible objective that is consistent with mainstream scientific standards. Moreover, it is a target that publishers, authors, and editors could easily uphold and adhere to. The requirement that neurobiological hypotheses of mental disorders be consistently and thoroughly critiqued would ostensibly garner considerable support within the academic community as well.

It also warrants mentioning here that the *American Psychiatric Publishing Textbook on Psychopharmacology* (2009) included a chapter on *Ethical Considerations in Psychopharmacological Treatment and Research* (not a required reading for the examined psychologist postdoctoral psychopharmacology training program) (Schatzberg & Nemeroff, 2009). This chapter reflects a willingness to examine issues surrounding public trust, research methodology, conflicts of interest, informed consent, the patient-doctor relationship, and implications of prescribing powerful medications. Next, the chapter cited works by a number of critics (i.e., Joanna Moncrieff, Daniel Carlat, Thomas Szasz, and Gardiner Harris), which reflects a consideration of non-conventional viewpoints about psychopharmacology. Consequently, the provision of an additional chapter focusing on controversial issues in psychopharmacology could be an effective strategy, among others, for increasing critical content within psychopharmacology textbooks.

Another recommendation is that psychologist postdoctoral psychopharmacology training materials incorporate readings from consumers/survivors/ex-patients. In addition

to academic publications, this might include stories and perspectives of c/s/x activists (e.g., from Madness Radio's, 2015, audio archives or through reading the Community Consortium Interviews, 2015, of various c/s/x activists like the late Judi Chamberlin and Larry Roberts). Next, these programs should consult with c/s/x activists about how best to educate prescribing psychologists about the c/s/x population and its movement. Here, instructors might reach out to c/s/x activists that teach classes in Mad History (i.e., David Reville from Ryerson University and Geoffrey Reaume from York University's Disability Studies programs).

Curriculum materials for introducing students to the c/s/x movement could also include an exploration of c/s/x history. For example, psychologist postdoctoral psychopharmacology programs could require students to examine the websites of MindFreedom ([www.MindFreedomInternational.org](http://www.MindFreedomInternational.org)), The National Empowerment Center ([www.power2u.org](http://www.power2u.org)) and the National Coalition for Mental Health Recovery ([www.ncmhr.org](http://www.ncmhr.org)). In addition, Pat Deegan's ([www.patdeegan.com/blog/categories/consumersurvivorexpatient-history](http://www.patdeegan.com/blog/categories/consumersurvivorexpatient-history)) and Pat Risser's ([www.patrisser.com/~PatRisser/index.html](http://www.patrisser.com/~PatRisser/index.html)) websites, the Psychiatric Survivor's Archives of Toronto ([www.psychiatricurvivorarchives.com/](http://www.psychiatricurvivorarchives.com/)), and Judi Chamberlin's (1990) article, *The Ex-patients ' Movement: Where We've Been and Where We Are Going*, address important aspects of c/s/x history that could be helpful in this regard.

The diversity of perspectives within the c/s/x movement requires that students be exposed to a variety of c/s/x activists' voices. Here, an edited volume like *Mad Matters*, where a number of different authors examine and discuss a spectrum of c/s/x issues from differing perspectives could be particularly useful (LeFrancois et al., 2013). For instance,

this book provides the reader with a variety of c/s/x viewpoints on issues including, but not limited to, the language used for conceptualizing mental distress, identity politics, experiences of women in asylums during the 19th century, and the diversity of the c/s/x community based upon differing “priorities, needs, identities, experiences, and strategies” (Diamond, as cited in LeFrancois et al., 2013, p. 67). Further, the book includes articles which explore the importance of celebrating the c/s/x movement’s successes, challenges to the concept of sanity, critiques of electroshock therapy, and civil rights within the context of mental health, the law, and public policy.

Information challenging the legitimacy of the c/s/x movement should also be integrated within psychologist postdoctoral psychopharmacology training materials. To access this information, students could visit the website for the Treatment Advocacy Center ([www.treatmentadvocacycenter.org](http://www.treatmentadvocacycenter.org)) in addition to reading articles by critics of the c/s/x movement like Edwin Fuller Torrey (1997), and Sally Satel (2000). In this regard, consideration should be given to the potential risks of abolishing forced hospitalization and forced drugging and, conceivably, about the systemic issues which curtail the analysis of safety to such limited options.

## **Conclusion**

This study was unique because it was the first to analyze the extent to which critiques of conventional perspectives about psychopharmacology (i.e., surrounding a neurobiological etiology of depression and the rigor of the DSM) were being critiqued within psychologist postdoctoral psychopharmacology training materials. The findings suggest that critical discourse about neurobiological hypotheses for the etiology of depression and challenges to the rigor of the DSM were not receiving sufficient attention

within the examined materials being used to train psychologists for prescriptive authority. It also appeared that the implications of these gaps in scientific evidence were not being considered within the context of treating marginalized groups like the c/s/x population whose voice was not prioritized within the examined syllabi, readings, and videos. This indicates that additional attention to critical discourse and the consideration of the c/s/x population is warranted when it comes to developing curricula to train psychologists for prescriptive privileges. These findings should be interpreted conservatively in light of the study's methodological limitations.

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## Appendix A

### Coding Framework for Neurobiologically Based Hypotheses for Etiology of Depression

## Content

### Operational Definition

The neurobiological etiology of depression refers to any brain based hypotheses or theories about the causes of depression.

### Inclusion Criterion

The sentence/paragraph is likely making a specific reference to a neurobiological hypothesis/theory for the etiology of depression if:

1) A neurobiological hypothesis/theory for the etiology of depression is a header (title) for a section.

*E.g., a paragraph or set of paragraphs headed by the phrase “The Monoamine Hypothesis of Depression.”*

2) References to studies appear to be clearly related to the text’s goals of elucidating a particular neurobiological hypothesis/theory of depression’s etiology.

*E.g., “Other studies suggest that neuropeptides impact brain chemistry and depressive symptomatology”, is an example of content elucidating the neuropeptide/neuroendocrine hypothesis for depression.*

3). When words and phrases including, but not limited to, “pathophysiology”, “pathogenesis”, “etiology”, “aetiology”, “influences”, “source”, “cause”, “mediate” “leads to”, “candidate mechanism”, “predict”, “molecular basis”, “implicate”, “role of”, “regulators of”, and “linked to”, are clearly used to describe a particular neurobiological hypothesis/theory for the etiology of depression.

*E.g., “The source of depressive symptomatology has also been linked to encephalins”, is an example of content which refers to the neuropeptide hypothesis of depression.*

4. When words or phrases including, but not limited to, “deficiency”, “inefficient”, “malfunctioning”, “abnormality”, “out of tune”, “problem”, “magnification” and “pathological”, are clearly used to describe a particular neurobiological hypothesis/theory of depression.

*E.g., “Historically, depression has been conceptualized as a deficiency involving neurotransmitters ...” refers to the neurotransmitter hypothesis of depression.*

5. When depression is clearly referred to via terms like “depression”, “mood disorder”, “affective disorders”, “unipolar depression” “dysthymia”, and “major depressive episode”, in conjunction with comments about the proposed neurobiological determinants of the disorder.

Also note that references to neurobiological hypotheses/theories for the etiology of bipolar disorder do not meet criteria for inclusion unless they specifically refer to depression’s etiology.

Next, references to antidepressant treatment and the respective mechanisms of action of antidepressants will not be considered as meeting the criteria for inclusion unless these discussions clearly reference a neurobiological etiology of depression. For example, a text might discuss a neurobiological hypothesis/theory for depression in the midst of mentioning research findings on antidepressants.

*E.g., “SSRIs have been shown to raise serotonin levels which are thought to be lower in depressed individuals”, uses evidence from studies on antidepressants to discuss the aminergic hypothesis of depression.*

Hypotheses/theories which are not clearly neurobiological in nature will not be considered as meeting criteria for content about a neurobiological hypothesis/theory for the etiology of depression (e.g., environmental factors like low omega-3 fatty acids).

Next, neurobiological illnesses which may include depressive symptomatology will not be counted as content. Thus, depression’s etiology discussed within the context of Parkinson’s, Alzheimer’s, Cushing Syndrome, and Huntington’s disease will not meet criteria for coding unless the content clearly and explicitly elucidates what the neurobiological source of the depression is.

### Breadth

The coder must read each of the pages selected for their analysis (i.e., start at the beginning of the first sentence even if it commences on the previous page, and end your reading at the completion of the last sentence even if it terminates on a subsequent page).

### Coding Categories

Once it has been established that the content refers to a neurobiological hypothesis/theory for the etiology of depression, it is time to choose which of the categories apply. Remember to look for clues surrounding which hypothesis or hypotheses is/are being discussed. For example, there may be a header or title introducing the section, and/or the hypothesis/theory may be explicitly identified within the paragraph (“e.g., the neurogenesis hypothesis...”).

In other cases the author(s) will include discussions about aspects of a particular hypothesis/theory which won’t be identified as hypotheses/theories in and of themselves. For example, irregularities with the HPA-Axis or glucocorticoid levels may be mentioned within a discussion about the neurogenesis hypothesis of depression. In this case, unless those factors are being clearly discussed as separate hypotheses - don’t document these mechanisms – simply code for the primary hypothesis/theory.

One of the challenges in coding for this topic is that the author’s do not always explicitly identify a hypothesis as such. For example, they may simply refer to Glutamate, Gaba, Neuropeptides, Neurotrophins, etc. as potential mechanisms involved in depression without explicitly referencing them as hypotheses or theories. Regardless, the coder

should still document these mechanisms under the appropriate coding category for neurobiological based hypotheses/theories for depression's etiology.

#### 1) Neurotransmitters

There have been a variety of different theories and nomenclature used to discuss neurotransmitter hypotheses/theories of depression. These include the aminergic, chemical imbalance, monoamine, catecholamine, and Adrenergic-Cholinergic Balance hypotheses. In other cases the text may simply refer to low levels of a particular neurotransmitter like serotonin, dopamine, or norepinephrine, or allude to "chemical" abnormalities or "chemical problems" which should also be coded as "neurotransmitters."

#### 2) Neurotransmitter Receptors

Neurotransmitter receptors have also been implicated in depression's etiology.

#### 3) Neurotrophins/Proteins (i.e., BDNF, P11, CREB, cAMP)

Neurotrophins are a family of proteins that facilitate the survival, development, and functioning of neurons. They belong to a class of growth factors that have the capacity to signal cell survival, differentiation, and growth. Growth factors such as neurotrophins, which are involved in promoting the survival of neurons are known as neurotrophic factors.

Neurotrophic factors are secreted by target tissue and prevent neurons from initiating programmed cell death (apoptosis). Neurotrophins also initiate the differentiation of progenitor cells, which results in the formation of neurons. The majority of neurons in the mammalian brain develop prenatally. However, parts of the adult brain retain the ability to grow new neurons from neural stem cells - a process known as neurogenesis.

Neurotrophins are chemicals that help to stimulate and control neurogenesis.

#### 4) Neuroendocrine/Neuropeptide (i.e., HPA-Axis, HPT-Axis, glucocorticoids, estrogen, CRF, CRH, Corticosteroid Receptor Hypothesis; Hormone Receptor Hypothesis; VGF, NPY, Tachykinins, Hypothalamic Peptides, Enkephalins).

The Neuroendocrine system consists of various glands that produce and secrete hormones including Neuropeptides, steroids, and neuroamines.

Neuropeptides are small protein-like molecules (peptides) used by neurons to communicate with each other. Neuropeptides modulate neuronal communication by acting on cell surface receptors.

#### 5) Inflammation – Immunological

Neurobiologically based inflammation and immunological processes are hypothesized to play a role in depression. Cytokines have been implicated in this process. Cytokines are a broad and loose category of small proteins that play an important role in cell signaling.

They act through receptors and are especially important in the immune system modulating the balance between humoral and cell-based immune responses in order to regulate the maturation, growth, and responsiveness of particular cell populations.

#### 6) Genetics

Research suggests that neurobiological abnormalities associated with depression are linked to genetics.

#### 7) Glutamate (i.e., NMDA; glutamate receptors)

Glutamate is an excitatory neurotransmitter located within the nervous system. Glutamate is stored in vesicles within chemical synapses. Nerve impulses facilitate glutamate's release from the pre-synaptic cell. Glutamate acts on ionotropic and metabotropic (G-protein coupled) receptors. Glutamate receptors like NMDA receptor or the AMPA receptor, bind glutamate.

Glutamate plays a role in synaptic plasticity whereby it facilitates long-term potentiation at glutamatergic synapses in the hippocampus, neocortex, and other parts of the brain.

In brain injury or disease, excess glutamate can accumulate outside cells. This process causes calcium ions to enter cells via NMDA receptor channels damaging neurons and eventual cell death (apoptosis) – a process called excitotoxicity. Excitotoxicity due to excessive glutamate release and impaired uptake occurs as part of the ischemic cascade and has been linked to the neurobiological etiology of depression.

#### 8) GABA

GABA acts at inhibitory synapses through binding to transmembrane receptors in the plasma membrane. It is involved in both pre- and postsynaptic neuronal processes. GABA helps to facilitate the opening of ion-channels to permit the flow of either negatively charged chloride ions into the cell or positively charged potassium ions out of the cell. This action leads to a negative change in the transmembrane potential, typically resulting in hyperpolarization.

GABA is a regulator of many CNS processes including neurogenesis. Some research suggests that GABAergic neurotransmission may be altered in mood disorders.

#### 9) Neurogenesis/Neurogenic Hypothesis

Research indicates that the brain can form new neurons in a process called neurogenesis, and that existing neurons can repair and remodel themselves. Research also suggests that the hippocampus is particularly vulnerable to stress/trauma, and that stress has the capacity to damage neurons in the hippocampus and the frontal cortex. Further, damage in these regions has been linked to increased amounts of glucocorticoids, abnormalities within the HPA-Axis and decreased levels of BDNF – all of which are associated with depression. Sometimes neurogenesis is described as a remodeling process (“synaptic plasticity”) that involves the shaping of new neurons and the loss of old ones. Other

discussions about the neurogenic hypothesis may highlight the degradation of the hippocampus resulting from insufficient amounts of Neurotrophins.

#### 10) Circadian Rhythm

A circadian rhythm is any biological process that displays an endogenous, entrainable, oscillation of about 24 hours. Disturbances in the circadian rhythm governing sleep are implicated in depression's etiology. Note that circadian rhythms may be referred to as "chronobiological aspects."

#### 11) Neuroanatomical/Circuits

Neuroanatomy has been implicated in the neurobiological etiology of depression. This includes particular regions of the brain (e.g., frontal cortex) as well as specific parts like the hippocampus or amygdale. Circuits refers to neural circuitry that some researchers suggest is dysfunctional in depression.

#### 12) Enzymes/Cofactors

Enzymes are highly selective macromolecular biological catalysts which can accelerate both the rate and specificity of metabolic chemical reactions. A number of enzymes are thought to play a role in depression's etiology including Monoamine-Oxidase and Dopamine Beta-Hydroxylase.

#### 13) Other

In cases where the coder comes across a hypothesis or theory for the neurobiological etiology of depression that has not been addressed within the researcher's list of hypotheses/theories, the coding category of "Other" can be selected. The coder should document their reasoning for choosing this category, within the "Comments" section of the spreadsheet and do their best to identify what hypothesis/theory they believe is being discussed.

#### Anomalies

If there are any cases in which content which does not fit within the existing framework including the "Other" category, make note of this within the "Comments" section.

#### Critiques

Critiques include any challenges to a particular neurobiologically based hypothesis/theory for the etiology of depression. This includes any comments about the limitations of associated research evidence.

E.g., "*In spite of these research findings implicating Neuropeptides in the etiology of depression the results are far from definitive...*" indicates that empirical support for the neuropeptides hypothesis of depression is limited.

#### Critics



The coder will also be required to document which, if any, of the researcher's listed critics are explicitly mentioned or cited within content about the neurobiological etiology of depression. A complete list of critic's works will be provided to help facilitate this.

Cases in which a listed critic was explicitly mentioned or cited within content that is outside of the 3 topic areas being studied (i.e., not about a neurobiological etiology for depression, the DSM, or the C/S/X movement), should also be documented.

#### Reference Pages

The coder must check every reference within selected reference pages to establish whether or not any of the researcher's listed critics were cited.

## Appendix B

### Coding Framework for DSM Content

### Operational Definition

The Diagnostic and Statistics Manual of Mental Disorders (DSM) is published by the American Psychiatric Association. There are five editions of the DSM, plus two formal revisions (I, II, III, III-R, IV, IV-TR, and V).

### Inclusion Criteria

In order to meet the criteria for one of the coding categories, the DSM must be explicitly mentioned and/or cited within the page being analyzed. Discussions about, or references to, the International Classification of Diseases (ICD) manual are not considered part of this topic unless these discussions clearly reference the DSM.

### Breadth

The coder must read each of the chapters or in some cases individual pages, selected for the analysis.

### Coding Categories

The coder will need to identify which of the DSM coding categories apply for each of the pages read. In some cases more than one coding category will apply. The coder only needs to record a coding category once on any particular page.

#### 1) Guidelines/Applications/General

Note that ANY explicit mention of the DSM meets the criteria for Guidelines/Applications/ General and must be coded as such. This category is also a catch-all for any discussions surrounding the DSM criterion for disorders, general comments about the structure of the manual, the manual's applications with other screening tests, and research involving the DSM.

#### 2) Development

This category is for any content which discusses the process of how the DSM was developed and revised. It is also the category by which ANY versions of the DSM that predate the DSM-IV (i.e., DSM I, II, III, III-R) should be identified. Similarly, this category should be used to document content about the DSM V. Note that comments about a "new condition" or "young disorder" are indicative of development when it comes to discussions about the manual and its disorders.

#### 3) Benefits/Merits

Any positive aspects of the DSM should be assigned this category. Examples of benefits/merits include but are not limited to assertions that the manual aids research, helps to facilitate the organization and classification of psychological symptomatology,

aids assessment, informs treatment, enables billing and insurance coverage, and facilitates program support.

#### 4) Criticisms/Limitations/Controversy

This category should be assigned to any content which critiques the DSM in any way. For example, any content which identifies problems with the manual's inter-rater reliability and construct validity. Content which discusses limitations of using the manual (e.g., that the DSM requires considerable training to utilize and there is considerable overlap between disorders) or controversy (e.g., disagreements about a category based approach), should be documented via this coding category.

#### 5) Other

In cases where the discussion about the DSM is not effectively captured by the four aforementioned coding categories the coder should identify this as "Other." The coder should then document their reasoning for choosing this category, within the "Comments" section of the spreadsheet.

#### Anomalies

In cases where the coder determines that the DSM is clearly being discussed, albeit without any explicit mention or citation of the manual itself, this must be documented within the comments section. The coder must still make sure to note what topics were discussed, whether or not the DSM was critiqued, whether inter-rater reliability and/or construct validity were addressed and whether any of the researcher's listed critics were cited on the topic.

Note that simple mentions of a diagnosis (e.g., depression, schizophrenia, bipolar) do not meet the requirements for documentation within the comments section (when the manual has not been explicitly mentioned. However, the discussion of any DSM disorder's symptomatology (e.g., sleep difficulties, appetite change, loss of interest for depression, or hallucinations, delusions, or more general mentions of positive/negative symptoms for schizophrenia, or comments about low mood or mania in bipolar, the mention of inattention for ADHD, and/or noting sleep problems in PTSD), are considered worthy of inclusion within the comments section for the DSM topic in cases where the manual was not explicitly cited or mentioned. References to animal models of depression do not meet criteria for content.

Any problems or concerns with the overall coding framework should be identified within the comments section of the excel spreadsheet.

#### Critiques

Critiques are documented in 3 ways.

- 1) If the DSM is explicitly mentioned, the coder should document any associated critiques for that page within the DSM spreadsheet via the coding category of “Criticisms/Limitations/ Controversy” under “DSM Topics.”

DSM Topics <i>Criticisms/Limitations/Controversy</i>	DSM Inter-Rater Reliability and/or Construct Validity Challenged	Critic(s) cited on Topic
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- 2) If the content about the DSM contains a critique that clearly challenges the DSM’s levels of inter-rater reliability and/or construct validity, this must be documented under “DSM Inter-Rater Reliability and/or Construct Validity Challenged” within the DSM spreadsheet (make sure to document which of these two measures of scientific rigor apply). See below.

DSM Topics	DSM Inter-Rater Reliability and/or Construct Validity Challenged	Critic(s) cited on Topic
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Inter-Rater Reliability pertains to the likelihood that different clinicians or coders will obtain similar results when diagnosing the same patient. Note that comments about a clinician’s choice of DSM diagnoses potentially differing based on their level of training would be considered as a challenge to the manual’s level of inter-rater reliability.

E.g., “There is considerable variation in the diagnoses different clinicians will assign to the same case”, is an example of a critique that is challenging the manual’s level of inter-rater reliability.

Construct Validity pertains to whether or not the construct – in this case any of the DSM diagnoses – actually measures what it claims, or purports, to be measuring.

E.g., “Some critics have pointed out that DSM diagnoses are not based on objective evidence”, is an example of a critique that is challenging the construct validity of the manual.

In addition, the coder must document whether or not any statistics on the reliability and validity of the DSM were identified. See below.

DSM Inter-Rater Reliability and/or Construct Validity Challenged	Critic(s) cited on Topic	Statistics on Reliability/Validity of DSM Presented Y/N
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- 3) If a critique occurs on a page with content about the DSM, albeit without any explicit mention or citation of the manual, this should be documented within the “Comments” section. See highlighted box from DSM spreadsheet below with *italicized* example.

Critic(s) cited on Topic	Statistics on Reliability/Validity of DSM Presented Y/N	Comments E.g., <i>The DSM's construct validity was critiqued. For example...</i>
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### Critics

The coder will also be required to document which, if any, of the researcher’s listed critics are explicitly mentioned or cited within content about the DSM. The critic(s) should be documented under the heading “Critic(s) cited on Topic.”

DSM Topics	DSM Inter-Rater Reliability and/or Construct Validity Challenged	Critic(s) cited on Topic
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A list of critic's works will help to facilitate this. In particular, the list of papers where the first author is not a critic will be helpful when the references list uses "et al." instead of including the names of all of the writers.

In cases where a listed critic is explicitly mentioned or cited within content that is outside of the 3 topic areas being studied (i.e., not about the a neurobiological etiology for depression, the DSM, or the C/S/X movement), this should be documented within the "REFERENCES" spreadsheet under "Critics cited OUTSIDE of 3 topic areas." The coder must document both the critic's(s') name(s) and the topic(s) being discussed. See highlighted boxes below from the DSM spreadsheet.

Critic(s) cited OUTSIDE of 3 topic areas	Topic(s)	Reference Page Critics
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For reference pages, the coder should document any of the researcher's listed critics under the REFERENCES spreadsheet via the "References Page Critics" box.

Critic(s) cited OUTSIDE of 3 topic areas	Topic(s)	Reference Page Critics
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In some cases a page will contain discussions about a topic and some portion of the chapter's reference section (i.e., the first page of the reference list also includes the conclusion of the article or chapter). In this case the discussion should first be coded for content under the usual three spreadsheets (ETXD, DSM, C/S/X), and then the REFERENCE spreadsheet to document nay of the researcher's listed critics.

## Appendix C

### Coding Framework for C/s/x Content



### Operational Definition

C/s/x content is defined as content about the Consumer/Survivor/Ex-Patient movement. It also refers to materials or comments by individuals who identify or are deemed to identify as C/s/x members.

### Inclusion Criterion

In order for content to meet the criteria for inclusion, the sentence/paragraph must explicitly mention the C/s/x movement, C/s/x organizations, and/or C/s/x identified members.

Inclusions of the terms “consumer(s)”, “survivor(s)”, or “ex-patient(s)” on their own do not meet the criteria for C/s/x content unless the statement in which they’re embedded is clearly referring to the C/s/x movement. Clues that these terms used in isolation are intended to describe the C/s/x movement are, 1) the c/s/x movement is mentioned in that particular chapter’s title or subtitle(s), 2) the particular section in question is identified as having c/s/x content within the book’s index, and 3) the reference to consumers, survivors, or ex-patients is clearly building upon prior mention(s) of the c/s/x movement outlined in previous sentences/paragraphs within the chapter.

C/s/x organizations include but are not limited to the following groups: MindFreedom, Support Coalition International, MadPride, Icarus Project, Hearing Voices Network, National Empowerment Center, National Disability Rights Network, National Mental Health Consumers, Self-Help Clearing House, Mental Health Consumer/Survivor Network, European Network of (Ex-) Users and Survivors of Psychiatry, Psych-Rights, The Community Consortium – Building Inclusive Communities for People with Psychiatric Disabilities, World Network of Users and Survivors of Psychiatry, the Citizens Commission for Human Rights, and the Center for Human Rights of Users and Survivors of Psychiatry. Given the size of the c/s/x movement it is possible that a text might mention an organization that was not included within this list. If in any doubt about whether a particular group is affiliated with the c/s/x movement, the organization should be called or emailed for confirmation.

Determining who identifies as c/s/x may take some work given the possibility that an explicit reference to an individual having formally identified themselves as such may not exist. Consequently, for the purposes of this study, any individual who has been diagnosed with a mental illness and, a) works or volunteers for a c/s/x organization and/or, b) attends c/s/x functions, and/or c) openly endorses c/s/x initiatives (e.g., increased collaboration between mental health consumers and mental health professionals) will be considered as having met the criteria for this designation.

Individuals who meet this criteria and may be referred to within the literature include, but are not limited to, Linda Andre, David Armes, George Badillo, Clifford W. Beers, Frank Blankenship, Monica Briggs, Jean Campbell, Ted Chabasinski, Judi Chamberlin, Oryx Cohen, Pat Deegan, Sasha Altman DuBrul, Erick Fabris, Alison Faulkner, Dr. Daniel Fischer, Leonard Roy Frank, Howard Geld, Jim Gottstein, Will Hall, Gail Hornstein, Joan Hughes, Rachel Klein, Peter Lehmann, Dr. Nathaniel Lehrman, Cathy Levin, Dr. Rufus May, Jacks Ashley McNamara, Kate Millet, John Modrow, Linda Morrison, David Oakes, Theresa Parkes, Dr. Geoffery Reaume, David Reville, Pat Risser, Joe and Susan Rogers, Jasna Russo, Melissa Sances, Judene Shelley, Ruth Ruth Stackhouse, Heather Johnson Straughn, Jan Wallcraft, David Webb, and Don Weitz.

In cases where the rater is uncertain about whether or not an individual being discussed and/or referenced in association with the c/s/x movement meets the criteria for someone who identifies as c/s/x, further investigation will be required. This can include internet searches, academic literature searches or, when feasible, contacting the individual themselves.

#### Breadth

The coder must read each of the examined chapters and/or specifically identified pages in their respective entirety.

#### Coding Categories

The coder will need to identify which of the following coding categories apply for each of the pages being examined. In some cases more than one coding category will apply. A coding category only needs to be recorded once on any particular page. In the c/s/x spreadsheet, this content should be documented under “c/s/x topics.”

C/s/x Topics	Criticisms About c/s/x  Critiqued? Y/N	Critic(s) cited on Topic
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1) C/s/x History

2) C/s/x Organizations

3) C/s/x Campaigns/Events/Activism

Some examples of content that meets the requirement for this category are the MindFreedom hunger strike, MadPride marches and concerts, and PsychRights court cases.

4) Challenges to the Legitimacy of C/s/x

This includes criticisms levied against the C/s/x movement. For example, conceptualizing the C/s/x movement as being anti-psychiatry or anti-medication, argument that the C/s/x movement undermines the constructive efforts of organizations like the American Psychiatric Association and NAMI, asserting that the C/s/x movement and its organizations might increase the likelihood of harm to self and others due to its purported anti-medication message, and arguing that C/s/x members and their organizations are uninformed.

#### 5) Personal Stories of C/s/x Members

This category refers to any individual who identifies as C/s/x or who a) works or volunteers for a C/s/x organization and/or, b) attends C/s/x functions, and/or c) openly endorses C/s/x initiatives (e.g., increased collaboration between mental health consumers and mental health professionals). This category does not include advocates or allies who support the movement and its initiatives but who do not self-identify as C/s/x themselves.

#### f) Other

The “Other” category should be selected for C/s/x content that cannot be conceptualized by one of the other coding categories.

#### Anomalies

Cases in which the coder encounter problems with the coding framework including when the “other” coding category is selected, should be identified within the “Comments” section of the Excel coding spreadsheet for c/s/x content.

Page #	What Topic?	Comments
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#### Critiques

The coder will be required to document whether or not any critiques were levied against the C/S/X movement under “C/s/x Topics” via the coding category of “Challenges to the Legitimacy of the C/s/x.” The coder must also identify whether or not any criticisms about the c/s/x were in turn critiqued by noting this under “Criticisms about the C/s/x Critiqued?”

C/s/x Topics	Criticisms About C/s/x Critiqued? Y/N	Critic(s) cited on Topic
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#### Critics

The coder must document whether or not any of the researcher's listed critics were explicitly mentioned or cited within content about the c/s/x, under the column headed "Critic(s) cited on Topic."

C/s/x Topics	Criticisms About C/s/x Critiqued? Y/N	Critic(s) cited on Topic
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In cases where a listed critic is explicitly mentioned or cited within content that is outside of the 3 topic areas being studied (i.e., not about a neurobiological etiology for depression, the DSM, or the c/s/x movement), this should be documented within the "REFERENCES" spreadsheet under "Critics cited OUTSIDE of 3 topic areas." Both the critic's(s) name(s) and the topic(s) being discussed should be documented.

Critic(s) cited OUTSIDE of 3 topic areas	Topic(s)	Reference Page Critics
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In some cases a page will contain discussions about a topic and some portion of the chapter's reference section (i.e., the first page of the reference list also includes the conclusion of the article or chapter). In this case the discussion should first be coded for content under the usual three spreadsheets (ETXD, DSM, C/S/X), and then the REFERENCE spreadsheet to document any of the researcher's listed critics.

Critic(s) cited OUTSIDE of 3 topic areas	Topic(s)	Reference Page Critics
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For cases in which a page contains discussions about a topic and some portion of the reference section for the chapter or article (i.e., the first page of the reference list also includes the conclusion of the article or chapter), the coder should document the discussion under the usual three spreadsheets (ETXD, DSM, C/S/X). The coder should then go to the REFERENCE spreadsheet to code for any of the researcher's listed critics identified within the reference list.

Work by C/S/X Individuals

The coder must record any cited works by c/s/x individuals. These works and their authors should be documented within the C/S/X spreadsheet under “Cited Works from C/s/x Identified Individuals (Title/Author).”

Critic(s) cited on Topic	Criticisms About C/S/X Critiqued? Y/N	Cited Works from C/S/X identified individuals (Title/Author)
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## Appendix D

Letter to Psychologist Postdoctoral Psychopharmacology Program Directors

Dear Dr. \_\_\_\_\_,

I'm a doctoral student at Antioch University's clinical psychology program in Seattle, WA, and a member of the American Psychological Association and Division 55. I am writing to request your support with my dissertation study which is researching the extent to which alternative perspectives about psychopharmacology are being integrated within current prescription privilege training programs for psychologists in the United States.

More specifically, I'm hoping to analyze psychologist psychopharmacology training materials for alternative perspectives surrounding a neurobiological etiology for depression, the DSM's rigor, the idea that, overall, antidepressants' risks outweigh their benefits, the legitimacy of c/s/x organizations, and the rigor of using inert placebos within antidepressant drug trials.

I am not looking to prove that these alternative perspectives represent the entire truth. Rather, I'm interested in determining the extent to which non-conventional ideas about the aforementioned topics are being integrated within existing psychologist postdoctoral psychopharmacology program curricula materials.

I'm requesting a complete set of syllabi from your most recent psychologist postdoctoral psychopharmacology training curriculum. I'm also hoping that you, as the program Director, will consider taking part in a telephone interview subsequent to the initial quantitative analysis of the data. The goal of the interview would be to shed additional light on the reasoning behind which alternative perspectives are being addressed or excluded.

To address concerns I'm anticipating with regard to the ownership and protection of your respective psychologist postdoctoral psychopharmacology curriculum materials, only three copies of your program's syllabi would be printed and all would be kept in locked filing cabinets when not being actively used by the researcher or other dissertation committee members. Next, subsequent to my analysis, two sets of the syllabi would be destroyed and the remaining one would be placed in a sealed envelope within a locked filing cabinet within the researcher's home office. The syllabi would then be destroyed seven years thereafter.

If you were to send these materials online, the downloaded syllabi would be erased after three complete paper copies were made. If the requested materials were sent by postal mail, only two additional copies of the original document would be made. The reason for requesting three copies is that the researcher lives in Port Moody, British Columbia, while two of his committee members are in Seattle, Washington and another in Hamilton, Ontario.

The name of your university and your identity as the psychologist postdoctoral psychopharmacology program's Director would also be kept confidential by the use of a nondescript numeric code. At no time would the university or the identity of a program's Director be revealed, either within the study's write-up or any other verbal or written discussions thereof.

I also appreciate that you might have other concerns and/or questions you would like addressed and I'm hoping that, if this is the case, you'll contact me or my committee chair, Dr. Alex Suarez P.M.S.P., at your soonest convenience. Many thanks for your consideration surrounding this request.

Sincerely,

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Appendix E  
Required Books

- Brunton, L., Chabner, B., & Knollman, B. (Eds.). (2010). *Goodman & Gilman's The Pharmacological Basis of Therapeutics* (12th ed.). New York: McGraw-Hill
- Gomella, L. G., Haist, S. A., & Adams, A. G. (Eds.). (2009). *Clinician's Pocket Drug Reference 2014*. New York: McGraw-Hill.
- Julien, R. M., Advokat, C. D., & Comaty, J. E. (2010). *A primer of drug action: A comprehensive guide to the actions, uses, and side effects of psychoactive drugs* (12th ed.). New York: Worth.
- McCance, K., Huether, S., Brashers, V.L., & Rote, N.S. (Eds.). (2002). *Pathophysiology: The biological basis for disease in adults and children*. (6th ed.). Maryland Heights, MO:Elsevier Mosby.
- McGrath, R. E., & Moore, B. A. (Eds.). (2010). *Pharmacotherapy for psychologists: Prescribing and collaborative roles*. Washington DC: APA Books.
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Appendix F

Semester Project Books

- Carlat, D. (2010). *Unhinged: The trouble with psychiatry—A doctor's revelations about a profession in crisis*. New York: Free Press.
- Conrad, P. (2007). *The medicalization of society: On the transformation of human conditions into treatable disorders*. Baltimore, MD: Johns Hopkins University Press.
- Dubovsky, A. N., & Dubovsky, S. L. (2007). *Psychotropic drug prescriber's survival guide: Ethical mental health treatment in the age of big pharma*. New York: Norton.
- Healy, D. (2006). *Let them eat Prozac: The unhealthy relationship between the pharmaceutical industry and depression*. New York: New York University Press.
- Moynihan, R., & Cassels, A. (2006). *Selling sickness: How the world's biggest pharmaceutical companies are turning us all into patients*. New York: Nation Books.
- Whitaker, R. (2010). *Anatomy of an epidemic: Magic bullets, psychiatric drugs, and the astonishing rise of mental illness in America*. New York: Crown.

## Appendix G

### Required Non-Textbook Readings

- Addington & Rapoport (2012). Annual research review: Impact of advances in genetics in understanding developmental psychopathology. *Journal of Child Psychology and Psychiatry*, 53, 510-518.
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- Global HealthEd. (2001b). Treatment plan. Unpublished Manuscript, p.1.
- Global HealthEd. (2001c). Treatment plan. Unpublished Manuscript, p.1.
- Global HealthEd. (2001d). Clinical note. Unpublished Manuscript, p.1.
- Global HealthEd. (2001e). Measurement Conversion Charts. Unpublished Manuscript, p.1.
- Global HealthEd. (2001f). Pharmaceutical abbreviations. Unpublished Manuscript, p.1-2
- Global HealthEd. (2001g). Sample prescription form. Unpublished Manuscript, p.1.
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## Appendix H

### Recommended Textbooks

- Bezchlibnyk-Butler, K. Z., & Virani, A. S. (Eds.) *Clinical handbook of psychotropic drugs for children and adolescents* (2nd rev. ed.). Seattle: Hogrefe and Huber.
- Blumenfeld, H. (2010). *Neuroanatomy through clinical cases* (2nd ed.). Sunderland MA: Sinauer Associates.
- Dipiro, J. T., Talbert, R. L., Yee, G. C., Wells, B. G., & Posey, L. M. (Eds.). (2011). *Pharmacotherapy: A pathophysiological approach*. (8th ed.). New York: McGraw-Hill.
- Hales, R. E., Shahrokh, N. C., Schatzberg, A. F., & Nemeroff, C. B. (2009). *Study guide to Clinical Psychopharmacology: A Companion to The American Psychiatric Textbook of Psychopharmacology*. Washington DC: American Psychiatric.
- Hendelman, W. J. (2005). *Atlas of functional neuroanatomy* (2nd ed.). Boca Raton, FL: CRC Press.
- Jarvis, C. (2011). *Physical examination & health assessment* (6th Ed.). St. Louis: Saunders.
- Jarvis, C. (2011). *Pocket Companion for Physical examination & health assessment* (6th Ed.). St. Louis: Saunders.
- Leahy, L. G., & Kohler, C. G. (2013). *Manual of clinical psychopharmacology for nurses*. Washington, DC: American Psychiatric Publishing.
- Van Putte, C., Regan, J., & Russo, A. (2010). *Seeley's Anatomy and Physiology* (9th ed.). New York: McGraw-Hill.
- Venes, D. (2013). *Taber's cyclopedic medical dictionary* (21st ed.). New York: F.A. Davis.

## Appendix I

### Recommended Non-Textbook Readings

*Carlat Psychiatry Report.* <http://www.thecarlatreport.com>

*Journal of Clinical Psychiatry.* <https://www.psychiatrist.com>

*Psychiatry Drug Alerts.* <http://www.alertpubs.com>

## Appendix J

## Ratio of Non-Semester Book Chapters Read to Total Chapters

Title	<u>Chapters Read/Total</u> <u>Chapters</u>	<u>Required</u>
Pathophysiology: The Biological Basis for Disease	23/47	Yes
A Primer of Drug Action	11/20	Yes
Neuroscience	7 + 8 pg.'s & Appendix, & Atlas/ 31, appendix & Atlas	Yes
Goodman & Gillman's: The Pharmacological Basis of Therapeutics	38 & 1 Appendix/67 & 2 Appendixes	Yes
Pharmacotherapy for Psychologists: Prescribing & Collaborative Roles	5/13	Yes
Stahl's Essential Psychopharmacology: Neuroscientific Basis and Practical Applications	8/14	Yes
Case Studies: Stahls' Essential Psychopharmacology	10/40	Yes
Essential Evidence Based Psychopharmacology	2/14	Yes
Clinical Handbook of Psychotropic Drugs	19/19	Yes
American Psychiatric Publishing Textbook of Psychopharmacology	17 + 6 pg's / 67	Yes



Manual of Clinical Psychopharmacology for Nurses	14/14	No
Pharmacotherapy: A Pathophysiological Approach	2/18	Yes
Seeley's Anatomy and Physiology	29/29	No
Atlas of Functional Anatomy	25/25	No
Neuroanatomy through Clinical Cases	19/19 & Epilogue	Yes
Study Guide to Psychopharmacology	18/67	Yes
Unhinged: The Trouble with Psychiatry	10/10	No
The Medicalization of Society	8/8	No
Selling Sickness	10/10	No
Let Them Eat Prozac	13/13	No
Psychotropic Drug Prescriber's Survival Guide	9/9	No
Anatomy of an Epidemic	16/16	No

## Appendix K

Ratio of Pages Read to Total Number of Pages within Non-Textbook Readings

Title	<u>Pages/Read</u>	<u>Required</u>
Following the Script: How Drug Reps Make Friends & Influence Doctors	5	Yes
Simple strategies to avoid medication errors	7	Yes
Psychotropic drug prescriptions by medical specialty	1	Yes
Avoiding medical errors: JCAHO documentation requirements	3	Yes
Do Not Use List	2	Yes
Annual research review: Impact of advances in genetics	9	Yes
Genetic testing for psychiatric disorders	5	Yes
County-level estimates of need for mental health professionals	8	Yes
County-level estimates of mental health professional supply	8	Yes
County-level estimates of mental health professional shortage	6	Yes
Mental illness surveillance among adults in the United States	30	Yes
What price prescribing?	6	Yes
Prescribing and primary care psychology	8	Yes
Practice guidelines regarding psychologists' involvement	15	Yes
Prescriptive authority for psychologists.	27	Yes
Malpractice claims experiences of psychologists	3	Yes
Helping doctors and patients make sense of health statistics	44	Yes
Antidepressant drug effects and depression severity	7	Yes
Relative effects of CBT and pharma in depression versus anxiety	8	Yes
What doctors don't know about the drugs they prescribe	1	Yes
Research shows that all too often, Americans are taking Rx	4	Yes
Psychotherapy and psychopharmacology	11	Yes
Bupropion - SR, Sertraline, Venlafaxine	12	Yes
Efficacy of psychotherapy & pharmacotherapy in treating depressive & anxiety disorders	12	Yes

Serotonin Syndrome	4	Yes
MAOIs	5	Yes
NEJM lab values	17	Yes
Warfarin	6	Yes
Harvard Medical School Algorithm	12	Yes
Texas Medication Algorithm Project	68	Yes
The choice of antipsychotic drugs for schizophrenia	3	Yes
Effectiveness of antipsychotic drugs in patients with chronic schizophrenia	15	Yes
Comparative efficacy and tolerability of 15 antipsychotic drugs in schizophrenia	12	Yes
Treatment of acute agitation in psychiatric disorders	9	Yes
Pharmacological Management of Acutely Agitated Pediatric Patients	10	Yes
DOD VA PTSD Guidelines	253	Yes
Dangerous Abbreviations List	1	Yes
Error Prone Abbreviations List	2	Yes
Jack Wiggins' RxP Fact Sheet	2	Yes
Ax Template 1	3	Yes
Tx Plan Template	1	Yes
Clinical Note Template	1	Yes
Clinical Note Template	1	Yes
Pharmaceutical Abbreviations	1	Yes
Rx Form Template	1	Yes
Drug References	1	Yes
Measurement Conversions	1	Yes
Medwatch Form	2	Yes
AACAP Guideline for Ax and TX of Children and Adolescents with ADHD	28	Yes
Title	<u>Pages/Read</u>	<u>Required</u>
AACAP Guideline for Ax and TX of Children and Adolescents with Bipolar Disorder	19	Yes

AACAP Guideline for Ax and TX of Children and Adolescents with Anxiety Disorders	17	Yes
AACAP Guideline for Ax and TX of Children and Adolescents with Depressive Disorders	24	Yes

Appendix L

Omitted Readings

Bezchlibnyk-Butler, K. Z., & Virani, A. S. (Eds.) *Clinical handbook of psychotropic drugs for children and adolescents* (2nd rev. ed.). Seattle: Hogrefe and Huber.

*Carlat Psychiatry Report*. <http://www.thecarlatreport.com>

Jarvis, C. (2011). *Physical examination & health assessment* (6th Ed.). St. Louis: Saunders

Jarvis, C. (2011). *Pocket Companion for Physical examination & health assessment* (6th Ed.). St. Louis: Saunders.

*Journal of Clinical Psychiatry*. <https://www.psychiatrist.com>

*Psychiatry Drug Alerts*. <http://www.alertpubs.com>

Venes, D. (2013). *Taber's cyclopedic medical dictionary* (21st ed.). New York: F.A. Davis.

Appendix M  
Coding Algorithm



for Neurobiological Hypotheses for the Etiology of Depression Content

IS there a heading/title identifying a particular hypothesis or theory for a neurobiological etiology of depression?

IF NO, still look for any content about the neurobiological etiology for depression and, if located, determine which of the coding categories applies. If there is no such content simply record this as “0” and then make sure to complete the analysis for DSM and c/s/x content before proceeding to the next page for analysis.

IF YES, document the hypothesis or theory using the coding categories provided. Also make sure to check the section for other theories that may be explicitly mentioned. Do not document all of the sub-hypotheses/theories for a hypothesis/theory when these other mechanisms are primarily being used to describe the main hypothesis/theory. For example, CREB, Neurotrophins (i.e., BDNF), and irregularities with the HPA-Axis or glucocorticoid levels may be mentioned within a discussion about the neurogenesis hypothesis for depression. In this cases, unless those factors are explicitly identified as another hypothesis/theory, do not document these mechanisms – simply code for the primary hypothesis/theory.

In some cases the authors(s) will not use a header or title to identify a particular hypothesis or theory. Further, they may simply refer to Glutamate, GABA, Neuropeptides, Neurotrophins, etc. as potential mechanisms involved in depression

without explicitly referencing them as hypotheses or theories. Regardless, the coder should still document these mechanisms as hypotheses using the coding categories.

IF UNSURE, look again to see whether or not there is a heading to the section which helps identify which hypothesis or theory is being discussed. Next, revisit the question, “is this content making a comment about a proposed neurobiological etiology for depression?” If the coder’s answer is affirmative then code accordingly. If the answer is negative, code accordingly. If still unsure, document this within the “Comments” section.

IF the coder discovers a hypothesis or theory for the etiology of depression which can’t be conceptualized via the existing list of hypotheses within the coding categories, select “other” and do your best to identify what the hypothesis is. The “Comments” section is another place where you can indentify any problems encountered with the overall coding framework.

**REMEMBER** to complete all of the columns for each of the spreadsheets.

## Appendix N

### Coding Algorithm for DSM Content

IS the DSM explicitly mentioned and/or cited?

IF NO, still look for any content about the DSM (i.e., about diagnosis and references to DSM symptomatology for a particular disorder – i.e., hallucinations for schizophrenia) that you feel is relevant to the topic. If such content is located, document this within the “Comments” section. Note that simple mentions of a disorders name (i.e., depression) does not warrant mentioning within the comments section. Also make sure to note whether or not the inter-rater reliability and/or construct validity of the manual was challenged, whether or not any critics were listed, and whether any statistics about the reliability and/or construct validity were included. If there is no content about the DSM then code this as “0” under “DSM Topics.”

IF YES, document this content via the appropriate coding categories provided.

Remember that EVERY mention of the DSM warrants coding for Guidelines/Applications/General. Also remember that a coding category must only be documented once for any given page even if there are a number of instances where it occurs within that page.

IF UNSURE: revisit the question, “is this content clearly related to a discussion about the DSM?” If the answer is affirmative then code accordingly. If the answer is negative code accordingly.

IF the existing coding categories do not capture a topic about the DSM, document this as “Other” within the “DSM Topics” column and explain this decision within the “Comments” section. Similarly use the “Comments” section to document cases in which the DSM is clearly being discussed but not explicitly mentioned.

**REMEMBER** to complete all of the columns for each of the spreadsheets.

## Appendix O

### Coding Algorithm for C/s/x Content

IS some aspect of the c/s/x movement clearly addressed?

IF NO, proceed to the next page of analysis.

IF YES, document this content via the appropriate Coding Categories provided.

IF UNSURE, revisit the question, “is this content clearly related to a discussion about the c/s/x movement?” If the answer is affirmative then code accordingly. If the answer is negative, code accordingly. If still unsure, document this clearly in the “Comments” section.

IF the coding framework does not capture a topic about the c/s/x within the page being analyzed, document this as “Other” within the “C/s/x Topics” column and identify what category is most appropriate. The “Comments” section is another place any problems encountered with the overall coding framework can be documented.

REMEMBER to complete all of the columns for each of the spreadsheets.

## Appendix P

### Coding Algorithm for References



ARE there any citations, mentions of a particular individual, or references?

IF NO, code applicable columns with a “0” and complete analysis for DSM and ETXD content before moving on to the next page of analysis.

IF YES, clarify whether or not the citation is from a critic and, if it is, whether or not the citation is being used to discuss one of the 3 topics being analyzed (i.e., ETXD, DSM,C/S/X). If it is, document the critic’s name under “Critic(s) Cited on Topic” within the appropriate spreadsheet for that topic. If the citation is for a critic but the topic they are being cited for lies outside of the three topic areas, document this within the REFERENCES spreadsheet under “Critics Cited OUTSIDE of 3 Topic Areas.”

IF UNSURE, revisit the question, “is this citation clearly related to a discussion about the C/S/X movement?” If the answer is affirmative then code accordingly. If the answer is negative, code accordingly. If still unsure, document this clearly in the “Comments” section.

IF the coding categories do not capture some aspect of the references effectively,  
document this within the “Comments” section of the appropriate spreadsheet.

REMEMBER to use the list of references for each of the listed critics including the list in which a non-critic was the first author.

REMEMBER to complete all of the columns for each of the spreadsheets.

Appendix Q  
Video Presentations

7910

Basic Cellular Concepts  
Cardiovascular System  
Respiratory System  
Renal/Genitourinary System  
Acid/Base Balance

7915

Hematology and Immunology  
Gastrointestinal and Hepatic Systems  
Endocrine System  
Musculoskeletal and Dermatologic Systems  
Reproductive System

7920

Neuroanatomy  
Neuron and Electrical Transmission  
Synaptic Transmission  
Classical Neurotransmitters in Synaptic Transmission  
Neurological Examination and Assessment  
Nervous System Pathology: Sensory

7925

Nervous System Pathology: Motor & Memory  
Basic Pharmacology  
Introduction to Psychotropics  
Other Transmitter Substances  
Peptide Transmission  
Neuroendocrinology

7930

General Principles of Pharmacology  
Drug Metabolism and Pharmacogenomics  
Drugs Affecting the Autonomic Nervous System  
Drugs Affecting the Cardiovascular and Respiratory Systems  
Drugs Affecting the Central Nervous System  
Anti-Infectives, Antibiotics, and Antivirals  
Drugs Affecting the Endocrine System  
Lifestyle Drugs  
Chronic Pain

7935

Prescriptions and Medical Orders  
Professional Relationships and Communications  
Professional Issues and Bioethics  
Issues in Prescriptive Practice  
Being an Informed Consumer of Drug Research  
Integrating Psychotherapy with Pharmacotherapy

7940

Biological Basis of Affective Disorders \*  
Pharmacotherapy of Depression \*  
Pharmacotherapy of Bipolar Disorders  
Treatment Guidelines and Considerations for Bipolar Disorder

7945

Neurobiology of Schizophrenia and Other Psychotic Disorders  
Neuropharmacology of Antipsychotics  
Antipsychotic Medications  
Evidence Based Management of Acute Agitation

7950

Overview of the Anxiety Disorders  
General Pharmacology of the Anxiety Disorders  
Special Populations  
Confounding Drugs and Disease States  
Generalized Anxiety and Panic  
Phobias and OCD  
PTSD

7955

Cognitive Disorders  
Sleep Disorders  
Substance-Related Disorders  
Personality Disorders  
Childhood/Adolescent Disorders

\* = Video Examine

## Appendix R

## Table

*NSP Books: Number of Pages with Content on a Neurobiological Etiology of Depression*

NSP Book Title	NT	NTR EC	NTR OP	N/C	GEN	GABA	GLUT	NE/NP	NGEN	IMM	CR	ENZ
P: T. Biological Basis for Disease	1 (1)	0	0	0	1 (1)	1 (1)	0	2 (1)	0	0	0	0
A Primer of Drug Action	4 (2)	3 (1)	2	0	0	1	3	2	8	0	1	0
Neuroscience	2	0	0	0	0	0	0	0	0	0	0	0
Goodman & Gilman's	2	0	0	0	1	0	0	2 (1)	0	0	0	0
Pharmacotherapy for Psychol.	0	0	0	0	0	0	0	0	0	0	0	0
Ess. Psychopharm: N.B & A.	18 (3)	7 (2)	3	6	13 (3)	0	1	6	5	0	4	1
Case Studies	0	0	1	2	0	0	0	0	1	0	0	0
Essential E. Based Psychopharm.	0	0	0	0	0	0	1	0	0	0	0	0
Clinical Handbook	0	0	0	0	0	0	0	0	0	0	0	0
American Psychiatric Publishing	9	3 (1)	0	0	4 (1)	0	3 (1)	11	4 (2)	0	1	0
Manual of Clinical Psychopharm*	5 (1)	0	0	2	0	0	0	1 (1)	0	0	0	0
Pharmacotherapy*	0	0	0	0	0	0	0	0	0	0	0	0
Seeley's Anatomy & Physiology*	0	0	0	0	0	0	0	0	0	0	0	0
Atlas of Fx Neuroanatomy*	0	0	0	0	0	0	0	0	0	0	0	0
Neuroanatomy*	3	0	1	1	0	0	0	2	0	1	0	0
Study Guide*	0	0	0	0	0	0	0	2	3	0	0	0

Critiques in Brackets = ( )

NT = neurotransmitter; NTR = neurotransmitter receptor; NTROP = neurotrophins; N/C = neuroanatomy/circuits;

GEN = genetics; GABA; GLUT = glutamate; NE/NP = neuroendocrine/neuropeptides; NGEN = neurogenesis;

IMM = immunological; CR = circadian rhythms; ENZ = enzymes.

Pathophysiology: The Biological Basis for Disease, 6th ed. (23 chapters)

A Primer of Drug Action, 12th ed. (11 chapters)

Essential Psychopharmacology: Neuroscientific Basis & Practical Applications, 4<sup>th</sup> ed. (9 chapters)

The American Psychiatric Publishing Textbook of Psychopharmacology (16 chapters)

Neurosciences, 5th ed. (7 chapters; 8 pages from 3 other chapters; Appendix and Atlas)

Goodman & Gilman's The Pharmacological Basis of Therapeutics, 12th ed. (38 chapters; Appendix 1)

Pharmacotherapy for Psychologists (5 chapters)

Case Studies: Essential Psychopharmacology (10 chapters)

Essential Evidence Based Psychopharmacology, 2<sup>nd</sup> ed. (3 chapters)

Clinical Handbook of Psychotropic Drugs 19<sup>th</sup> ed. (Entire Book Examined)

Manual of Clinical Psychopharmacology for Nurses (Entire Book Examined)\*

Pharmacotherapy: A Pathophysiological Approach, 8<sup>th</sup> ed. (2 chapters) \*

Seeley's Anatomy & Physiology, 9<sup>th</sup> ed. (Entire Book Examined)\*

Atlas of Functional Neuroanatomy, 2<sup>nd</sup> ed. (Entire Book Examined)\*

Neuroanatomy Through clinical cases, 2nd ed. (Entire Book Examined)\*

Study guide to Clinical Psychopharmacology: A Companion (16 chapters)\*

\* = Recommended



## Appendix S

## Table

*SP Books: Number of Pages in which Depressive Etiological Content  
was Found and Critiqued*

**Semester**

<b>Project Book Title</b>	<b>NT</b>	<b>NTRE</b>	<b>NTROP</b>	<b>N/C</b>	<b>GEN</b>	<b>GABA</b>	<b>GLUT</b>	<b>NE/NP</b>	<b>NGEN</b>	<b>IMM</b>	<b>CR</b>	<b>ENZ</b>
Unhinged	10(9)	0	1(1)	2(2)	2(2)	0	1(1)	1(1)	0	0	0	0
Medicalization of Society	0	0	0	0	1	0	0	0	0	0	0	0
Selling Sickness	5(5)	0	0	0	0	0	0	0	0	0	0	0
Let Them Eat Prozac	20(13)	0	0	0	1	0	0	0	0	0	0	0
Psychotropic RX Guide	1(1)	0	0	0	0	0	0	0	0	0	0	0

NT = neurotransmitter; NTRE = neurotransmitter receptor; NTROP = neurotrophins; N/C = neuroanatomy/circuits;

GEN = genetics; GABA; GLUT = glutamate; NE/NP = neuroendocrine/neuropeptides; NGEN = neurogenesis;

IMM = immunological; CR = circadian rhythms; ENZ = enzymes.

Appendix T  
Interview Questions

## **Curriculum**

I'm wondering if you'd be willing to begin by telling me some of the ways in which the curriculum has changed since the 2013/2014 iteration that I analyzed? In particular, I'm wondering if there have been any changes to the semester project which required students to read a book that critiqued some aspects of psychopharmacology? For example, have you added or removed any of the books on the reading list?

How did you choose the titles for the semester project readings?

## **Etiology of Depression**

In the *Biological Basis of Affective Disorders* video you sent me, the presenter discussed the chemical imbalance hypothesis of depression without explicitly challenging this hypothesis. With regard to the serotonin transporter gene implicated in depression, the presenter did note that these findings had been, "replicated by a few labs" and that, "there have also been some negative findings...so this is still theoretical." However, no details about the negative findings were addressed. Can you comment on these observations?

The presenter also noted that, "so these are theories – too many receptors, too little transmitters, but when you look at parts of the brain being hyper or hypoactive these are real findings in real patients." Nonetheless, there were no discussions about reductionism or the limitations of neuroimaging and animal models frequently used to support hypotheses about the etiology of depression. If my findings are correct, and students are not being sufficiently exposed to critical viewpoints about neurobiologically based theories for the etiology of depression within the Program's training video for the *Biological Basis of Affective Disorders*, where would they encounter such information in the curriculum?

My analysis indicated that neurobiologically based hypotheses for the etiology of depression were not consistently critiqued within the examined required and recommended readings. For example, in Stahl's *Essential Psychopharmacology: Neuroscientific Basis and Practical Applications*, four of the ten hypotheses presented were challenged. In a *Primer of Drug Action*, eight hypotheses were discussed but I could only find critiques for the monoamine hypothesis and neurotransmitter receptor hypothesis for depression. Next, other than Carlat's, *Unhinged*, the semester project books only presented and critiqued the monoamine and genetic hypotheses for depression. I'm wondering if you can comment on these findings?

## **DSM**

A number of the required readings provided challenges to the rigor of the DSM. However, these instances were rare within the non-semester project readings. For example, in the eighteen chapters of required readings within the *American Psychiatric Publishing Textbook*, I could only find three examples where the DSM's rigor was critiqued. In one case this textbook noted that the clinical features of bipolar disorder in children might differ from adolescents and adults. In another instance the textbook noted uncertainties about the best ways to classify GAD. In the *Manual of Clinical Psychopharmacology for Nurses*, there were two brief examples of a critique on two pages. In the eleven *Primer of Drug Action* chapter readings I could not find any cases where the DSM was challenged.

There were no examples of the DSM's kappa scores being discussed in any of the materials and, based on my analysis, the only substantive critiques about the manual occurred within four of the six semester project books. Was a more thorough critique of the DSM's rigor covered in any of the videos I didn't watch?

The *PEP Training DVD* talked about treating symptoms and not diagnoses, which is consistent with Stephen Stahl's approach. I'm wondering if there is a particular philosophical leaning in this regard within your program and I'm also wondering what your thoughts are about the RDoC (Research Domain Criteria) and its implications for training prescribing psychologists?

## **C/s/x**

My analysis found that information about the c/s/x movement was discussed with some detail in *Anatomy of an Epidemic* and it was briefly mentioned within David Healy and Daniel Carlat's books. However, I couldn't find any other mentions of the movement within the other curriculum readings I analyzed. Can you comment on the apparent dearth of information about the c/s/x population within your program's training materials?

## **Additional Questions**

How confident are you that psychologists are well versed in the literature which critiques conventional viewpoints about psychopharmacology, prior to their enrolling in a psychologist postdoctoral psychopharmacology training program?

Stephen Stahl's book, *Essentials of Psychopharmacology*, included some suggested readings but no formal citations. I'm wondering about your thoughts on this?

## Appendix U

Table *Non-Textbook Articles: Number of Pages with Neurobiological Etiology of Expression Content that Met Study's Coding Category Requirements.*









NSP Book Title	NT	NTR EC	NTR OP	N/C	GE N	GAB A	GL UT	NE/ NP	NGE N	IM M	C R	E N Z
Ax Template 2	0	0	0	0	0	0	0	0	0	0	0	0
Tx Plan Template	0	0	0	0	0	0	0	0	0	0	0	0
Clinical Note Template	0	0	0	0	0	0	0	0	0	0	0	0
Rx Form Template	0	0	0	0	0	0	0	0	0	0	0	0
Drug References	0	0	0	0	0	0	0	0	0	0	0	0
Measurement Conversion Sheet	0	0	0	0	0	0	0	0	0	0	0	0
Medwatch Form	0	0	0	0	0	0	0	0	0	0	0	0

NT = neurotransmitter; NTREC = neurotransmitter receptor; NTROP = neurotrophins; N/C = neuroanatomy/circuits;

GEN = genetics; GABA; GLUT = glutamate; NE/NP = neuroendocrine/neuropeptides; NGEN = neurogenesis;

IMM = immunological; CR = circadian rhythms; ENZ = enzymes.

## Appendix V

## Table

*NSP Books: Number of Pages for DSM Content that Met Study's Coding Category  
Requirements*

Table  
*NSP Books: Number of Pages for DSM Content that Met Study's Coding Category Requirements*

NSP Book Title	G/A/G	DEV	BEN	C/L/C	CV	IR
P: T. Biological Basis for Disease	4	0	0	2	0	0
A Primer of Drug Action	4	1	0	0	0	0
Neuroscience	1	0	0	0	0	0
Goodman & Gilman's	1	1	0	0	0	0
Pharmacotherapy for Psychol.	2	1	1	1	0	0
Essential Psychopharmacology: N.B & A.	12	0	0	3	2	0
Case Studies	2	0	0	1	1	0
Essential E. Based Psychopharmacology	0	0	0	0	0	0
Clinical Handbook	0	0	0	0	0	0
American Psychiatric Publishing Textbook	36	15	0	1	1	0
Manual of Clinical Psychopharmacology*	55	6	0	4	2	0
Pharmacotherapy*	3	2	2	1	0	0
Seeley's Anatomy & Physiology*	0	0	0	0	0	0
Atlas of Functional Neuroanatomy*	0	0	0	0	0	0
Neuroanatomy*	0	0	0	0	0	0
Study Guide*	1	1	0	0	0	0

G/A/G = general/applications/guidelines; DEV = development; BEN = benefits; C/L/C = criticisms/limitations/controversy's; CV = construct validity; IR = inter-rater

Pathophysiology: The Biological Basis for Disease, 6th ed. (23 chapters)

A Primer of Drug Action, 12th ed. (11 chapters)

Essential Psychopharmacology: Neuroscientific Basis & Practical Applications, 4<sup>th</sup> ed. (9 chapters)

The American Psychiatric Publishing Textbook of Psychopharmacology (16 chapters)

Neurosciences, 5th ed. (7 chapters; 8 pages from 3 other chapters; Appendix and Atlas)

Goodman & Gilman's The Pharmacological Basis of Therapeutics, 12th ed. (38 chapters; Appendix 1)

Pharmacotherapy for Psychologists (5 chapters)

Case Studies: Essential Psychopharmacology (10 chapters)

Essential Evidence Based Psychopharmacology, 2<sup>nd</sup> ed. (3 chapters)

Clinical Handbook of Psychotropic Drugs 19<sup>th</sup> ed. (Entire Book Examined)

Manual of Clinical Psychopharmacology for Nurses (Entire Book Examined)\*

Pharmacotherapy: A Pathophysiological Approach, 8<sup>th</sup> ed. (2 chapters) \*

Seeley's Anatomy & Physiology, 9<sup>th</sup> ed. (Entire Book Examined)\*

Atlas of Functional Neuroanatomy, 2<sup>nd</sup> ed. (Entire Book Examined)\*

Neuroanatomy Through clinical cases, 2nd ed. (Entire Book Examined)\*

Study guide to Clinical Psychopharmacology: A Companion (16 chapters)\*

\* = Recommended

## Appendix W

## Table

*SP Books: Number of Pages for DSM Content that  
Met Study's Coding Category Requirements*

Semester Project Book Title	G/A/G	DEV	BEN	C/L/ C	CV	REL
Unhinged	37	18	11	22	19	1
The Medicalization of Society	27	21	3	12	8	0
Selling Sickness	20	12	4	10	11*	0
Let them Eat Prozac	3	2	0	2	2	0
Psychotropic Drug Prescriber's Guide	1	0	0	0	0	0
Anatomy of an Epidemic	19	15	3*	7	6	1

G/A/G = general/applications/guidelines; DEV = development; BEN = benefits; C/L/C = criticisms/limitations/controversy's; CV = construct validity; IR = inter-rater

## Appendix X

## Table

*Non-Textbook Articles: Number of Pages for DSM Content that  
Met Study's Coding Category Requirements.*



NSP Book Title	G/A/G	DEV	BEN	C/L/C	CV	REL
Following the script:	0	0	0	0	0	0
Simple strategies to avoid med. errors	0	0	0	0	0	0
Psychotropic drug Rx's by medical spec.	0	0	0	0	0	0
Avoiding medical errors: JCAHO	0	0	0	0	0	0
Do not use list:	0	0	0	0	0	0
Annual Research Review	2	2	1	1	0	0
Genetic testing for psychiatric disorders:	0	0	0	0	0	0
County-level est. mental health prof. US	0	0	0	0	0	0
County-level est. mental health prof. US	0	0	0	0	0	0
County-level est. mental health prof. US	0	0	0	0	0	0
Mental illness surveillance among adults	3	1	1	2	1	1
What price prescribing?	0	0	0	0	0	0
Prescribing and primary care psychology:	0	0	0	0	0	0
Practice g-lines regarding psychologists'	0	0	0	0	0	0
Prescriptive authority for psychologists	0	0	0	0	0	0
Malpractice claims exp. of psychologists	0	0	0	0	0	0
Helping doctors and patients	0	0	0	0	0	0
AD rx effects & depression severity	0	0	0	0	0	0
Relative effects of CBT and pharma	0	0	0	0	0	0
What doctors don't know about	0	0	0	0	0	0
Research shows that all too often	0	0	0	0	0	0
Psychotherapy and psychopharmacology:	0	0	0	0	0	0
Bupropion - SR, Sertraline, Venlafaxine	1	0	0	0	0	0
The efficacy of psychotherapy and	1	0	0	0	0	0
Serotonin Syndrome Article	0	0	0	0	0	0
Monoamine Oxidase Inhibitors	0	0	0	0	0	0
NEJM lab values	0	0	0	0	0	0
Warfarin	0	0	0	0	0	0
Harvard Medical School Algorithm	0	0	0	0	0	0

Texas Medication Algorithm Project	2	0	0	0	0	0
The choice of antipsychotic drugs	0	0	0	0	0	0
Effectiveness of antipsychotic drugs	2	0	0	0	0	0
Comparative efficacy and tolerability	0	0	0	0	0	0
Treatment of acute agitation	0	0	0	0	0	0
Pharm. Mgmt of Acutely Agitated	0	0	0	0	0	0
DOD VA PTSD Guidelines	21	2	3	4	3	0
AACAP Guideline for ADHD	7	0	1	3	2	0
Dangerous Abbreviations List	0	0	0	0	0	0
Error Prone Abbreviations List	0	0	0	0	0	0
AACAP Guideline for Bipolar Disorder	11	2	0	10	9	1
NSP Book Title	G/A/G	DEV	BEN	C/L/C	CV	REL
AACAP Guideline for Anxiety Disorders	6	1	0	1	1	0
AACAP Guideline Depressive Disorders	6	0	0	1	0	0
Wiggins RxP Fact Sheet	0	0	0	0	0	0
Ax Template 1	0	0	0	0	0	0
Ax Template 2	0	0	0	0	0	0
Tx Plan Template	0	0	0	0	0	0
Clinical Note Template	0	0	0	0	0	0
Pharmaceutical Abbreviations	0	0	0	0	0	0
Rx Form Template	0	0	0	0	0	0
Drug References	0	0	0	0	0	0
Measurement Conversion Sheet	0	0	0	0	0	0
Medwatch Form	0	0	0	0	0	0