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An empirical and theoretical investigation of psychodynamic psychotherapy and neuroleptic medication for the treatment of schizophrenia

A Dissertation Project
Presented for the
Doctor of Philosophy
Degree
The University of Tennessee, Knoxville

Eric J. Peters
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DEDICATION

To Kelly,
for being you

To Bev, because you *know* in your experience and very being:
"Through the Thou a person becomes I"

To Jani, for providing a healing space

To Bertram Karon and Steven Copeland,
for living lives worthy of emulation

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ABSTRACT

Since the early 1950s, biopsychiatric conceptualizations have dominated empirical, theoretical and therapeutic efforts to understand the treatment of schizophrenia. The contemporary preeminence of biopsychiatric conceptualizations of schizophrenia have overshadowed other perspectives that might contribute fruitfully to our capacity to understand and aid individuals suffering with this devastating emotional disorder. The origin of modern biopsychiatric conceptualizations is deconstructed by illuminating the non-epistemic underpinnings of Emil Kraeplin's *dementia praecox* concept, which is the forerunner of the modern schizophrenia construct. Two widely held assumptions of the biomedical model, namely: 1) that schizophrenia is a degenerative, organic brain disease; and 2) that neuroleptic medications are the most effective and safest treatment of schizophrenia are empirically reviewed. Psychodynamic theory and therapy alternatives are also reviewed empirically and theoretically. The comparative effectiveness of Psychodynamic Psychotherapy-only (n = 9) and Medication-only (n = 12) was investigated using an experimental design (Karon & VandenBos, 1981). All patients were administered the Thematic Apperception Test (TAT) at pre- and post-treatment (20 months). Westen's (1995; Hilsenroth, Stein, & Pinsker, 2004) Social Cognition and Object Relations Scale (SCORS) was used to rate the pre- and post-treatment TAT narratives in order to assess changes in the cognitive and affective aspects of patients' object relations throughout treatment. Jacobson and Truax' (1991) Clinical Significance methodology was used to detect clinically significant change for each individual patient. Results show the SCORS is a reliable and valid instrument for use with a schizophrenia sample. Treatment outcome results suggest that patients receiving

psychodynamic psychotherapy exhibit clinically significant change in a variety of object relations domains when assessed at the group and individual levels. Comparative analyses indicated that Psychodynamic Psychotherapy-only patients outperformed Medication-only patients in regard to changes in a variety of object relations domains. Medication-only patients did not outperform Psychodynamic Psychotherapy-only patients in any domain of object relations. Significantly more Medication-only patients exhibited clinical regression compared to patients receiving psychodynamic psychotherapy. (Dr. Leonard Handler served as the chairperson of this dissertation committee.)

TABLE OF CONTENTS

I. INTRODUCTION 1
Section I: Emil Kraeplin, Degeneration Theory, and Philosophical Realism 13
Philosophical Realism and Kraeplin's Dementia Praecox Construct 17
Section II: The Empirical Validity of the Degenerative Disease Construct 19
Duration of Untreated Schizophrenia: Psychotoxicity and Clinical Outcome 20
Hope or Dread: Longitudinal Studies of Schizophrenia 24
Section III: The Effectiveness and Safety of Neuroleptic Medications 32
Effectiveness of the Typical Neuroleptics 32
Effectiveness Research Including Typical and Atypical Neuroleptics 40
Atypical Neuroleptics versus Typical Neuroleptics 42
How a Psychiatric Drug Comes to the Marketplace 43
The RCT: A Scientifically and Clinically Flawed Approach 45
CATIE: The Largest Naturalistic Study of Neuroleptics to Date 56
Side-Effects of Neuroleptic Medications 58
Section IV: The Effectiveness of Psychodynamic Psychotherapy of Schizophrenia 65
Research Investigating the Psychodynamic Psychotherapy of Schizophrenia 69
II. METHODS 75
Section V: Pre- to Post-Treatment Change in the Object Relations of Schizophrenic Patients 75
Clinical Participants 76
Therapists 80
Explanation of Psychotherapeutic Treatment 81
Measure 82
Explanation of Analyses 86
III. RESULTS 93
Convergent and Divergent Validity 93
Within-Group Analyses of Change 94
Traditional Analyses of Between-Groups Change Using 'Difference Scores' 95
'Recovered' and/or 'Positive Response' to Treatment 95
Group Differences for RCI Scores 101
IV. CONCLUSION 103
LIST OF REFERENCES 116
APPENDIX A 144
VITA 161

LIST OF TABLES

Table Page

1. Most Commonly Prescribed Neuroleptic Medications for the Treatment of Schizophrenia……145
2. Example of Non-Equivalent Dosing…….146
3. Interrater Reliability of the SCORS Variables……..147
4. SCORS Means of Outpatients Experiencing Mild to Moderate Distress as Reported in Peters et al. (2006)…….....148
5. Convergent and Divergent Validity of the SCORS with a Schizophrenic Sample…….149
6. Means, Standard Deviations, and Paired-Samples *T* Tests for Psychotherapy Group……...150
7. Means, Standard Deviations, and Paired-Samples *T* Tests for Medication Group…….……...151
8. Traditional Analyses of Between-Groups Change Using 'Difference Scores'……..152
9. Number of Individual Psychotherapy and Medication Patients Meeting Clinical Significance Criteria for 'Recovered', 'Positive Response', and Peters et al. (2006) for SCORS Complexity of Relationships (COM)…….153
10. Number of Individual Psychotherapy and Medication Patients Meeting Clinical Significance Criteria for 'Recovered', 'Positive Response', and Peters et al. (2006) for SCORS Affect Tone of Representations (AFF)…….……...154
11. Number of Individual Psychotherapy and Medication Patients Meeting Clinical Significance Criteria for 'Recovered', 'Positive Response', and Peters et al. (2006) for SCORS Emotional Investment in Relationships (EIR)……...…155
12. Number of Individual Psychotherapy and Medication Patients Meeting Clinical Significance Criteria for 'Recovered', 'Positive Response', and Peters et al. (2006) for SCORS Understanding of Social Causality (USC)…….....156
13. Number of Individual Psychotherapy and Medication Patients Meeting Clinical Significance Criteria for 'Recovered', 'Positive Response', and Peters et al. (2006) for SCORS Self-Esteem (SE)……...157
14. Number of Individual Psychotherapy and Medication Patients Meeting Clinical Significance Criteria for 'Recovered', 'Positive Response', and Peters et al. (2006) for SCORS Identity and Coherence of the Self (ICS)…….….158
15. Number of Individual Psychotherapy and Medication Patients Meeting Clinical Significance Criteria for 'Recovered' and 'Positive Response' for SCORS-C….....159

16. Differences in RCI between Psychotherapy and Medication Treatments……160

I. INTRODUCTION

> "All I want to do is to follow the advice given by Elihu, the son of Berachel of old, who said, 'I will speak that I may feel relief'; for there is a redemptive quality for an agitated mind in the spoken word."
>
> -J.B. Soloveitchik
> in *The Lonely Man of Faith*

Despite serious empirical and clinical questions regarding diagnostic reliability and validity (Blom, 2003; Boyle, 2002; Whitaker, 1992), the *Diagnostic and Statistical Manual-IV* (*DSM-IV*; American Psychiatric Association [APA], 1994) provides the most widely accepted diagnostic criteria for schizophrenia. Due to the wide availability of the *DSM-IV* only the most essential characteristics of the schizophrenia diagnosis will be discussed below.

The most characteristic criteria necessary to receive a diagnosis of schizophrenia are the presence of two (or more) of the following symptoms, each present for a significant portion of time during a one month period: 1) delusions; 2) hallucinations; 3) disorganized speech; 4) grossly disorganized or catatonic behavior; and/or 5) negative symptoms (Criterion A). It is important to note that only one Criterion A symptom is required if delusions are bizarre, hallucinations consist of a voice keeping a running commentary on the person's behavior or thoughts, or two or more voices are conversing with each other. Bizarre delusions are defined as anything that is entirely improbable. For example, a young man of twenty-one fears an alien with three heads is tracking his every movement because his patterns of gesticulation are the key to unlocking the mysteries of the universe. Importantly, a delusion that is fantastic but possible is *not* bizarre. For example, a schizophrenic man of thirty-five fears the FBI is spying on his family. This is unrealistic but nonetheless possible since the FBI could spy on someone with the

appropriate motivation, whereas three headed gumshoe aliens are not in the realm of possibility.

Characteristic symptoms fall into two broad categories: positive and negative. Simply, positive symptoms appear to reflect an excess or distortion of normal functions, whereas negative symptoms appear to reflect a diminution or loss of normal functions. The positive symptoms (Criteria A1-4) include distortions in thought content (delusions), perception (hallucinations), language and thought processes (disorganized speech) and grossly disorganized or catatonic behavior. Negative symptoms (Criterion A5) include restriction in the range and intensity of emotional expression (flattening), diminishment of fluency and productivity of thought and speech (alogia), and greatly decreased degree of goal-directed behavior (avolition).

Concordant with these primary symptoms are distinct and pervasive social/occupational dysfunction (Criterion B). Continuous signs of the disturbance must persist for at least six months which must include at least one month of symptoms that meet Criterion A (Criterion C). The remaining criteria include: exclusion of schizoaffective disorder and/or mood disorder (Criterion D), exclusion of a substance abuse or general medical condition (Criterion E), and if there is a history of a pervasive developmental disorder (e.g., autism) a diagnosis of schizophrenia is made only if prominent delusions or hallucinations are also present for at least one month (Criterion F). *DSM-IV* recognizes 7 subtypes of schizophrenia (Paranoid, Disorganized, Catatonic, Undifferentiated, Residual, Schizophreniform, and Schizoaffective; see APA, 1994, pp. 286-295).

Demographically, in the United States (U.S.), the onset of psychosis commonly occurs between the ages 17 to 27 for males and 20 to 37 for females. In the U.S., race does not seem to impact the distribution of schizophrenia. Schizophrenia affects 1-1.5% of the U.S. population and this percentage is consistent worldwide (Silverstein, Spaulding, & Menditto, 2006). Consistently, schizophrenia is among the top 10 leading causes of disability in adults both in the U.S. and worldwide, therefore representing a disorder of significant fiscal importance. In the U.S., nearly 80% of persons with schizophrenia are unemployed and many of the remaining 20% are significantly under-employed indicating overall a crippling social and vocational condition. In the U.S. up to 13% of people with schizophrenia commit suicide.

Of particular relevance to this study, almost 100% of the people diagnosed with schizophrenia in the United States have been or will be treated with psychotropic drugs (Jackson, 2005). A very small percentage of people suffering from this mental illness receive psychotherapy and an even smaller percentage will be offered psychodynamic psychotherapy, despite evidence that a variety of forms of psychotherapy are effective for psychotic disorders such as schizophrenia (Jackson, 2005; Karon, 1989; Whitaker, 1992).

An honest review of the empirical and clinical literature is imperative so that psychiatry and psychology can develop the most effective treatments for people suffering from schizophrenia. Such an exploration might inevitably lead to a major transformation whereby the mental health field can begin speaking about catharsis and recovery, as opposed to mere management and containment of schizophrenia. The latter pessimism is a reflection of the non-empirically supported notion that schizophrenia is solely an irreversible, organic, degenerative brain disease (Harding & Zahniser, 1994; Harman,

2003, Siebert, 1999). This unfounded assumption drives medication-only treatments despite the fact that there are no replicable studies to validate this widely held idea. In fact, as will be shown, there are a multitude of studies investigating the impact of untreated schizophrenia, as well as longitudinal outcome studies refuting the degenerative assumption.

Considering the monolithic medication approach to schizophrenia one would suspect that ample evidence exists regarding the physiological etiology of this alleged brain disease. However, laboratory findings diagnostic of schizophrenia are inconsistent and, more importantly, confounded by the significant physiological side-effects of an individual's past and present use of neuroleptic medications (APA, 1994; Harding & Zahniser , 1994; Harman, 2003; Jackson, 2005; Sullivan, Owen, O'Donovan, & Freedman, 2006; Warner, 2004). Thus, the idea that schizophrenia is a purely biologically-derived, degenerative brain disease is "totally unfounded and unsupported by scientific evidence…Rather the basis of the appellation 'brain disease' is based on *theories* of possible etiologies of those diagnosed with schizophrenia." (Harman, 2003, p. 240). The APA-sponsored *DSM-IV* honestly writes, "…laboratory findings may also be noted as a complication either of schizophrenia or of its treatment" (APA, 1994, p. 306). In fact, the side-effects of neuroleptic treatments are so hazardous that the *DSM-IV* has a diagnostic category known as "Neuroleptic induced disorders" (APA; pp. 735-805). As the *DSM-IV* acknowledges, an additional problem is that there are no replicable studies identifying the biological correlates of schizophrenia using drug virgins (APA, 1994). 'Drug virgins' are defined as individuals free of the damaging and confounding side-effects of antipsychotic medications. When drug virgin patients are investigated, the

results indicate that medications rather than the illness itself are generally responsible for any brain differences between schizophrenics and normals (e.g., Gur et al., 1998). The overt contradiction between what is known about schizophrenia and how it is treated should not be overlooked here. Although the APA-published *DSM-IV* admits that there are no studies that document the biological underpinnings of schizophrenia, it is this very same body that advocates medication as the primary and, in general, sole treatment approach.

Although some degree of genetic effect is likely present in the development of schizophrenia, the genetic contribution is undoubtedly non-linear and far more limited than originally suspected. Harvard geneticist Richard Lewontin (2000) comments on the neurogenetic reductionism fueling the assumption that genetic variation can explain all forms of human illness, he writes,

> The search for genetic variations is a major preoccupation of medical research [but]…it is bad biology… There exists, and has existed for a long time, a large body of evidence that demonstrates that the ontogeny of an organism is the consequence of a unique interaction between the gene it carries, *the temporal sequence of external environments through which it passes during life*, and random molecular interactions within individual cells. It is these interactions that must be incorporated into any proper account of how an organism is formed (pp. 17-18; my emphasis).

Indeed, what etiological evidence does exist point to poly-genetic factors of low to moderate effect in a dose-response, non-linear relationship with a variety of high-risk environmental factors such as deviant communication between children and caregivers

(Wynne & Singer, 1963), disturbed interactions within a family (Alanen, 1980, 1997; Tienari et al., 1985), childhood trauma (Read, Perry, Moskowitz, & Connolly, 2001; Read & Ross, 2003), and prenatal and perinatal factors (Gilmore & Murray, 2006). Thus, assumptions regarding the linear biological or genetic correlates of schizophrenia are, at least for now, empirically and theoretically untenable. Koehler (2006) more concisely describes the complex thinking needed if we are to develop a more sophisticated view of schizophrenia development and treatment,

> …reductionism in psychiatry is what it has always been, a costly and erroneous shortcut to the comprehensive understanding and treatment of complex mental disorders which arise from complex non-linear interactions between genes, brain, mind (which included multiple factors at the level of the individual and the family), as well as the socio-cultural surround…(p. 3).

By taking seriously the disconnect between how little is known about the etiology of schizophrenia and the rigid certainty with which medical treatments are offered, it is the stated intention of this dissertation project to arrive at an empirically and theoretically sober understanding of current psychotherapeutic and psychopharmacological treatment practices. This will be accomplished by investigating five interrelated sections.

Section I: *Emil Kraeplin, Degeneration Theory, and Philosophical Realism.* Since neuroleptic treatments overwhelmingly dominate the modern treatment approach for schizophrenia, it is helpful to understand the epistemology of the schizophrenia construct that continues to impact biological conceptualizations and treatment decisions. This section will focus on two of the non-epistemic philosophical underpinnings of the

schizophrenia construct originally developed by the founding father of modern psychiatry Emil Kraeplin: degeneration theory and philosophical realism.

Section II: *The Empirical Validity of the Degenerative Disease Construct*. Philosophical deconstruction of a prevailing construct does not invalidate its core assumptions. That is, simply because modern biopsychiatric conceptions of schizophrenia are substantially influenced by a multitude of non-epistemic factors does not necessarily mean that the illness is not biologically-based and degenerative. That being said, if the biopsychiatric notion that schizophrenia is a chronic degenerative brain disease resulting in a psychotoxic impact on the brain is accurate, then naturally most if not all cases of schizophrenia (treated and untreated) would ultimately leave a person significantly incapacitated *ad infinitum*. After all, biological medication treatments are only designed to slow this allegedly inevitable process and mask rather than cure the most striking features of the illness (i.e., positive symptoms such as hallucinations). To determine the validity of the degenerative/psychotoxic assumptions of biopsychiatry, I will review two interrelated empirical questions: 1) does the duration of untreated schizophrenia predict greater severity of illness, poorer overall outcome, and psychotoxic brain damage; and 2) are there longitudinal studies that report any degree of significant improvement or recovery for persons diagnosed with schizophrenia?

Section III: *The Effectiveness and Safety of Neuroleptic Medications.* For the reason that neuroleptic medications are used as the frontline - and usually only - treatment of schizophrenia (Breggin, 1991; Karon, 1989; Whitaker, 2002, 2005), it is imperative to summarize the empirical literature that has investigated the effectiveness and safety of these medications. The effectiveness of neurolepetc medications will be

evaluated on four fronts by: 1) presenting data regarding the effectiveness of older (typical) and newer (atypical) neuroleptic medications (Table 1 lists all of the medications still commonly used to treat schizophrenia; Appendix A[1]); 2) presenting a critique of the methodological limitations inherent in the Food and Drug Administration (FDA) and non-FDA randomized control medication trials (RCT) specific to schizophrenia research[2]; 3) summarizing the most recent, large-scale, naturalistic study investigating the comparative effectiveness and safety of the newer versus older neuroleptic drugs; and 4) presenting the variety of side-effects resulting from exposure to typical and atypical neuroleptic medications.

Section IV: *The Effectiveness of Psychodynamic Psychotherapy of Schizophrenia*. This section will explore the empirical effectiveness of the psychodynamic treatment of schizophrenia. There is a growing body of empirical and theoretical literature of effective *non*-psychodynamic approaches to schizophrenia (e.g., cognitive-behavioral therapy) that will not be addressed here. Focus on psychodynamic treatment will be primary because this is the modality used by the therapists that treated the patients in the empirical component of this dissertation project.

Section V: *Pre- to Post-Treatment Change in the Object Relations of Schizophrenic Patients*. Data for this project were generously provided by the Michigan State Psychotherapy Project archives (Karon & Vandenbos, 1981). The empirical section of this dissertation project applies a modern measure of object relations to pre- and post-treatment Thematic Apperception Test (TAT; Murray, 1943) narratives of schizophrenic

[1] All tables are located in Appendix A
[2] The critique presented below is not intended to be a general critique of RCT methodology. Rather, it is a critique specific to the methodology of schizophrenia drug research.

patients divided into two groups: 1) individual psychodynamic psychotherapy without medication (Psychotherapy; $n = 9$); and 2) routine medication-only (typical neuroleptics only) treatment (Medication; $n = 12$) to determine clinically significant treatment effects within- and between-groups. That is, it is the primary purpose of this study to investigate two particular research questions: 1) can individual psychodynamic psychotherapy for schizophrenia - *without any medication* - result in positive outcome in terms of object relations; and 2) what is the comparative effectiveness of medication-only versus individual psychodynamic psychotherapy-only treatment of schizophrenia?

The experimental design utilized in the original study conducted by Karon and Vandenbos (1981) called for the selection of schizophrenic patients to be assigned on the basis of a random number table to one of three treatments: 1) psychotherapy without medication (Psychotherapy-only A); 2) psychotherapy with adjunctive medication (Mixed B); and 3) routine hospital treatment consisting primarily of phenothiazines (i.e., typical neuroleptics; Medication-only C). The adjunctive medication provided to individuals of the Mixed Group (B) was discontinued after the first few weeks of the study. Overall, 36 patients were evenly distributed among the three treatment groups. Due to incomplete data for the Mixed group (B) this cohort has been removed from the current study. Although this is an unfortunate loss of potentially useful and interesting data, the primary and unique aspect of the current experiment is the assessment of a completely non-medicated group. That is, while there are studies exploring mixed treatment approaches (see Gottdiemer & Haslam, 2002) there are, as far as could be determined, no published U.S.-based studies in the last 25 years that utilize a fully non-medicated group.

Karon and Vandenbos' (1981) administered four assessment instruments measuring patients' thought disorder and two personality projective instruments at pre- and post-treatment to all patients. Number of days spent in the hospital and a blindly rated clinical status interview were also used as measures of outcome. At the conclusion of their 20-month investigation researchers found that patients receiving psychotherapy (Groups A & B) significantly outperformed individuals receiving medication-only (Group C) on three of the four instruments assessing patients' thought disorder, on the clinical status interview, and number of days spent in the hospital due to mental dysfunction. On the fourth measure of thought disorder differences between the psychotherapy and medication-only patients nearly reached significance ($p = .07$), again favoring the psychotherapy patients. Individuals of the medication-only group did *not* fare better in any area assessed.

One of the two measures for which there was no significant finding was the TAT. During the time period Karon and Vandenbos collected and reported their findings there were no reliable and valid narrative measures to adequately assess TAT protocols. In lieu of a reliable and valid measure Karon and Vandenbos applied a global score to pre- and post-treatment TAT protocols to assess general health-sickness in a manner akin to the three Axis V scales described in the *DSM-IV* (APA, 1994). As a result, they were unable to fully tap the information inherent in the pre- and post-treatment TAT protocols. This study corrects this original deficiency by using Westen's reliable and valid Social Cognition and Object Relations Scale (SCORS; Westen, 1995; Hilsenroth, Stein, & Pinsker, 2004) to score the original pre- and post-treatment TAT protocols for both groups.

The SCORS was used to measure the object relations and social cognitive functioning of patients from their pre- and post-treatment TAT protocols. Westen and Gabbard (1999) defined object relations broadly as a person's enduring patterns of interpersonal functioning in intimate relationships, beginning - but not concluding - with the mother/infant dyad. Therefore, object relations are the interpretation and mental internalization of relationships between the self and other people or objects (i.e., places, things, constructs). As Summers (1994) states, "[T]he structure of the self is formed from the internalization of early attachment relationships and is based on the symbolic meaning the child gives to its early object ties. In this sense, these early object relations not only endure, but define the sense of self and influence later relationships with others" (p. 350). As will be shown throughout this dissertation project, schizophrenia is far more than a biochemical illness defined by disturbed visual and auditory perceptual processes. Rather, it is a mental disorder driven by core disturbances in a person's capacity to relate to his or her self and others; and, even the most bizarre of the psychotic hallucinations and delusions, as well as massive social isolation common to schizophrenic patients, are best understood in the framework of a grave interpersonal disturbance. As such, measuring an individual's object relations pre- and post-treatment is essential to assessing the degree and quality of therapeutic relief provided by any given treatment.

The SCORS is a narrative-based object relations measure designed to assess a variety of dynamic personality features beyond the overt symptomatic presentation of the patient (see Methods below). As far as can be discerned from the extant literature, the current investigation was the first to apply the SCORS to a strictly schizophrenic sample, thus providing important reliability and validity information for a psychotic population.

Assessing observer-rated object relations adds incrementally to the overwhelming majority of the schizophrenia outcome literature that are generally limited to assessment of overt psychiatric symptoms and/or drug tolerability (e.g., Lieberman et al., 2005). Furthermore, the current analysis is unique to the extant literature in that it uses an experimental design that includes a fully non-medicated group. Such studies are relatively rare and generally considered irresponsible "malpractice" (Karon, 2003, p. 96) due to the non-empirically supported assumption that medication treatments are the only viable treatment for schizophrenia (Bola, 2006). In fact, despite considerable counter-evidence to be provided below, the schizophrenia Patient Outcome Research Team (PORT; Lehman & Steinwachs, 1998) referred to psychodynamic psychotherapy as "harmful" for patients suffering with schizophrenia (p. 8). The PORT is regarded as one of the authoritative voices in the treatment of schizophrenia and its conclusions are accepted as empirical fact despite its evident distortion of the psychodynamic treatment literature (see for a critique of the PORT Gottdiener & Haslam, 2003; Silver, 2003; ver eecke, 2003). Considering the PORT study's comments neglectful of available empirical evidence, hypotheses for the current study include: 1) The SCORS will exhibit satisfactory reliability and validity for use with a strictly schizophrenia sample; 2) Psychotherapy-only patients will *not* clinically regress, rather, they will exhibit clinically reliable improvement in their object relations; and 3) the object relations of the Psychotherapy-only patients will exhibit significantly greater clinically reliable change compared to the Medication-only patients due to psychodynamic psychotherapy's explicit emphasis on past and present interpersonal functioning.

Section I: Emil Kraeplin, Degeneration Theory, and Philosophical Realism

Emil Kraeplin, born in 1856, is considered the father of modern descriptive psychiatry and founder of the '*dementia praecox*' concept that was later changed to what we now term 'schizophrenia' (Bleuler, 1911). Kraeplin's professional life was committed to the classification of mental phenomena. His nosological thinking focused almost exclusively on the relationship between manifest symptoms and the developmental course and outcome of mental disorders. Kraeplin's ideas are still highly influential today (Gottesman, 1999; Harman, 2003).

Kraeplin required an overarching organizing principle with which to ground his observations, a principle that would provide a classificatory framework useful for categorizing the complexity of observed mental phenomena. He had little to draw on from previous research since psychiatry itself had only existed as a medical profession since the 1850s (Gottesman, 1999). In fact, the first three editions of his masterwork, *Compendium der Psychiatrie*, made only incremental contributions to the field of descriptive psychiatry and they were received indifferently by the psychiatric establishment of the time. However, Kraeplin's fourth edition published in 1893 made the first imprint of what would become a lasting mark on modern psychiatry.

In this fourth edition, Kraeplin operationalized a unique organizing principle to ground his observations of a wide array of mental phenomena: the syndrome-course unit Blom (2003). Adoption of this umbrella notion allowed Kraeplin to distinguish symptom clusters with a relatively good outcome from those with a poor outcome. As such, he introduced *outcome* as a diagnostic criterion. As will be seen, this move certainly had practical advantages for his capacity to organize ever-greater numbers of mental

phenomena, but was void of empirical backing and beholden to a variety of non-epistemic assumptions that have profoundly shaped the modern biopsychiatric conception of schizophrenia.

The term '*dementia praecox*' first appeared in the fourth edition of Kraeplin's *Compendium* in 1893. This publication is considered the birth of the modern schizophrenia concept. Using the notion of the syndrome-course unit, Kraeplin categorized *dementia praecox* under the heading, 'mental degenerative processes', thereby suggesting that its outcome was inherently poor. On what basis did Kraeplin make this classification? This is a question often left unanswered despite its monumental significance for contemporary research and treatment strategies. In his postmodern deconstruction of schizophrenia, Blom (2003) attempts to answer this question by assessing the role of degeneration theory in Kraeplin's thought, he writes,

> Degeneration [theory] referred to a supposed hereditary predisposition to all kinds of misery, such as physical and mental disease, addiction, moral decay, sexual aberrations, and criminal behavior. In Kraeplin's view, mental disorders originated from some inherited degenerative trait or from an external cause - or possibly from both (p. 55).

In Kraeplin's writings, "external cause" was meant to capture the effects of brain trauma, epidemics, pre-natal trauma, etc. That is, they were not meant to reflect the vicissitudes of psychosocial life, a factor Kraeplin seemed to deem generally irrelevant. As is evident in Kraeplin's primary writings, he organized almost all mental phenomena based on 19th century degeneration theory though he never explicitly stated this as such. Rather, he

cited the more scientific sounding 'syndrome-course unit' as his primary organizing principle.

In the 19th century, degeneration was deemed the polar opposite of successful social and physical evolution. Kraeplin borrowed heavily from the degenerationist idea that certain patterns of the brain were remnants of our ancient evolutionary past or in Kraeplin's words, "tribal history" (as cited in Blom, 2003, p. 75). Degeneration theory argues that such maladaptive patterns manifest as regressive cognitive and behavioral functioning. As Blom (2003) notes, this theory held wide appeal in 19th century Europe because it gave society a framework for explaining modernity's budding "misfortunes and wickedness" (p. 75). Anthropologist R. Barrett's (1996) comments illuminate the prolific nature of degenerationist ideas in Europe at this historical moment,

> Thus in their physiognomy and behaviour [sic], criminals and the insane were likened to primitives… The urban poor, including prostitutes, criminals, and other immoral types, were degenerates, exemplified by their low brain weights, protruding jaws, and misshapen skulls… Degeneration theory had to do with the proper place of races and classes… Most importantly, it was used to explain the apparent increase in the rates of mental illness and incurability… (p. 67).

Kraeplin's enthusiasm for degenerationist thought was translated into an uncritical use of this non-epistemic construct throughout the development of his *dementia praecox* concept (Blom, 2003). For example, Kraeplin observed, "About 75% of the cases that go to mental asylums appear to reach the higher grades of dementia. The patients gradually sink deeper and deeper, become dull and apathetic and lose all understanding for those around them" (1899, p. 117). These observations led Kraeplin to consider schizophrenia

as degenerative in nature. Unintentionally, Kraeplin's *a priori* commitment to the biological notion of degeneration prevented him from considering the effects of isolation, maltreatment, the cruel experimental treatments frequently used in the institutions of the 19th century, and the social stigma insane people endured at the time (Gilman, 1982), all of which undoubtedly affected clinical course. Despite the fact that Kraeplin himself observed that between 13-20% of these patients improved almost completely, he still spoke monolithically of a degenerative trait inherent in schizophrenia. Kraeplin could not explain this phenomenon of recovery. Interestingly, modern psychiatry has found a solution to Kraeplin's dilemma by often dismissing improved and recovered patients as a result of misdiagnosis (Karon, 2003; Whitaker, 1992). That is, if a person with schizophrenia exhibits significant improvement, today or yesteryear, it is assumed that they were never truly schizophrenic because, the circular logic concludes, schizophrenia is a degenerative disease. Such contemporary proclamations indicate the degree to which Kraeplin's degenerative emphasis has remained.

As can be seen from the above discussion, prior to philosophical post-modernism it was uncommon for scientists to introspect about non-epistemic influences informing their work. After all, science, unlike religion and philosophy, was deemed a mode of investigation exempt from external contaminants and conceived as 'truly objective'. Such thinking formed the basis of Kraeplin's nosological creations and by default allowed him to feel confident that *dementia praecox* existed completely independent of his chosen organizing principles (i.e., syndrome-course unit and degenerative thought). Thus, Kraeplin deemed his description of individuals suffering with *dementia praecox* as a purely objective, in-the-world natural phenomenon open to scientific discovery. In

Hegelian terms, Kraeplin 'removed himself from history' and was able to see the world as it 'truly' exists. This is best known as philosophical realism and it too has had a profound impact on modern biopsychiatric conceptions of schizophrenia (Blom, 2003).

Philosophical Realism and Kraeplin's Dementia Praecox Construct

Reliance on a realist position reifies schizophrenia as a natural disease entity that allegedly exists independent of the observer. A consequence of this in daily practice is the location of schizophrenia as a phenomenon to be examined outside the ebb and flow of common human experience and this has contributed to the suspension of curiosity in regard to the psychosocial and mentalized aspects of schizophrenia.

Positioning the observer outside the schizophrenic experience, whether the observer is a researcher or practitioner, accomplishes a few things. First, a concretized professional-patient hierarchy is constructed within which information flows uni-directionally. Unto itself the professional-patient hierarchy is not detrimental and will of course exist in a situation when one person (the patient) seeks professional assistance from a trained other (psychotherapist, psychiatrist, etc.). However, it is my assertion that in the biopsychiatric model the doctor/schizophrenic split is hypertrophied due to its epistemological relationship to degeneration theory. That is, the patient is perceived as wholly different due to an inherent, archaic defect and the gap between schizophrenic patient and doctor will only widen as the degenerative trait runs its inevitable course. At best, this concretized hierarchical relationship results in the benevolent transmission of sympathy and basic human concern for the welfare of a suffering other. Yet, this hierarchy can also result in an institutionalized, unilateral mandate for compliance to *en*

vogue treatments. Modern mental health strategies for schizophrenia are a combination of these two outcomes, with an unfortunate emphasis on the latter as seen in many community agencies, day care treatment facilities, and group homes that impose medication compliance as a prerequisite for participation in their services (Karon, 2003). Medication non-compliance is interpreted as a 'lack of insight' on the part of the sufferer and is considered symptomatic of schizophrenia itself, rather than an expression of a person's discomfort with side-effects, lack of drug effectiveness, or his or her demand to be treated as a person with a story to tell. In this sense, the schizophrenic person is regularly considered alien from prototypical insight common to the non-schizophrenic 'normal'. A person suffering with schizophrenia is viewed as lacking sentience and judgment - two definitive aspects of human-ness (Whitaker, 1992). The real-world consequence of this concrete split is that the schizophrenic is typically not considered integral to the development or modification of treatment strategies due to their assumed lack of insight.

It is essential to note that the push for current medication compliance should not be interpreted as pure malevolence. Rather, it is an expression of the basic suppositions of biopsychiatry and is undoubtedly a well-meaning attempt by the majority of practicing psychiatrists to alleviate real human suffering. As such, it is all the more important for psychiatrists to understand the philosophical roots driving their clinical recommendations in order to develop the best possible approach to schizophrenic suffering. Understanding the realist underpinnings of biopsychiatric thought better equips one to comprehend the primacy of current neuroleptic treatment strategies. After all, if the origin of madness is genetic, internal to the sufferer from birth and is disconnected from the impact of human

interactions, then there is no reason to consider any treatments other than biologically-oriented ones. Further, and of particular relevance to this dissertation project, if a schizophrenic lacks the capacity for insight and is excluded from treatment decisions in general, psychodynamic psychotherapy will be the first to be labeled useless and ineffective. Indeed, marginalization of psychodynamic psychotherapy for the treatment of schizophrenia has been made official by the schizophrenia PORT recommendations (Lehman & Steinwachs, 1998) and others (Drake & Sederer, 1986), despite the lack of empirical backing for such assertions (Gottdiener & Haslam, 2002; Karon, 2003; ver Eecke, 2003).

In concluding this philosophical section, Blom (2003) asserts that if current treatment approaches are to prove effective we must come to appreciate that the biopsychiatric notion of schizophrenia is "embedded in a matrix of scientific assumptions… guided by various culture-religious themes which dominated psychiatric thinking at the time [of its development]…and [has been] applied rather uncritically within the field…" (p. 9). Others have similarly investigated the scientific immaturity of the schizophrenia construct (Barrett, 1996; Bentall, 1998; Boyle, 2002; Warner, 2004).

Section II: The Empirical Validity of the Degenerative Disease Construct

If schizophrenia is a chronic, degenerative brain disease then naturally untreated schizophrenia would ultimately leave a person significantly incapacitated or dead. That is, for people suffering with schizophrenia it would be impossible to evidence signs of recovery. This is the idea driving the 'management rather than catharsis' strategy of medication treatments. However, what does the empirical literature have to say in regard

to the degeneration assumption? The answer can be found by asking two interrelated questions: 1) does the duration of untreated schizophrenia predict psychotoxic brain damage and/or poorer overall outcome as insisted by biopsychiatry; and 2) are there studies exhibiting any degree of significant improvement or recovery for persons diagnosed with schizophrenia?

Duration of Untreated Schizophrenia: Psychotoxicity and Clinical Outcome

Based on the biopsychiatric assumption that schizophrenia is a degenerative brain disease, biopsychiatry has concerned itself with the potential negative consequences of not treating schizophrenia as soon as possible. More specifically, it is a widely held belief that there is a "potential for untreated psychosis to be toxic to the brain" (Ho et al., 2003, p. 142). 'Untreated psychosis' is defined as the interval from first psychotic symptom to first treatment received. This question is very important to the main question of this dissertation project because the idea that psychosis left untreated by medications results in psychotoxic effects has raised ethical questions regarding the use of placebo controls in experimental drug trials (Craig et al., 2000). Similarly, and quite pertinent to this dissertation project, this notion has made inclusion of un-medicated schizophrenics in psychotherapy outcome studies subject to professional scorn and open to allegations of malpractice (Bola, 2006; Harman, 2003; Karon, 1989, 2003). Therefore, empirical resolution of this hypothesis is necessary for opening up new areas of psychotherapy research and expanding our perspective beyond a medication-only treatment approach.

Using structured interviews with schizophrenic patients and their families, as well MRI scan measurements, Hoff and colleagues (2000) found no significant correlations

between duration of untreated schizophrenia and severity of either cognitive or structural brain deficits. The authors conclude that an "uncontrolled toxic brain process [is an] unlikely" explanation of schizophrenic suffering (p. 1824).

Using a variety of clinical measures to assess positive and negative psychotic symptoms, hopelessness, depression and anxiety, Craig and colleagues (2000) found that the duration of untreated schizophrenia was not significantly associated with severity or course of illness. In fact, the only statistically significant finding suggests that the earlier neuroleptic drugs are given, the more disturbed is the course and severity in regard to anxiety and depression. The authors conclude, "…our results failed to support the contention that a longer exposure to psychotic symptoms *per se* leads to a worse clinical outcome…[and]…do not support the suggestion of a psychotoxic effect exerted by prolonged exposure to psychosis" (p. 66).

Utilizing advanced neurological measurement devices, Ho and colleagues (2003) found no significant correlation between length of untreated schizophrenia and neurocognitive functioning, brain volumetric measurements, or surface anatomy measurements. The authors conclude, "[Our results] suggest that large-scale initiatives[3] designed to prevent neural injury through early [medication] intervention in the pre-psychotic or early psychosis phase may be based on incorrect assumptions" (p. 142).

Perkins, Gu, Boteva and Lieberman (2005) performed a meta-analysis of forty-three studies that investigated the impact of the duration of untreated schizophrenia. The

[3] David (2004) addresses the economic and clinical wisdom of early, large-scale medication initiatives proposed by pharmaceutical companies. He states, "From a dispassionate appraisal of the research literature on the treatment of schizophrenia…If an early intervention service is to be useful, it should be at the population level, free of side-effects and inexpensive, and should not undermine clinical practice" (e.g., medication effects causing brain damage that makes quality, continuous treatment less effective and less probable; p. 111).

results of this meta-analytic study found no association between untreated schizophrenia with neurocognitive deficits or morphological changes in the brain, therefore refuting the idea that schizophrenia is a degenerative brain illness that has psychotoxic effects on the brain. Further, this meta-analysis did not bear out support for early medication intervention strategies since it found no association between the duration of untreated schizophrenia and symptom relapse.

In their own review of the literature investigating the impact of the duration of untreated schizophrenia, Segarra and colleagues (2006) call for a redefinition and reconsideration of the assumptions that the duration of untreated schizophrenia deleteriously impacts clinical course, outcome, and brain structure. Based on this redefinition, the authors suggest that the design of early intervention programs reflect a heterogeneous rather than medication-only approach.

Ayres and her Brazilian colleagues (in press) recently explored the impact of the duration of untreated schizophrenia in a large-scale São Paulo study. Using cognitive tests to assess working memory, attention, and verbal fluency of patients with first-onset schizophrenia the authors found no correlation between the duration of untreated schizophrenia and cognitive deficits.

Of course, there are studies that find a relationship between the duration of untreated psychosis and clinical outcome (e.g., Clarke et al, 2006). After all, a person left to suffer without intervention – whether psychotherapy, medication, or both – may of course regress emotionally, though physiological damage appears unlikely. However, the majority of empirical evidence, including meta-analyses, refutes the degenerative/neurotoxic position. Rather, clinical regression is more likely to be

indicative of the existential, vocational, interpersonal and social alienation resulting from the onset of a frightening schizophrenic condition, rather than the supposed neurotoxiciy of schizophrenia without medication treatment (Karon, 2003). Concerning the neurotoxic assumption McGlashan (2006) asks and replies,

> [Does active psychosis] engineer brain cell death and deterioration? This hypothesis is currently popular as an explanation of the duration of untreated psychosis effect in early schizophrenia. The clinical and neurobiological evidence for its validity is…found wanting (p. 609).

If anything, research is far more supportive of the idea that any significant neurological damage associated with schizophrenia is likely related to the medications used to treat the illness rather than the illness itself (APA, 1994; Breggin, 1991; Jackson, 2005; Whitaker, 2002, 2005).

Taking all this into consideration, it becomes all the more pressing to design early-intervention strategies based on research of all possible short- and long-term treatments (e.g., Alanen, 1997), rather than the false and draconian assumption that not using medications is psychically and organically destructive. That is, it is a primary, though false assumption of biopsychiatry that not administering drugs immediately is malpractice (Bola, 2006; Breggin, 1991).

Of course, altruistically, psychiatrists want to minimize the subjective suffering of a person suffering from distressing symptoms. This is often stated as a further justification for the heavy reliance on psychiatric drugs, especially in the early period of the illness when the symptoms are particularly acute and disturbing for all involved. However, it is also false that *only* drugs can quickly minimize acute psychotic symptoms

such as frightening hallucinations and disorienting delusional constellations (Alanen, 1997; Irwin, 2004a; Karon & Vandenbos, 1981; Harding & Zahniser, 1994; Paul, Tobias, & Holly, 1972; Paul & Lentz, 1977). Most recently, research emanating from the Scandanavian research of Alanen (1997) and Seikkula and colleagues (2000, 2003) speak to the empirical and clinical effectiveness of psychological intervention for early and acute positive symptoms with little to no use of neuroleptic medications.

This entire discussion is relatively moot if there is little in the way of counter-evidence suggesting that there is real, long-term hope for people suffering from schizophrenia. A look at the body of longitudinal schizophrenia outcome studies adds additional empirical evidence that schizophrenia is *not* a chronic, progressively degenerative brain disease leaving such people without hope.

Hope or Dread: Longitudinal Studies of Schizophrenia

International and U.S.-based studies have investigated the degenerative assumption and they have found significant heterogeneity of outcome with a variety of received treatments (e.g., psychotherapy and medication) and even without treatment. I will provide an illustrative summary of the longitudinal evidence, but for the interested reader more extensive summaries of the longitudinal literature are available elsewhere (Leff, Sartorius, Jablensky, Korten, & Ernberg, 1992; Harrison et al., 2001; Hopper, Harrison, Aleksander, & Sartiorius, 2007; Hopper and Wanderling, 2000; Jablensky et al., 1992; Menezes et al., 2006). To more clearly understand the studies presented below it is important to hold in mind the operational definition of 'recovery' imparted by Harding and Zhasiner (1994) based on their summary of the longitudinal evidence,

> The universal criteria for recovery have been defined as no current signs and symptoms of any mental illness, no current medications, working, relating well to family and friends, integrated into the community, and behaving in such a way as to not being able to detect having ever been hospitalized for any kind of psychiatric problems (p. 141).

Clearly, the working definition of 'recovery' used in the following studies to which I will now turn is quite strict and highly relevant to day-to-day functioning.

In 1968, Murphy and Raman (1971) relocated 89 of 90 first-episode schizophrenics, admitted twelve years earlier to a hospital in the developing country of Mauritius. Patients and a person close to them throughout the intervening years were interviewed to determine current mental state and whether any relapses had occurred. Despite the fact that *no* neuroleptics were used in this developing nation at the time, 64% of the subjects were found to be currently free of symptoms of any kind. Of these 64%, only 8% went on to experience an additional psychotic episode at some point. Not only did the lack of early medication do no harm to these patients, this fact might actually help explain why these relatively low relapse rates are superior to relapse rates in the developed world where neuroleptics are widely used (Hopper, Harrison, Aleksander, & Sartiorius, 2007; Irwin, 2004a; Menezes et al., 2006; Warner, 1985).

Following discharge from a psychiatric hospital, Bleuler (1978) maintained contact with 208 of his schizophrenic patients for 23 years in Zurich, Switzerland. In his analysis of first-admission patients 23% had achieved full recovery, 43% were significantly improved, and 34% had little to no change. Although Bleuler unfortunately did not provide statistical results across treatment interventions, his treatment approach

minimized the use of biological treatments and strongly favored psychosocial approaches for those patients he followed clinically.

Tsuang, Woolson, and Fleming (1979) reported on 186 of 200 people admitted to an Iowa psychiatric hospital between the years 1934 and 1944. Structured interviews were completed with the patients and a first-degree relative of each. This study reported that 20% of the patients had little or no psychopathology at follow-up, 26% had mild symptoms but were generally doing well; and 54% had severe symptoms. Of the studies being reviewed this one provides the most pessimistic results and yet it still refutes the idea that schizophrenia is a progressively degenerative disease since almost half of the patients fared quite well after 35 years.

Huber, Gross, Schuttler, & Linz (1980) reported follow-up results on 502 first admissions diagnosed with schizophrenia in the 1950s. Face-to-face interviews were done with each patient. After an average of 22 years 22% had achieved a full recovery; 43% were described as recovered with no psychotic symptoms; and 35% still endured characteristic schizophrenia symptoms. Were maintenance neuroleptics predominantly responsible for these positive results? The authors report that this was not the case. In fact, *all* of the individuals assessed to be among the healthiest 22% were *not* receiving neuroleptic medications.

Ciompi (1980) published approximately 37-year follow-up data for 289 first admission schizophrenic patients in Switzerland from 1900 onward. He found the course of the disease to be highly variable based on two hours of interviews with former patients and information received from family members and hospital files. He found that 27% had achieved a complete remission and 22% had only residual, non-psychotic symptoms.

That is, there is at least a 50/50 hope for successful outcome. The idea that schizophrenia is degenerative - and even more rapidly degenerative without neuroleptics - is specifically unsettled by Ciompi's findings because more than 50% of the people in his cohort were diagnosed before the invention of any modern neuroleptic treatments. Ciompi concluded that the course of illness is most significantly impacted by "the expectations of the patient himself, his family, and surrounding persons which… seem often to act strongly as self-fulfilling prophecies" (cited in Irwin, 2004a, p. 58)

In contrast to the poor outcome predicted by the biopsychiatric conceptualization of schizophrenia, Harding et al. (1987a, 1987b) found that the course of schizophrenia is significantly heterogeneous even for allegedly treatment-refractory, severely chronic schizophrenics. The authors provide long-term outcome data for 82 of 118 patients who were alive and interviewed 20-25 years after their entry into a Vermont psychosocial treatment project for chronic schizophrenia. In the mid-1950s when patients became part of the study, all of them were chosen to participate in a unique psychosocial rehabilitation program designed for patients previously deemed resistant to medication and other biological treatments. In fact, these were classic 'back ward' patients usually given-up-on as hopeless. Severity and chronicity is attested to by the fact that at the initiation of the rehabilitation/longitudinal study, the average patient had been ill for 16 years, was totally disabled for an average of 10 years, and had been continuously hospitalized for six years. Following the psychosocial rehabilitation program they were released to the community in the mid-to-late 1950s.

Interviews with participants and people close to them, as well as self-report and observational data measuring positive and negative symptoms, hospital records, social

and vocational functioning, number of hospitalizations, and overall quality of life, were all used to assess each patient's current status. Harding et al. (1987a, 1987b) found that 45% of the participants had no psychiatric symptoms and another 23% had some psychiatric symptoms, but none that were of a psychotic nature. That is, an astounding 78% of the once biological treatment resistant, chronic, back ward patients fared very well 20-25 years later. The authors point to the comprehensive rehabilitation program as a potential reason for the success of 78% of this cohort. They specifically highlight the clinical utility of "…a pervasive attitude of hope and optimism about human potential, through the vision that, if given the opportunity, persons with mental illness could become self sufficient" (DeSistro, Harding, McCormick, Ashikaga, & Brooks, 1995, cited in Irwin, 2004a, p. 59).

Ogawa and colleagues (1987) followed 140 people diagnosed with schizophrenia for 21-27 years. The study was designed as a relapse prevention study emphasizing neuroleptic treatment. At follow-up 31% of the 98 people researchers were able to track down were completely free of positive symptoms. The authors failed to report other significant areas that would speak to overall functioning, such as social and vocational functioning and negative symptoms. At follow-up, 65% of the patients were still taking neuroleptics and the suicide rate was a relatively high 11%, suggesting that this neuroleptic-only cohort did not fare favorably to other cohorts with less (or no) neuroleptic use.

Harding (1995) summarized five U.S. and international longitudinal studies that followed schizophrenics for no less than 25 years. The authors found that 30% fully recovered in the long run and that 60-70% became self-sufficient. Importantly, results indicated no

further improvement in outcome since the introduction of neuroleptic medication. To the contrary, the long-term evidence suggests that outcomes have deteriorated since the introduction of medication-only clinical practices (Whitaker, 2002, 2005). In this regard, Harding (1995) made a startling observation: *all* of the patients who fully recovered were among the 50% of the original sample who reported to have stopped taking their medication. This could suggest that either the higher functioning patients felt freer to discontinue their medications - though this is not supported by the neuroleptic side-effect or outcome literature (Breggin, 1991) - or, more likely, that medication may preclude full recovery.

Kua and colleagues (2003) published results of a 20-year longitudinal study carried out in Singapore. In this cohort, 44% of the participants were found to be in good to excellent psychological condition as measured by a global assessment scale. Of the total subject pool 48% were not receiving any neuroleptic medication and *all* of the 28% of the subjects with the highest level of functioning were among those *not* using neuroleptics.

Menezes and colleagues (2006) reviewed 37 studies with a total of 4100 patients and a mean follow-up period of 35 months. Assessing a multitude of variables (e.g., country of study, psychosocial rehabilitation, age), they found that the sole use of neuroleptic medications was associated with worse outcome, whereas a combination approach (i.e., medications plus psychosocial rehabilitation) was a predictor of positive outcome. Unfortunately, psychotherapy-only treatments were not analyzed.

Results of the World Health Organization (WHO) studies are extensively reported elsewhere (Harrison et al., 2001; Hopper et al., 2007; Hopper and Wanderling, 2000; Jablensky et al., 1992) and only a brief summary will be provided here. These studies

compare longitudinal outcome data in developed versus developing countries, using results from 2, 5, 15, and 25 years of follow-up data. The generally positive outcomes of people with schizophrenia in these studies provide additional evidence refuting the degenerative brain disease assumption. Importantly, the WHO-sponsored studies all used the same standardized outcome measures thus making the results highly generalizable. Quite pertinent for this dissertation project, the identical assessment methods provide fascinating data regarding the potential role of neuroleptic medications in the less favorable outcomes of the richer and allegedly more clinically advanced nations.

In sum, 38% to 48% schizophrenic participants recovered across geographic locales. In poorer developing countries, participants were two to three times *more* likely to have good clinical outcomes. In regard to vocational functioning, people in poorer developing nations were three to four times *more* likely to be holding down a job (Hopper & Wanderling, 2000). Suicide rates were higher in the economically richer, more medication-reliant countries to the point that Harrison and colleagues (2001) recommend special suicide prevention programs be established in the richer developed nations. Follow-up research revealed that the poorer countries used significantly less medications (Jablensky et al., 1992). A striking example of the potential negative effects of medication is seen when comparing Agra, India to the far richer Aarhus, Denmark. In Agra, outcomes revealed that 63% of the participants had experienced complete remission, compared to only 17.5% in Aarhus.

Thus, based on these international and U.S.-based longitudinal studies, the *DSM-IV* statement (p. 282) reflecting a main supposition of biopsychiatry, "Complete remission (i.e., a return to full pre-morbid functioning) is probably not common in this

disorder", is quite clearly empirically invalid. To conclude, it is worth citing the entirety of Harding and Zhasiner (1994) summarization of the longitudinal literature,

> …all of these studies have come to the same conclusion. The longer investigators followed an identified intact cohort (whether probands were in or out of treatment), the more pronounced the picture of heterogeneity and improvement in function. These studies have found that half to two thirds of patients significantly improved or recovered, including some cohorts of very chronic cases…All of these investigators of long-term studies were trained in the older, more pessimistic conceptual models [i.e., Kraeplian degenerative brain disease model] and were surprised by their own findings. Because the myths have been repeated so often, they had become reified. The strong belief systems and resistance, encountered by these investigators, were caused by many factors and were not easily altered by one study. However, there is now a confluence of results (p. 141).

These longitudinal studies provide significant hope that schizophrenia is not degenerative and that significant improvement and even recovery is quite possible, especially if medication treatments are not relied on as the sole or primary intervention.

Simply because the biological and degenerative basis of schizophrenia is empirically unfounded does not necessarily translate into the *ineffectiveness* of medication treatments, though the poorer results of the richer, drug-reliant nations is not a good starting point to claim clinical effectiveness. That is, it still may be possible to successfully treat schizophrenia biologically even if it seems to be far more than just a biological disease. Let us now turn to the empirical literature investigating the clinical effectiveness of older and newer neuroleptic treatments.

Section III: The Effectiveness and Safety of Neuroleptic Medications

The lack of evidence delineating the biological correlates of schizophrenia is not what most sufferers and their families and friends are informed about schizophrenia (Jackson, 2005). Breggin (1991) asserts that if patients were accurately informed of what psychiatry *does not know*, use of neuroleptic medications as the primary treatment would undoubtedly be questioned more deeply. Despite ever-ready proclamations of new miracle biological therapies that will solve the schizophrenic puzzle (e.g., insulin coma, lobotomy, electroshock, typical and atypical neuroleptics), none of these treatments have ever proven to be very effective (Whitaker, 2002, 2005). This review will begin with the older, typical neuroleptics.

Effectiveness of the Typical Neuroleptics

Epstein and colleagues (1962) investigated two groups of male, first-episode schizophrenics admitted to nine California State Hospitals during 1956 (n = 673) and 1957 (n = 740). In 1956, 36% (n = 245) received neuroleptic treatment and 64% received no medications. In 1957, 48% (n = 355) of the 740 patients received neuroleptic drug treatment, compared to 52% (n = 385) who received none. The authors set out to investigate whether or not drug treatments shortened hospital stays. Defying the myth that neuroleptic drugs were responsible for 'emptying the asylums' (for a critique see Jackson, 2005; Harding & Zahniser, 1994; Whitaker, 2002, 2005), Epstein et al. found that schizophrenic patients treated with neuroleptics consistently – across nine different

sites – had significantly longer hospital stays compared to patients who did not receive medication treatment.

In 1967, Schooler and colleagues published one-year follow-up results for their National Institute of Health project. The authors reported that the placebo group fared significantly better in terms of clinical symptoms and reduced rates of hospitalization compared to the three neuroleptic groups (chlorpromazine, fluphenazine, and thioridazine). *Post hoc* analyses revealed that following discharge, psychotherapy but not drug treatment, was associated with a higher level of social interaction and "a greater likelihood of a wage earner's job being commensurate with his training" (p. 991). Further, rates of extrapyramidal side-effects were so severe in the chlorpromazine group that 37% of the patients had to be placed on antiparkinsonian chemical agents. As will be discussed below, these side-effects are largely permanent and at times fatal (Jackson, 2005). Ultimately, rather than staying close to the data and concluding that people with schizophrenia do better with minimal neuroleptic exposure and/or psychotherapy-only, Schooler et al. concluded, "…because we were unprepared to recommend placebo as treatment of choice, we explored a number of possible variables that might have caused this" (cited in Irwin, 2004a, p. 61). The variable with which they hoped to explain these results is as follows: "when lack of improvement was observed in the patient [of the placebo group]…it may be that the staff responded to the 'deprived' [off medication] patient with some special quality of care" (p. 61). Needless to say, this conclusion is clearly hyperbolic and has no supporting empirical evidence. Even if it was a valid consideration it would actually only serve to advance the idea that human interactions are quite helpful for people suffering with schizophrenia (e.g., milieu therapy, individual

psychotherapy). Despite the explicit data, the author's endorsement of neuroleptic medications in their concluding remarks provide us perhaps with an early, though instructive example, of the growing drug bias enveloping the field.

Prien and colleagues (1968) investigated relapse rates among chronic schizophrenic patients over a six-month period when medications were withdrawn. Relapse was defined as a return of positive symptoms, extreme hostility and excitement, and/or threatening behaviors. Results were highly suggestive of a positive statistical relationship between medication use and relapse. That is, relapse rates were positively associated with neuroleptic medication dosage. Only 18% of the 65 patients receiving less than 300 mg/day of chlorpromazine relapsed following medication withdrawal. In contrast, of the 60 patients on moderate doses (300-500 mg/day) 47% relapsed; and, of the 53 patients on high doses (greater than 500 mg/day) 58% relapsed. The relapse rate of the low group was significantly different than the higher dose groups, although there was no significant difference found between the moderate and high groups. Most relative to this dissertation project is that of the 18 patients who were *medication-free* during the entirety of the study, only one subject (6%) relapsed over the next six months (6%). In sum, patients exposed to relatively less or no medications seemed to progress better in terms of positive symptoms and interpersonally, whereas exposure to medications precluded recovery and was associated with a greater potential for a return to psychotic functioning.

In a subsequent study, Prien and colleagues (1971) replicated these results. Once again, those patients who did not receive any medications throughout the study fared significantly better. Only 2 of 30 patients who were on placebo at the start of the study

relapsed during the next 24 weeks (7%), whereas the rate of relapse in the drug groups were equal to the high rates found in the initial study. As a result, the authors concluded once again, "Relapse was found to be significantly related to the dose of the tranquilizing medication the patient was receiving before he was put on placebo - the higher the dose, the greater the probability of relapse" (p. 22).

Another manner by which drug effectiveness can be determined is to compare pre-drug era with drug era outcomes. Bockoven and Solomon (1975) compared relapse rates of these two eras and found that 45 of the 100 randomly selected schizophrenic patients treated at the Boston Psychopathic Hospital in 1947 (the pre-drug era) had not relapsed in the five years following discharge. They also determined that 76% were successfully living in the community at the end of that follow-up period. However, of the 100 randomly selected patients treated with medications in 1967 at a Boston community health center, only 31 remained relapse-free for the next five years. The authors found another troubling result from both a clinical and economic perspective: the medication treated patients were much more "socially dependent" compared to the un-medicated patients (i.e., in greater need of continuous state-supported day program treatment and social welfare benefits; p. 801). As a result, the authors concluded, "One unexpected finding of the comparison…is that these drugs might not be indispensable; in fact, they might actually prolong the social dependency of some discharged patients" (p. 796). Bockoven and Solomon's results replicated larger sample studies in other cities confirming the growing suspicion that neuroleptic medications have significant limitations and dangers (Lehrman, 1960; Rachlin, Guritz, Lurie, & Rachlin, 1956).

In their review of 13 neuroleptic treatment studies conducted between 1959 and 1974, Gardos and Cole (1977) found a significant difference between the relative psychological health of relapsers depending on prior treatment with placebo or neuroleptics. They reported that people who relapse following drug exposure "are sicker than placebo relapsers" (p. 34). Due to the 'medicate-first' trend that had already entrenched itself within the field they wrote, "The suggestion that as many as half of schizophrenic patients might not be worse off their…medication…will surely meet with some raised eyebrows" (p. 34). Nonetheless, sticking close to their data they suggested, "The major principle we wish to stress is that every chronic schizophrenia outpatient maintained on an antipsychotic medication should have the benefit of an adequate trial *without drugs*" (p.35; my emphasis). The authors admonished psychiatrists to rethink current medication treatment strategies based not only on their specific relapse results, but also as a result of the permanent side-effects induced by these drugs.

In the late 1970s, investigators helped explain existing evidence indicating that neuroleptic drugs cause a greater number of relapses, more regressed relapses, as well as poorer symptomatic and social outcome, by identifying the specific biological changes induced in the brain by neuroleptic medications. Chouinard and colleagues (Chouinard, Jones, & Annable 1978; Chouinard & Jones, 1980; Muller & Seeman, 1978) found that because neuroleptic drugs greatly decrease dopamine activity the brain compensates by becoming 'supersensitive' to dopamine by initiating a proliferation of dopamine receptors. As a result, when a patient stops taking dopamine blockers (i.e., neuroleptics) the brain is standing by with a far greater number of dopamine receptors to take in an

unusually high amount of dopamine and is therefore more biologically prone to psychosis. Chouinard (1978) remarks in this regard,

> Neuroleptics can produce a dopamine supersensitivity that leads to both dyskinetic (i.e., parkinsonian symptoms) and psychotic symptoms. An implication is that the tendency toward psychotic relapse in a patient who has developed such a supersensitivity is determined by m*ore than just the normal course of the illness...[and] the need for continued neuroleptic treatment may itself be drug-induced* (p. 1410; my emphasis).

Although psychiatrists often interpret the resurgence of psychotic symptoms as a return of the underlying schizophrenic brain disease and justification for lifelong neuroleptic management, this research conducted almost 40 years ago definitively showed that relapse is more related to drug-induced psychosis and not to any pre-existing schizophrenic deficit.

Recently, Moncrieff (2006) summarized the literature exploring the relationship between drug exposure and 'supersensitivity psychosis' and confirmed these original studies of the 1970s. Moncrieff's research, unlike the earlier studies, was able to include the newer generation of atypical neuroleptics in his review and found that they too are implicated in withdrawal-related psychosis. He writes, "It appears that the psychosis [evident upon relapse] may be a feature of drug withdrawal rather than the emergence of the underlying illness…" (p. 1). In other words, not only have neuroleptics been shown to be ineffective but also harmful above and beyond the well-known side-effects to be discussed below. These authors and others have also concluded that these supersensitivity

effects may be irreversible similar to tardive dyskinesia[4] and other diseases (e.g., diabetes) produced by neuroleptic medications.

Needless to say, the NIMH of the 1970s was startled by these findings that indicated the ineffectiveness and dangerousness of neuroleptic drugs. As a result, the NIMH was compelled to revisit whether or not schizophrenia could be successfully treated without drugs (Whitaker, 2002). Three NIMH-funded studies were conducted to explore this idea, all of which concluded that newly admitted schizophrenics fared better when treated *without* drugs.

Carpenter, McGlashan and Strauss (1977) conducted the first of three NIMH studies. In this investigation, 49 schizophrenia patients were placed in an experimental hospital program that provided them with individual and group psychodynamic psychotherapy. These patients were compared to 73 schizophrenia patients receiving status quo drug treatments. Only 35% of the non-medicated patients relapsed within a year after discharge, compared to 45% of those treated with medication. The medicated patients also experienced more depression, blunted emotions, and retarded movements. An obvious but no less important observation was that the non-drug treated patients did not have to endure the many different marked physiological and emotional side-effects associated with neuroleptic use.

In a study of young male schizophrenics, Rappaport and colleagues (1978) found that patients treated *without* mediations while in the hospital and did *not* use medications during the three year follow-up period fared significantly better than 1) patients treated

[4] Tardive dyskinesia is an irreversible physiological disease that results in involuntary and irregular muscle movements, usually, but not limited to, the facial region. Tardive dyskinesia and a number of other extrapyramidal side-effects associated with neuroleptic exposure will be discussed in greater detail below.

with medications while in and outside the hospital; 2) patients treated with medications while in but not outside the hospital; as well as 3) patients not treated with medications in the hospital but with medications during the follow-up period. That is, simply stated, the least possible exposure to medications the better, particularly in regard to symptoms, re-hospitalizations, need for continued treatment and communal functioning. The authors concluded, "Routine and continuous use of [neuroleptics] may be contraindicated…" (p. 107). Further, the authors make a statement one would be hard-pressed to find in the current mainstream biopsychiatric literature, "The findings underlie the need for further study of how to utilize antipsychotic medication more selectively in the treatment of schizophrenia" (p. 100).

In the last of the three NIMH studies, Matthews and colleagues (1979) reported that patients treated without neuroleptics in a non-professionally staffed milieu setting fared better than patients treated with medically trained doctors administering neuroleptics. By six weeks, the two groups fared equally well in regard to psychopathology, refuting the claim that neuroleptics are far superior for short-term decreases in acute positive symptoms. Over the two-year follow-up period, the un-medicated patients had a healthier level of social functioning, reported less subjective distress, and were incurring less treatment costs compared to the drug group. These results have been replicated by U.S. and international researchers (Cullberg, 1991; Karon & Vandenbos, 1981; Lehtinen, Aaltonen, Koffert, Rakkolainen, & Syvalahti, 2000; Mosher, Vallone, and Menn, 1979). Carpenter et al. (1977) succinctly sums the NIMH-funded research of the 1970s,

There is no question that, once patients are placed on medication, they are less

> vulnerable to relapse if *maintained* on neuroleptics. But what if these patients had never been treated with drugs to begin with?... We raise the possibility that antipsychotic medication may make some schizophrenic patients more vulnerable to future relapse than would be the case in the natural course of the illness (p. 19).

Despite the clear-cut conclusions favoring the non-drug treatments of all three NIMH and replication studies, the NIMH has decidedly moved away from this type of research. These studies had virtually no impact on the dominant position of drugs in the treatment of schizophrenia – yesterday or today (Breggin, 1991; Jackson, 2005). These studies became even more irrelevant in the minds of mainstream biopsychiatric researchers and clinicians following the development of the newer atypical neuroleptics (in the 1990s) which biopsychiatry currently deems superior to the older typical neuroleptics. However, as will be seen below, the superiority of atypicals over typicals is a faulty assumption that has further contributed to the over-reliance of neuroleptics as the frontline treatment for schizophrenia sufferers. Prior to direct empirical comparison of the older and newer drugs, in the service of an honest appraisal of drug treatments for schizophrenia, further review of studies that include both older *and* newer neuroleptics is in order.

Effectiveness Research Including Typical and Atypical Neuroleptics

If typical and atypical neuroleptics truly have specific 'antipsychotic' effects then they should show superior effects compared to simple sedatives (e.g., benzodiazepines). Yet, all reviews of the relatively scant 'neuroleptic versus sedative' literature found no differences between the two treatments (Carpenter, 1999; Keck, 1989; Wolkowitz, 1991). Whitaker (2002) ironically summed the evidence, "Forty years after neuroleptics were

introduced, and still there was no convincing proof that the [neuroleptic] drugs were any better at knocking down psychosis than old-fashioned opium powder" (p. 200). In response to these unsettling results, Turns (1990) self-deprecatingly asks, "Has our clinical judgment about the efficacy of antipsychotics been a fixed, encapsulated, delusional perception…Are we back to square one in antipsychotic psychopharmacology?" (cited in Whitaker, 2002, p. 200).

Healy and colleagues (2005) took advantage of an idiosyncratic demographic situation in North Wales to study the effectiveness of psychiatric medications. In the region under investigation the population has been stable for at least a 100-year period in terms of number of people, age, ethnic mix and rurality. The authors found that there has been a three-fold increase in the rate of detention into psychiatric services and a 15-fold increase in the rate of hospitalizations since the introduction of medications. Speaking directly to medication effectiveness, the periods of *healthy* functioning for psychotic disorders such as bipolar disorders have actually become *shorter*, indicating that people are sicker more rather than less of the time. Overall, mentally ill patients spend a greater amount of time in a hospital bed today then they did 50 or 100 years ago. That is, treatments are becoming increasingly ineffective, even relative to Victorian times.

Not unlike the results presented earlier (Chouinard et al., 1978; Chouinard & Jones, 1980; Matthews et al., 1978; Mosher et al., 1979; Muller & Seeman, 1978), Whitaker (2005) has pointed out that medications are partially responsible for ever-greater cases of chronic schizophrenia. He reported multi-site MRI evidence that have shown a connection between neuroleptic drug usage and enlargement of the basal ganglia which has been linked to greater severity of both positive and negative symptoms (e.g.,

Gur et al., 1998). Whitaker (2005) concluded, "In other words, [research has] found that over the long term, the drugs cause changes in the brain associated with a worsening of the very symptoms the drugs are supposed to alleviate" (p. 8).

Right up until the advent of the newer and more expensive atypical neuroleptics the drug industry and mainstream biopsychiatry actively promoted the effectiveness and safety of the original typical neuroleptics. Upon the advent of the atypicals, one finds greater discussion of the danger and marginal effectiveness of the typical neuroleptics once deemed 'miracle cures'. Some believe that this honesty is better explained by the need for the drug-industry to promote and sell its more expensive atypicals now that the older drugs have lost their patents and thus profitability (Breggin, 1991; Whitaker, 2002). Within psychiatry and psychology, it is a widespread belief that the atypicals are clinically superior and safer than their predecessors. As will be shown, this belief is predicated upon information stemming from randomized controlled trials (RCT) mainly undertaken by drug companies during the FDA approval process. However, there are significant methodological flaws inherent in these trials, as well as a good deal of non-drug company research refuting the superior effectiveness and safety of the atypicals. To this topic I now turn.

Atypical Neuroleptics versus Typical Neuroleptics

There is little sound scientific evidence indicating the superior safety and effectiveness of the newer, atypical neuroleptic medications (Stip, 2002). Why then do the newer atypicals hold a 90% market share in the United States? The potential answer to this question may be that "physicians have come to rely increasingly upon information

from published sources (e.g., clinical drug trials) rather than from direct observation" or non-drug industry supported research (Jackson, 2005, p. 7) As the main data source for psychiatrists, it is imperative to understand the contemporary FDA drug trial research design (i.e., the Randomized Controlled Trials) employed by pharmaceutical companies that brings psychiatric drugs to the marketplace and justifies their widespread use for the treatment of schizophrenia.

How a Psychiatric Drug Comes to the Marketplace

In 1962 the U.S. Congress empowered the FDA with the responsibility of assessing the therapeutic efficacy and safety of medications for inclusion in the marketplace. Because psychiatrists and other medical professionals rely almost entirely on the data generated in these drug trials for informing treatment, it is essential to understand the methodology of this process. Further, it is important to keep in mind that the RCT is also the preferred design for the subsequent, post-FDA approval process studies also generally sponsored by pharmaceutical companies. Thus, the following critique is generalizable to the vast majority of pre- and post-approval drug efficacy studies (Hegarty, Baldessarini, Tohen, Waternaux, & Oepen, 1994; Jackson, 2005; Thornley & Adams, 1998).

The FDA drug development process is divided into four phases. Phase I, or the 'Pre-Clinical Investigations', involve the study of a drug's effects on animals, cell cultures, and tissue samples. This phase aids in the determination of metabolic pathways, possible mechanisms of action, and toxicity.

In Phases II-IV, the 'Pre-Marketing Clinical Investigations', human subjects are brought into the process. Phase II typically includes 100 healthy volunteers, with the intention of determining any adverse effects and general drug metabolism processes not detected in non-human species. This phase is essential for the development of a safe dose range without significant negative side effects.

Phase III includes between 100-300 individuals suffering from the illness the drug is meant to treat. The goal of Phase III is to fine tune dose range and nascent ideas regarding the drug's efficacy for use with actual patients.

Phase IV studies are the last investigations required before receiving FDA approval. These studies are randomized, controlled, double-blinded trials consisting of approximately 1000 participants and commonly known as Randomized Controlled Trials, or RCT's. RCT's are short-term (4-8 weeks) and involve the testing of the proposed drug against other biological treatments and placebo (usually sugar pills). Upon completion of Phase IV, the pharmaceutical company files a 'New Drug Application' with the FDA outlining the results of the four phases.

The FDA looks for efficacy in at least two similar and well-monitored Phase IV trials. Efficacy is defined as "a statistically significant, therapeutic effect in the absence of unacceptable toxicities" (Hall, 2003, cited in Jackson, 2005, p. 9). Effect in this case is defined by comparing untreated (i.e., placebo) with drug-treated subjects, therefore there is no requirement that the proposed drug exhibit statistical superiority to other treatments such as psychotherapy. The FDA's standard for psychiatric drug approval is "far more ambiguous than may be commonly assumed" (Jackson, 2005, p. 9). This ambiguity is not speculation, rather, it is a fact acknowledged by the FDA,

> The evidence adduced in the sponsor's short term studies, although it unquestionably provides compelling proof in principle of (the drug's) acute action, it does not provide a useful quantitative estimate of how effective the drug actually will be in the population for whom it is likely to be prescribed upon marketing (Leber, 1996, cited in Jackson, 2005, p. 10).

The RCT method and design that provides "proof in principal" but not a "useful quantitative estimate" may be seriously flawed. Considering that these drug trials provide the evidence that guides schizophrenia treatment a more nuanced breakdown of the RCT is in order to clearly ascertain what we know and do not know about the safety and efficacy of antipsychotic medications.

The RCT: A Scientifically and Clinically Flawed Approach

Published overviews regarding pharmaceutical company sponsored drug trials point out profound deficiencies in published and unpublished FDA research methods and designs (Antonuccio, Danton, & McClanahan, 2003; Breggin, 1991; Moncrieff, 2001; Safer, 2002; Whitaker, 2002). Again, it is essential to keep in mind that the RCT is the preferred design for subsequent, non-FDA studies as well. Thus the following critique is generalizable to the vast majority of drug efficacy studies used by psychiatrists when selecting treatments (Jackson, 2005; Thornley & Adams, 1998). Jackson (2005) provides the most recent integration of these critiques of the RCT and I will summarize her findings by highlighting the methodological flaws most relevant to the neuroleptic drugs currently used for the treatment of schizophrenia.

Flaw #1: Ecological Validity and Selection Bias. A main strategy in a drug study is to narrow the subjects as much as possible. Logically, this makes sense since the aim of a drug study is to determine efficacy of a certain drug with a certain illness. However, in the real world of clinical practice there is rarely such a thing as a 'pure schizophrenic'. Yet, this is what the RCT design attempts to achieve. Common exclusions in an RCT are any or all of the following: age younger than 18 or older than 65; co-morbid physical or mental illness; pregnancy; severe distress (e.g., suicidal thoughts); inpatient status; and poor response to previous treatment. Rather than including these all-too-common variables as potentially informative covariates, they are generally excluded. The co-morbidity exclusion is particularly problematic because contemporary research indicates that multiple Axis I problems are often intertwined with multiple Axis II personality processes greater than 50% of the time (Westen, Novotny, & Thompson-Brenner, 2004).

Flaw #2: Non-equivalent Dosing. In Phase IV of the FDA process, efficacy and safety are the target concerns. In these studies the newer atypical neuroleptics are compared to older typical neuroleptics. Equivalent dosing is defined as the administration of comparable doses of each drug, based on the well-defined chemical standards (Bezchlibynk-Butler & Jeffries, 2002). Non-equivalent dosing is the use of "super doses" (Jackson, 2005, p. 28) for one or the other medication in the trial. Since Phase IV concerns a patient simply staying on the drug for both a measure of efficacy and safety (i.e., no intolerable side effects resulting in discontinuation from drug exposure), dosing is an integral aspect of outcome. Obviously, it is scientifically *and* clinically unwise to use non-equivalent dosing since this will potentially lead to intolerable and/or dangerous side effects for a particular study group, and therefore disallow actual comparison of the

drugs themselves. Yet, this is the common practice for manufacturers seeking to gain approval for a new product. This strategy makes it more likely for researchers to conclude that the newer (i.e., more expensive product with a new patent) drug is safer and more effective. Of course, whatever symptom ratings scales are used will also reflect the newer drug's superiority, because patients being overdosed on the older drug are in tremendous distress, both mentally and physiologically. Further, the hyper-sedative and other deleterious side effects of overdosing will artificially widen the gap between the groups in regard to negative symptoms (e.g., apathy, social isolation, flat affect). This is the origin of the regularly reported belief that compared to older typical agents the atypical antipsychotics are more effective for the treatment of negative symptoms.

Phase IV of the atypical neuroleptic olanzapine approval process provides an illustrative example of non-equivalent dosing. In this study, olanzapine was compared to the typical neuroleptic haloperidol in the hope that it would provide compelling empirical proof to psychiatrists and other health care professionals that this new drug is safer and more effective and therefore should be turned to as a frontline treatment for schizophrenia. Table 2 illustrates the non-equivalent dosing strategy (Jackson, 2005, p. 29). A comparable dose of haloperidol to olanzapine is 2 mg to 10 mg, respectively. Thus, in the high dose groups, haloperidol subjects received 4-6 times the olanzapine dose; in the medium groups, haloperidol subjects received 7-8 times the olanzapine dose; and in the low dose groups, haloperidol subjects received 8-20 times the olanzapine dose. Subsequent studies using either equivalent dosing (Rosenheck et al., 2004) or naturalistic design (Lieberman et al., 2005) have not found any of the atypicals to be superior in most respects when compared to the older medications.

Flaw #3: Concomitant Medications. For patients with co-morbid ailments (e.g., diabetes), adjunctive medications are often being ingested during a drug trial. Of course, most if not all drugs cross the blood brain barrier and thus actively impact thinking, affect, and behavior (Breggin, 1991; Jackson, 2005). Although these potentially important covariates should undoubtedly be analyzed, they are ignored in RCT drug trials. Oddly, even when investigators prescribe a drug to ameliorate side effects of the study medication (e.g., drooling), this is not disclosed publicly nor is it analyzed empirically.

Flaw #4: Placebo Washout. Placebo washout is perhaps the most important factor leading to distorted information passed on to psychiatrists. Similar to non-equivalent dosing (Flaw #2), this practice is dangerous to the physiological and emotional well-being of research participants. 'Placebo washout' is the 7-10 day period during which all participants abruptly discontinue previous medications *immediately preceding* the initiation of an RCT study. The intent is to 'washout' the brain of any chemicals so that prior drug use does not influence the study. The word 'placebo' is used because all participants are given an inert substance during this period so that subjects are not aware they have discontinued their medications. Upon initiation of the study the placebo group will of course continue to receive the inert pill or an older typical neuroleptic, while the experimental group will receive the drug under investigation. There are a variety of critical problems with this approach that result in significantly biased results that favor the experimental drug.

First, any patients who exhibit a positive response during the washout phase are removed from the study. As Jackson (2005) writes,

> …it is impossible to discount the presence of the same *placebo effect*…in the subjects who go on to receive the active drug. As the placebo effect is necessarily a part of *all* healing rituals, it is illogically inconsistent to remove some subjects from a study, simply because they manifest this response too quickly (p. 31).

Data provided by Faries and colleagues (2001) show that these positive responders continue to do well as the trial continues without active drugs. Consequently, removing these patients artificially inflates the differences between the experimental and control groups. Second, and more importantly, it is common that people experiencing abrupt, 'cold turkey' discontinuation will experience substantial physiological and emotional fallout (Moncrieff, 2006). What is particularly strange about this practice is that abrupt withdrawal is widely documented within the mainstream psychiatric literature to be very dangerous (Addonizio, Susman, & Roth, 1987; Kornhuber & Weller, 1994). The most efficient, safest, and quickest way to moderate acute withdrawal symptoms is to provide a chemical intervention. Thus, most subjects in the placebo group experiencing acute withdrawal symptoms are at a significant disadvantage, since their bodies continue to adapt slowly to the sudden chemical changes. In contrast, the acute withdrawal symptoms of those in the experimental drug group are buffered more quickly with the administration of an active drug compound. This procedure translates into drug trials that are not measuring the effects of drugs or placebos on an underlying psychosis, but rather the treatment of acute *withdrawal* symptoms. Beyond this, it is empirically well-founded that the body cannot expunge the chemical properties of psychiatric agents within 7-10 days (Breggin & Cohen, 1999; Hubbard, Ganes, & Midha, 1987). Rather, the withdrawal process takes weeks or months, and if attempted too quickly has permanent consequences

on brain tissue and neurologic pathways (Viguera, Baldessarini, Hegarty, van Kammen, & Tohen, 1997). These factors inculcate a unique bias into the antipsychotic drug trial literature and clinical practice.

Flaw #5: Last Observation Carried Forward (LOCF) Analysis. LOCF is a statistical technique for reporting results of subjects who drop-out of a trial in which the researchers use the last recorded assessment for final outcome no matter the point at which they drop out (post-placebo washout). The assumption, of course, is that there would be no change if those participants had remained in the study. This often puts the placebo group at a profound disadvantage since many patients in this group drop out at their most distressed point of medication withdrawal immediately following the end of the placebo washout period. Of course, this practice artificially inflates the results of the experimental drug group.

Flaw #6: Unblinding. The double-blinded study design is intended to prevent patients, researchers, and clinicians (and anyone else connected to the study) from knowing group assignments. Due to the overt side effects of psychiatric medications, which are easily recognized by clinicians and researchers (and likely by patients), 'unblinding' is common in drug trials and thus results may be strongly influenced by treatment expectations.

Flaw #7: Omission of Data and Methodological Changes. In their review of 10 drugs approved by the FDA between 1987 and 1997, Khan and colleagues (2000, 2001) found that drug companies omitted important and relevant data. The most serious omissions were the unreported data tracking suicide attempts and completed suicides for two antidepressants and two atypical antipsychotics. Additionally, during the olanzapine

approval process researchers changed the study from a 6-week study to a 4-week study due to too many subjects in the olanzapine group dropping out as a result of intolerable side effects (Jackson, 2005). The 6-week data was omitted from the final reports and only discovered by a request taking advantage of the congressional Freedom of Information Act. Recalculation of the 6-week data revealed results that are far less positive for olanzapine. Of course, omitted data and methodological changes make it difficult for subsequent researchers and clinicians to study and use the drug most effectively, safely, or not at all. The most recent (January 2007), but not isolated example of data omission, was seen in Eli Lilly's recent $1.2 billion class-action settlement resulting from their failure to report results regarding the association between olanzapine, diabetes and morbid weight gain.

Flaw #8: Redundant Publications, Suppression of Negative Results, and Ghost-Writing. Recently, pharmaceutical company Eli Lilly has been found guilty of re-packaging the same trial data in a series of overlapping papers in regard to the olanzapine approval process. Similarly, Glaxo Smith Kline recently re-packaged data in an attempt to exhibit the superiority of its product over a competitor's drug. Besides this being a medical ethics violation, redundant publishing changes the findings of meta-analyses and thus evidenced-based medicine as we know it.

In regard to negative results, there are two important distortions that impact the objectivity and validity of the drug trial literature. First, litigation has shown that drug companies have hidden negative results not only from the public, but from the FDA as well (Jackson, 2005). Second, many researchers who have conducted FDA trials are silenced by non-disclosure agreements, thus greatly tying their hands in regard to

reporting or whistle-blowing about dangerous medications. Antonuccio, Danton, and McClanahan (2003), Breggin (1991), and Ross and Pam (1995) all provide extensive documentation of these ethical violations. Third, 'ghost writing' is when an academic is paid to place his or her name on a paper or conference presentation to lend it authority. According to the deputy editor of the *Journal of the American Medical Association*, "This practice is well-known, scandalous, and outrageous. It is a perfect illustration of deceptive authorship practices for commercial reasons" (Larkin, 1999, cited in Jackson, 2005, p. 39).

The substantial flaws of controlled studies have recently been quantified in an extensive review of neuroleptic research, the results of which further erode confidence in the efficacy literature. In 1998, British researchers analyzed 2000 controlled efficacy trials conducted over the past 50 years (Thornley & Adams, 1998). The authors rated research methodologies according to the quality of randomization, blinding procedures, and the handling of patient attrition. Studies included consisted of trials of older and newer neuroleptics.

In regard to overall quality of reporting, only 4% of the trials described how patients were assigned to experimental versus control groups; only 22% of the studies described blinding procedures; and only 42% of the trials described treatment withdrawals. The latter is highly significant because it speaks directly to the feasibility of treatment compliance, side-effects of the drugs, and physiological and psychological fallout resulting from placebo washout. Astoundingly, only 1% of the studies were considered of good quality.

Significantly, the duration of each trial was very brief. Whereas only 19% of the trials lasted for more than six months, more than half (54%) lasted six weeks or less. Considering the complex nature of schizophrenia and the amount of time it generally takes for people to show improvement (e.g., WHO studies), short trials are extremely non-representative of real-world treatment and clinical course. Furthermore, for a drug to be approved by the FDA and to be considered useful in general, it has to be shown to be not only efficacious, but safe for human consumption. Most central to safety, the very brief length of these studies makes it nearly impossible for drug trial researchers and the FDA to fully assess the inevitable short-term, but specifically long-term safety of neuroleptic drugs. This is highly problematic because it is common psychiatric practice to use neuroleptics as 'maintenance medications'. That is, based on the general biopsychiatric belief that schizophrenia is a degenerative, incurable, chronic brain disease, neuroleptics are used for many years. In this sense, the brief drug trials do not approach a realistic assessment of long-term physiological and emotional side-effects.

In sum, Thornley and Adams (1998) conclude, "The consistently poor quality of reporting is likely to have resulted in an overoptimistic estimation of the effects of [neuroleptic] treatment" (p. 1184). In response, Jackson (2005) writes of the accumulated neuroleptic effectiveness evidence,

> When, on rare occasion, the results of long-term…studies have appeared, the research has consistently supported the view that antipsychotics do more to impede, rather than facilitate, the recovery and longevity of many patients... Moreover, the low quality of the clinical trials have pervaded the psychiatric

> literature has contributed to a body of evidence…whose validity and clinical relevance remain contestable (p. 191).

Even with the pro-drug biases inherent in the FDA and non-FDA trial methodologies, the performance of new (and old) neuroleptics has been generally unremarkable (Stip, 2002). Despite the serious methodological flaws of the typical RCT, I will discuss the most recent meta-analyses and other studies to more clearly illuminate the strengths and weaknesses of the newer, atypical neuroleptics.

Chakos and colleagues (2001) investigated the comparative utility of the atypicals in a twelve-study meta-analysis of treatment-refractory schizophrenia and concluded, "Efficacy data for other second-generation antipsychotics in the treatment of patients with refractory schizophrenia were inconclusive" (p. 518). Considering that studies included in the meta-analysis suffer from the pro-atypical neuroleptic biases just presented ('superdoses' of older drugs, placebo washout, LOCF, etc.) it is particularly informative that the results still did not favor the newer drugs. Furthermore, Chakos et al. (2001) identified a positive association between trial duration and drug efficacy, with longer study periods (i.e., a scenario more comparable to day-to-day psychiatric practice) *eroding* the few observed differences found between the older and newer drugs. All in all this meta-analysis refutes the superiority of the newer medications.

Geddes and colleagues (2000) report results of a far larger meta-analysis (n = 12,649) exploring the comparative value of the older and newer neuroleptics. Their results run contrary to the mainstream perception that atypicals are more efficacious and easier to tolerate in regard to side-effects. The authors conclude,

> There is no clear evidence that atypical antipsychotics are more effective or are better tolerated than conventional antipsychotics. Conventional antipsychotics should usually be used in the initial treatment of an episode of schizophrenia unless the patient has previously not responded to these drugs or has unacceptable extrapyramidal side effects (p. 1371).

These meta-analytic results have been replicated in prospective studies involving relatively chronic schizophrenic patients (Rosenheck et al., 2004), as well as patients earlier in their course of illness (Lieberman et al., 2003).

In a prospective, 12-month, multi-site drug trial comparing the atypical olanzapine with the typical haloperidol, Rosenheck and colleagues (2004) found no advantages for the newer drug in regard to treatment compliance (i.e., tolerability of side-effects), psychotic symptoms, the development of extrapyramidal symptoms, improvement in interpersonal relationships, employment earnings, or overall quality of life. They did find that olanzapine reduced incidence of akathisia,[5] but that these lower risks were offset by the significant weight gain, the development of diabetes, and treatment costs associated with olanzapine.

Lieberman et al. (2003) conducted a multi-site trial of olanzapine versus haloperidol in the treatment of recent onset schizophrenia. Despite significant methodological confounds favoring the atypical olanzapine, the authors still failed to detect any significant differences between the two medications. One outcome that was surprising was the high incidence of parkinsonism and akathisa among the olanzapine

[5] Akathisia is a feeling of inner restlessness that results in a constant urge to move. While one cannot keep still when afflicted with akathisia, the movements are voluntary. This is distinct from the involuntary movements associated with tardive dyskinesia. These and other side-effects associated with exposure to neuroleptics will discussed in greater detail below.

participants. This was an unexpected finding because the newer drugs are supposed to be less dangerous in regard to extrapyramidal side effects.

Perhaps in response to the many flaws of the RCT efficacy trials, the National Institutes of Mental Health (NIMH) is currently funding the largest-to-date, multi-site drug study to determine the effectiveness of atypical versus typical neuroleptics in naturalistic settings (e.g., community clinics). The naturalistic, *effectiveness* design will allow for longer periods of observation, less exclusion criteria, and will be far more generalizable to day-to-day practice compared to the RCT *efficacy* trials (for a discussion of the difference between effectiveness versus efficacy in treatment outcome research see Hilsenroth, Ackerman, Blagys, Baity, & Mooney, 2003). Also, the un-ignorable side effects observed by real-world practitioners belies the supposed safety of these drugs as reported in very brief drug trials and has further motivated a more ecologically valid, effectiveness trial. This ongoing study is known as the Clinical Antipsychotics Trial of Intervention Effectiveness (CATIE).

CATIE: The Largest Naturalistic Study of Neuroleptics to Date

Lieberman and colleagues (2005) report the results of Phase I of CATIE that examined the comparative effectiveness of atypical versus typical antipsychotic medications. Their findings further refute the widely asserted proclamations that atypicals are actually more effective and have fewer side effects compared to older drugs.

Lieberman et al. (2005) report no significant differences in the effectiveness of typical versus atypical drugs (n = 1432). As a measure of outcome they used early discontinuation due to intolerable side effects (the maximum length of Phase I was set at

18 months). It is important to not overlook the assumptions inherent in using early discontinuation as an outcome measure. Inherently, this assumes that simply staying on a medication indicates positive outcome, rather than measuring subjective or other clinically-observed change variables. Nonetheless, early discontinuation due to side effects does illuminate, at least partially, the feasibility of older and newer neuroleptics. The authors report mean early discontinuation rates across all drugs was an astoundingly high 74%. That is, only one in four participants was able to tolerate the drug for the entirety of the study period. The older drug, perphenazine, was *not* outperformed by any of the newer, far more expensive atypicals used with the greatest frequency in current clinical practice. The authors acknowledge that there is nothing unique about this finding because it is actually "consistent with…previously observed" outcomes found in smaller trials (pp. 1217-8). They conclude that the use of typical and atypical drugs exhibit "substantial limitations" (p. 1218). Yet, the authors do not offer any alternative other than to inform "patients, families, and policymakers [to] evaluate the trade-offs between efficacy and side-effects" (p. 1222). This recommendation is made despite the admission that, "The results of [this and other] studies of the effects of treatment on cognitive impairments and mood symptoms have been inconclusive…and incompletely explored" (Lieberman, 2005, p 1210).

The authors of this paper fail to recommend that alternative approaches should be considered, despite available evidence indicating the safety and effectiveness of psychotherapeutic interventions (discussed below). Further, the fact that Lieberman et al. (2005) make conclusive remarks regarding the effectiveness of drugs based on approximately 25% of the subjects who managed to not drop-out speaks to their seeming

unwillingness to consider the overall danger and ineffectiveness of these treatments. After all, if three of four people stopped taking penicillin due to its intolerable side effects during WWII would the U.S. government have considered this an effective treatment for battle wounds, especially if viable alternatives were available? Such a scenario would be highly unlikely. Yet, when it comes to the neuroleptic treatment of schizophrenia, this level of ineffectiveness and physiological and emotional intolerableness is not merely acceptable, but deemed preferable.

In conclusion, Gabbard and Freedman (2006) refer to Harvard social psychiatrist Leon Eisenberg's cautionary comments on biopsychiatry's neurobiological reductionism and concordant mandate for a medication-only approach in our field. They note,

> These directions recall Leon Eisenberg's prediction that when the ultimate neurobiological treatment for schizophrenia is someday devised and everyone is marveling over the results on the computer monitor, there may be only one psychiatrist left who will remember to ask the patient, 'How do you feel' (p. 183).

Review of the most common side-effects of neruoleptic treatment is in order to further assess the effectiveness and safety of neuroleptic medications.

Side-Effects of Neuroleptic Medications

The neuroleptic toxicity literature is rapidly growing as the dangerous short- and long-term side-effects of these chemical agents become increasingly clear to psychiatrists, consumers, their loved ones, as well as concerned patients' rights advocates. While this review is not intended to be exhaustive, it will provide an

illustrative sampling of the most prevalent hazards associated with neuroleptic use. The interested reader would be well-served to read Jackson (2005) as well as Owens (1999) for more detailed accounts.

The most commonly recognized side-effects of neuroleptic use are known as 'extrapyramidal side-effects' (EPS). EPS are the various movement disorders suffered as a result of ingesting neuroleptic dopamine antagonists. The best known EPS is tardive dyskinesia (TD) which results in involuntary, irregular muscle movements usually, but not limited to the facial region. For example, the patient may have facial tics, roll the tongue, lick the lips, and/or have trembling hands. Although TD might be managed or minimized by reducing the medication dosage or by changing type of medication, the symptoms may persist for long periods and often permanently, even after neuroleptics are decreased or even discontinued (Breggin, 1991, 1997; Jackson, 2005).

Some forms of TD involve damage to the respiratory system which results in hyperventilation, abnormal vocalizations such as grunting, and violent chest contractions that can result in rib fractures (Hayashi, Nishikawa, Koga, Uchida & Yamawaki, 1996; Leung, Chung, Kam, & Wat, 2000). TD has regularly been empirically associated with an exacerbation of crippling negative symptoms such as social isolation and anhedonia (Waddington & Youssef, 1986), as well as positive symptoms (Miller et al., 2005).

Other common EPS include acute dystonia which is an abnormal drug-induced state of either excessive or inadequate muscle tone. There are many forms of dystonia disorders which cause involuntary movements and prolonged muscle contraction resulting in twisting body motions, tremor, and abnormal posture, all of which are very

painful for the sufferer. These movements may involve the entire body or only an isolated area (Jackson, 2005).

Many patients develop symptoms that mimic those of people suffering from Parkinson´s disease (Breggin, 1991). The symptoms include tremors, rigidity, temporary paralysis, reduced facial expression, shuffling gait and extreme slowness of movement. These symptoms usually appears in the first few days and weeks of neuroleptic medication administration.

Akathisia is a serious condition associated with the use of neuroleptic medications and characterized by an internal sense of extreme agitation and motor restlessness (Jackson, 2005). Patients often describe their experience as being defined by an absolute inability to resist the urge to move. The most widely observed form of akathisia involves pacing and an inability to sit still. This side effect is often very distressing to the patient and reduces his or her ability to perform every day tasks. This bizarre consequence of neuroleptics also invites odd stares and exacerbates an already significant level of social alienation.

A potentially fatal side-effect of neuroleptic treatment is known as neuroleptic malignant syndrome (NMS; Jackson, 2005). NMS includes diffuse muscle rigidity, tremor, high fever, labile blood pressure, cognitive dysfunction, and autonomic disturbances. This condition can be sudden and often occurs early in the course of treatment. However, it has also been documented to occur after months and years of neuroleptic medication use.

In a recent review of the EPS literature, Glazer (2000) summed evidence from a cohort of 362 patients. Glazer found a positive relationship between cumulative duration

of neuroleptic treatment and EPS, thereby replicating previous reviews (e.g., Glazer, 1992). It is widely assumed that EPS develops only after years of exposure to neuroleptics. However, Jeste and colleagues (1999) found that older patients are particularly susceptible to EPS, even when exposed to neuroleptics for a short period (e.g., within the first month of treatment). In regard to patients within a more representative age range (mean age of 28), Oosthuizen and colleagues (2003) have shown that even minimal exposure to neuroleptic treatment can cause substantial EPS in a relatively short period of time (i.e., at one year follow-up).

The emergence of the newer atypical neuroleptics has been accompanied by an assumption that these newer drugs reduce the risks of EPS. The summed research suggests that approximately 50-90% of all patients treated with the older typical neuroleptics will experience some form of EPS. In contrast it has been estimated that as many as 20-50% of atypical recipients will suffer the consequences of drug-induced EPS (Malla et al., 2004). Two recent studies report *no* significant differences between the newer and older drugs in regard to neurotoxicity (Bonelli et al., 2005; de Leon, in press). Therefore, while the reduced risks are questionable, it is clear is that the incidence rates of EPS associated with the newer atypical drugs are still quite substantial.

If there are reduced risks of EPS enjoyed by the atypical neuroleptics, they are potentially outweighed by the unique tendency of the atypicals to contribute to obesity (Meyer, 2001), cardiovascular disease (e.g., Osby et al., 2000), diabetes (e.g., Dixon et al., 2000) and dysmetabolic syndrome (e.g., McEvoy et al., 2005). The latter, dysmetabolic syndrome, is defined by clinical features that include obesity, abnormal density of lipoproteins, elevated fasting triglycerides, hypertension, and impaired fasting

glucose or overt diabetes mellitus. While diabetes represents a disease entity with a high risk for cardiovascular disease, dysmetabolic syndrome's cluster of physiological symptoms is deemed an even more dangerous precipitant to heart trouble.

McEvoy et al. (2005) found that compared to a matched normative sample, male and female schizophrenics exposed to atypical antipsychotics are respectively 138% and 251% more likely to have dysmetabolic syndrome. A staggering 41% of all schizophrenia patients in this sample developed dysmetabolic syndrome. In a 10-year longitudinal study, Henderson and colleagues (2005) report similar results specifically related to the atypical drug clozapine. In addition to these dymetabolic syndrome studies illuminating the greater risks neurolepticized schizophrenics face in regard to the *gradual* development of heart disease, Sabine and colleagues (2004) report neuroleptic use is also associated with sudden cardiac death, even at low doses.

In regard to sexual dysfunction, typical and atypical drugs have been shown to produce prolactin imbalances regularly linked to infertility (e.g., Alexiadis, Whitehorn, Woodley, & Kopala, 2002). Older and newer neuroleptics have also been linked to decreased libido, erectile and ejaculatory dysfunction, gynecomia, and amenorrhea (e.g., Bobes et al., 2003).

Recent studies have replicated results that have found a positive association between neuroleptic use and mortality. For example, Healy and colleagues (2006) present data indicating the greater risks of suicide faced by schizophrenics treated with typical *and* atypical neuroleptics compared to non-medicated schizophrenics. The investigators compared detailed asylum records of psychotic admissions in Victorian North Wales (1875-1924), within the same geographic locale to similar records kept between 1994 and

1998. The authors found a startling result: patients treated with the latest antipsychotic drugs had a 20-fold increased risk of suicide compared to those treated without drugs in the Victorian era. Data from the British Medicines Healthcare Regulatory Agency (Alliance for Human Research Protection, 2006) confirm that antipsychotics (as well as antidepressants) are implicated in the greatest number of suicides and suicide attempts in the United Kingdom. These data further suggest the decline in mental health care associated with medication-only treatments.

Joukamaa and his Finnish colleagues (2006) also found an association between typical neuroleptic use and mortality in schizophrenia in their 17-year longitudinal study. They found that schizophrenic individuals in general have a 1.29-to-1 relative risk of mortality compared to normal controls. Thus, just having schizophrenia puts you at risk above the general population. This is neither surprising nor unexpected. However, following adjustment for age, gender, somatic diseases and other potential risk factors for premature death, the authors found that "[t]he number of neuroleptics used at the time of the baseline survey showed a graded relation to mortality" (p. 122). Specifically, the relative risks for people with schizophrenia taking one, two, or three or more neuroleptics at baseline were (95% CI) 2.97, 3.21, and 6.83, respectively. The association remained stable throughout the 17-year follow-up period. According to these results, neuroleptics may be more than ineffective, they may be dangerous. These findings replicate previous studies that similarly found an association between polypharmacy and increased mortality rates with both typical and atypical neuroleptics (Bralet, Yon, Loas, & Noisette, 2000; Morgan et al., 2003; Osby, Correia, Ekbom, & Sparen, 2000; Reilly, Ayis, Ferrier, Jones, & Thomas, 2002; Wallington, Youssef, & Kinsella, 1998).

One of the predominant features of schizophrenia is disorganized thinking. As such, medications meant to treat schizophrenia should help rather than harm cognition. However, this is not always the case. Kasper and Resinger (2003) found that neuroleptics actually contribute to cognitive impairments rather than ameliorate them. Minzenburg and colleagues (2004) found that neuroleptic medications negatively impacted visual attention, general intelligence, and short-term declarative memory after controlling for psychopathology and cognitive ability. A variety of studies document the relationship between neuroleptic use and executive functioning and working memory (Gilbertson & van Kammen, 1997; Mehta, Manes, Magnolfi, Sahakian, & Robbins, 2004; Papageorgiou et al., 2003; Sweeney, Keilp, Haas, Hill, & Weiden, 1991; Vitiello et al., 1997), sustained attention and arousal (Cassens, Inglis, Appelbaum, & Gutheil, 1990; King, 1994), reaction times (Ridout & Hindmarch, 2003), information processing (Brebion, Amador, Smith, & Gorman, 1998), fine motor coordination (Cleghorn, Kaplan, Szechtman, Szechtman, & Brown, 1990), temporal processing (Rammsayer, 1997), latent inhibition (McCartan et al., 2001), and procedural learning (Scherer et al., 2004).

Clearly, the physiological and cognitive side-effects of neuroleptic treatment are serious consequences needing to be factored into our general treatment approach to schizophrenia, along with the fact that there is no empirical evidence documenting any deleterious side-effects of psychotherapeutic interventions. Therefore, it is not unreasonable to suggest that psychotherapy need only fare equal to, or of course better than, neuroleptics for treating schizophrenia since the neuroleptic side-effects place the medications at a serious clinical disadvantage. In this vein, I will now explore the empirical effectiveness of psychodynamic psychotherapy.

Section IV: The Effectiveness of Psychodynamic Psychotherapy of Schizophrenia

The "tragedy" of schizophrenia in our modern mental health care system is that we know of treatments that work – namely, psychodynamic psychotherapy - but very rarely offer adequate training or resources for this approach (Karon, 2003, p. 89). Although there are a number of psychodynamic perspectives that inform the treatment of schizophrenia, it is possible to extract a number of common threads that bind these approaches together. Some of the more prominent psychodynamic clinicians, theorists, and researchers contributing to these core ideas include Arieti (1974), Boyer, Giovacchini & Hoedemaker (1967), Fromm-Reichmann (1950), Karon & Vandenbos (1981), Searles (1960, 1965), Will (1968) and Sullivan (1953, 1962).

To start, what psychodynamic psychotherapy of schizophrenia is *not* is classically psychoanalytic. That is, clinicians working with this severe population do not have patients lying on a couch, facing away from them, or promote regression via a general stance of clinical abstinence and neutrality; nor do therapists aggressively provide genetic or defense interpretations as would be expected in the treatment of a neurotic-level patient. This is an important point because it is generally assumed by psychiatrists and non-psychodynamic therapists that there have been no amendments to the original Freudian approaches/tenets in regard to schizophrenia. This misunderstanding has been most recently repeated in the PORT study's out-of-hand rejection of the use of psychodynamic therapies based on the faulty notion that such treatments promote "regression and psychotic transference" (Lehman & Steinwachs, 1998, p. 8). Since the PORT was convened to review the clinical literature to create treatment

recommendations for practicing psychiatrists and psychologists this misperception is extremely significant. Fortunately, Ver Eecke (2003), Gottdiener and Haslam (2003), and Silver (2003) each provide a clear, theoretical and empirical-based critique of the mistaken conclusions of the PORT.

The contemporary psychodynamic approach to schizophrenia is more accurately depicted to include the following core tenets:

1) A patient has a narrative that is highly relevant to his or her psychological suffering. It is imperative to consider the role of the patient's early environment/experiences as a partial contributor to the patient's interpersonal difficulties, preferred defensive style, and overall symptomatic distress. Another way to express this idea is to consider a patient's life events, both positive and negative, as contributors to each patient's idiosyncratic (and often psychotic) attempts to adapt to the complexities of our social, vocational, and interpersonal world.

2) The therapist should listen seriously and respectfully to each individual's subjective affective and cognitive experience, no matter how seemingly bizarre or 'irrational'.

3) The therapist should attempt to understand the symptoms of schizophrenia as symbolic communications of unresolved and very distressing interpersonal dilemma(s) stemming from a core confusion in which "one cannot maintain a border between where [s/]he ends and where objects [i.e., other people] and their internalized representations start" (Volkan, 1995, p. 27). These object relations, that is, one's

internal representations of self/other and external patterns of relating, are believed to be exacerbated by, and a product of, an internal world full of split (i.e., conflicting affect states held apart from each other) or even fragmented self- and other-images often saturated with powerful, un-neutralized affect states.

4) The internal situation referred to in #3 results in a fairly constant panic-level anxiety that leaves the schizophrenic patient feeling as if he or she is in continuous existential danger. This internal dread is often externalized and obscurely communicated in delusional and hallucinatory symptoms often of a paranoid nature. Psychodynamic theory understands these psychotic symptoms, as well as acute withdrawal from social relations, as defenses (or, *poorly adaptive solutions*) used to protect the schizophrenic patient from powerful internal affect states that are psychotically distorted as external threats.

5) Due to its understanding of the intensity of affects underlying defenses in schizophrenia, psychodynamic therapy is designed to assist patients develop more adaptive defenses/solutions, as well as conscious insight to help patients more realistically express their internal experiences, and "convert raw anxiety into sublimated cohesive" self understanding (Silver, Karon, & Koehler, in press, cited in Silver, 2003, p. 330).

6) Karon and Vandenbos (1981) outline three essential functions of the in-the-room relationship. First, the therapist-patient relationship should provide sufficient protection and gratification to overcome the

conscious resistance the patient has to engaging the therapeutic process. Due to a history of very troubled interpersonal relationships, the therapist needs to present him- or herself as "a strong, protective, more gratifying figure than [s/]he would be in the therapy of [healthier, non-psychotic] patients" (p. 145). Second, that which transpires verbally and non-verbally in the room between the schizophrenic patient and therapist is highly relevant to his or her troubling patterns of internal/external interpersonal experiences. Thus, within the therapeutic relationship a schizophrenic patient, similar to any other patient, "relives feelings and experiences from the past [i.e., transference]…thus providing an enormous source of information as to what [the patient's] life history and problems were truly like" (p. 146). This type of information is intended to allow for slow resolution/understanding of a particular patient's core anxiety laden conflicts. Third, the therapist's sustained curiosity, warmth, desire to help, and refusal to accept therapeutic failure should function as a reliable model for identification. Identifying with the therapist in this way is intended to modify the unusually harsh super-ego's common to schizophrenia patients.

7) Psychodynamic theory holds that under enough stress, especially in the context of a limited support system, anyone is susceptible to degrees of psychoticism.

8) Psychodynamic approaches assert that schizophrenia is *not* solely a 'chemical imbalance' nor is it a degenerative brain disease. Rather,

schizophrenia is a complex and extreme biopsychosocial reaction to troubling real-world experiences.

9) Therapists working with schizophrenic patients must have a high tolerance for confusion and frustration and be free of the need to derive narcissistic gratification from the patient's therapeutic efforts and/or success (Bachmann, Resch, & Mundt, 2003).

Beyond psychodynamic psychotherapy there is growing evidence that other modalities, such as cognitive-behavioral treatment (e.g., Turkington et al., 2006), residential-based milieu treatment (e.g., Mosher, Vallone, & Menn, 1995), social learning treatment (e.g., Paul & Lentz, 1977), Open Dialogue treatment (Seikkula, Alakare, & Aaltonen, 2000; Seikkula et al., 2003) and family treatment (e.g., Schindler, 1980) are effective alternatives adjunctive to or independent of medication treatment. For the sake of brevity and because it reflects the empirical component of this dissertation project, this review will focus solely on the empirical literature exploring psychodynamic and psychodynamically-informed treatments.

Research Investigating the Psychodynamic Psychotherapy of Schizophrenia

Due largely to the drug-bias in the modern mental healthcare system there have been very few randomized studies that explore psychotherapy without adjunctive medication (Jackson, 2005). However, of the four that do exist the overall results indicate the promise of psychodynamic work with schizophrenia, despite significant limitations impacting two of these studies in particular (for a review see Karon, 1989).

Carl Rogers and colleagues (1967) performed a randomized clinical trial comparing person-centered therapy to a neuroleptic-only treatment approach. Although person-centered therapy is not psychodynamic, many of its core tenets are deeply influenced by and comparable to a contemporary psychodynamic approach – especially in regard to the treatment of the more severely ill. For example, Rogers' approach, similar to any psychodynamic approach, focuses a great deal of attention on the in-the-room relationship. Rogers et al. write, "…the most essential ingredient for change will be found in the attitudinal qualities of the person-to-person relationship (p. 92).

In this study, there were 16 early phase schizophrenics and 16 chronic schizophrenics. After matched pairing, the schizophrenic patients were randomly assigned to each of two treatment groups: psychotherapy-only and neuroleptic-only. Therapy was given twice a week for up to 2.5 years. Outcome was assessed by blind reviewers, the therapists and the patients themselves. All significant findings favored the psychotherapy group including improved interpersonal relationships, enhanced capacity to openly experience and express their emotions, and better ability to "face their environment and themselves" (cited in Irwin, 2004b, p. 100). Importantly, the psychotherapy group spent an average of 112 fewer days in the hospital following the end of the psychotherapy phase of the study. Therapeutic alliance defined by genuineness, empathic attunement, and affective congruence was positively related to outcome. Psychotherapy patients who did not experience as positive a therapeutic relationship did not fare as well by the study's end, suggesting the important mutative impact of the patient-doctor alliance. Limitations of the study included small sample size and lack of a placebo group. Strengths included treatment fidelity which was measured and rigorously

monitored. That is, the research and clinical supervisors of this study were committed to the psychotherapy component of the study.

May and colleagues (1976, 1981) randomized 228 early episode individuals suffering with schizophrenia to one of five treatments: psychodynamic psychotherapy without medication, psychodynamic psychotherapy with medication, medication-only, ECT, or milieu therapy. All groups received milieu therapy and therefore the milieu group served as the control cohort. Psychotherapy was provided for two years at two times per week. Outcome measures included number of days hospitalized, social competence, and global functioning derived from patient interviews, families, social workers and hospital records. After two years the only significant difference found was in number of hospital days, favoring the medication-only group; after 3, 4, and 5 years there were no significant differences found. Karon (1989) extensively critiques the quality of psychotherapy provided in this study based on the supervisors' reported lack of experience and negative expectations of the utility of psychotherapy for schizophrenia. Even with these limitations handicapping the psychotherapy patients, the medication-only individuals, in general, did not outperform them. Other limitations also include the lack of a placebo group and lack of blinding for most assessments (which likely deleteriously impacted the psychotherapy group due to negative treatment expectations held by the research team).

Grinspoon and colleagues (1972) randomized 41 people diagnosed with chronic schizophrenia into three groups: 21 remained in the hospital where they continued neuroleptics; 10 were given placebo for 13 weeks and then restarted on neuroleptics; and 10 were given placebo for 13 weeks and then provided with psychodynamic

psychotherapy. At five years two trends (p = .07) favoring the psychotherapy patients were detected, including decreases in psychotic symptoms and better capacity to adapt to life outside of the hospital. Perhaps due to their inability to shed their drug bias the researchers referred to the improvements in the psychotherapy group as "an artifact" (p. 1126). Karon and Vandenbos (1981), Karon (1989), as well as Irwin (2004b) outline substantial limitations of this study. First, over half of the psychotherapy patients had previously received at least one round of ECT, insulin shock, or both. As Karon and Vandenbos write, "Psychotherapists experienced with treating schizophrenic patients have doubts about attempting psychotherapy with patients who have had ECT or insulin comas [due to the organic damage resulting from such intrusive treatments]…No serious study of psychotherapy would include such patients…" (p. 380). Beyond this, the therapists used in this study had very little experience with treating schizophrenia or patients from the cultural and socioeconomic group of those included in the project. In fact, retrospectively, most of the therapists in this study explicitly expressed doubt about the quality of the psychotherapy provided in this study (see Karon, 1989). Lastly, the drug washout time of 13 weeks is not considered adequate (Breggin & Cohen, 1999), thereby making this a study of drug withdrawal as much as anything else. Despite these limitations negatively impacting the psychotherapy patients, these individual fared relatively well with the aid of psychodynamic psychotherapy.

Karon and Vandenbos (1981) completed the last randomized trial of psychodynamic psychotherapy of schizophrenia of the 20th century in the late 1960's. It is this study from which the current project's data are derived. As reported above, at the conclusion of their 20-month investigation the researchers found that patients receiving

psychotherapy significantly outperformed individuals receiving medication-only on three of four instruments assessing patients' thought disorder, on a blindly rated clinical status interview, and days spent in the hospital due to mental dysfunction. On the fourth measure of thought disorder, differences between the psychotherapy and medication-only patients approached significance (p = .07). Individuals of the medication-only group did not fare better in any area assessed. More specific details of Karon and Vandenbos' investigation will be explored in the 'Methods' section below.

Gottdiener & Haslam (2002) recently conducted the first comprehensive meta-analytic review of individual psychotherapy of schizophrenia, including in their analysis between- and within-groups designs as well as psychotherapy designs with or without adjunctive medication. These important inclusions have been missing from past meta-analyses investigating schizophrenia outcome research (Cormac, Jones, & Campbell, 2002; Malmberg & Fenton, 2001; Mojtabai, Nicholson & Carpenter, 1998; Smith, Glass, & Miller, 1980). Gottdiener and Haslam examined studies exploring cognitive-behavioral, psychodynamic, and non-psychodynamic supportive therapies.

Gottdiener & Haslam (2002) report an overall corrected effect size of r = .36 across the three treatments. Psychodynamic (r = .39) and cognitive-behavioral (r = .41) treatments with and without medications were found to be equally effective. Non-psychodynamic supportive therapy exhibited a relatively smaller effect (r = .27; no significance test was conducted). Despite the PORT recommendations (Lehman & Steinwachs, 1998) and other empirically unsupported admonishments (e.g., Drake & Sederer, 1986) against the use of psychodynamic therapy, insight-oriented therapy fared quite well relative to other psychotherapy modalities.

As the entirety of this dissertation project has made clear until this point, there is a strong biopsychiatric belief that neuroleptics are the most effective form of treatment for schizophrenia. However, Gottdiener and Haslam (2002) report that use of medications did not impact effect sizes at all. That is, when medications were used with psychotherapy the effect size was identical to when medication was withheld (r = .31). Of course, the participants being treated without medications were free of the various side-effects caused by these potent chemical agents. In sum, similar to the few randomized trials reported earlier, this meta-analysis provides additional evidence that psychodynamic psychotherapy is safe, effective, and without doubt worthy of additional empirical attention.

In concluding this introduction, based on 1) the philosophically and empirically untenable degenerative and psychotoxicity assumptions; 2) significant flaws in the FDA approval process and the RCT design in general for use in schizophrenia research; 3) the empirically documented ineffectiveness of typical and atypical neuroleptic treatments; 4) severe side-effects of neuroleptic treatments; and 5) the effectiveness of psychodynamic psychotherapy, one is left seriously reconsidering the wisdom of a medication-only treatment approach in lieu of existing alternatives. I will now continue with the empirical portion of this dissertation project.

II. METHODS

Section V: Pre- to Post-Treatment Change in the Object Relations of Schizophrenic Patients

As mentioned, the experimental design utilized in the original study conducted by Karon and Vandenbos (1981) called for the selection of schizophrenic patients to be assigned on the basis of a random number table to one of three treatments: 1) psychotherapy without medication (Psychotherapy-only, A); 2) psychotherapy with adjunctive medication (Mixed, B); and 3) routine hospital treatment consisting primarily of phenothiazines (i.e., typical neuroleptics; Medication-only, C). The adjunctive medication provided to individuals of the Mixed group (B) was discontinued after the first few weeks of the study. Overall, 36 patients were evenly distributed among the three treatment groups. The small number was necessary in order to be careful about gathering detailed and rigorous data throughout the 20-month study. The small numbers in the psychotherapy groups (A and B) also allowed for more efficient 'quality control' of psychotherapeutic treatment by experienced supervisors. This was particularly important because, as mentioned earlier, the quality of psychodynamic therapy provided in past randomized studies has been seriously called into question in regard to the quality of training the therapists had, the therapist's specific experience with psychotic patients, and researcher bias (Karon, 1989). Due to incomplete data available for the Mixed group (B), this cohort has been removed from the current study. Although this is an unfortunate loss of potentially useful and interesting data, the primary and unique aspect of the experiment is the assessment of a completely non-medicated group. That is, while there are studies exploring mixed treatment approaches (see for a review Gottdiemer & Haslam, 2002),

there are as far as could be determined no published U.S.-based studies in the last 25 years that utilized a fully non-medicated group.

Clinical Participants

All inpatient admissions to the Detroit Psychiatric Institute (DPI) were reviewed for possible inclusion in the project. Evaluation for selection was solely in terms of meeting the selection criteria discussed below. Selection was made by using the same criteria and research personnel throughout the entirety of the project. Patients were not selected by regular hospital staff or with an eye to whose patients they might become. The intent to obtain schizophrenic patients with no organic pathologic condition or previous hospitalizations required adequate case histories and thorough medical examinations before selection into the project. This process required two weeks for each case. Patients were not assigned to treatment groups until there were three patients who had completed preliminary medical and diagnostic examinations so that the 'set' could then be randomly assigned. If a patient was discharged before assessment was complete that patient was replaced as a potential project patient by another patient who met the requisite criteria and was still hospitalized.

The admission rate to the DPI ward was approximately 5,000 patients per year throughout the 1960s (Karon & Vandenbos, 1981). Hence, in any given week a pool of about 100 potential research patients would be reviewed using the initial criteria. However, roughly two-thirds of these patients were discharged within two weeks, therefore precluding completion of the extensive medical and diagnostic assessment. As a result, the actual pool of potential research subjects was about 35 per week. Only one set

of three patients was selected in a given week. All the patients were selected within a 4-month period. Any differences in patient characteristics reflect possible week-to-week fluctuation in admissions and therefore were well within the bounds to be expected from random variation.

Patients utilizing services at DPI were primarily poor, inner-city African-Americans. The initial selection process was to select acutely ill (i.e., not chronic) and clearly schizophrenic patients. Six criteria were mandatory for inclusion. The first and most important criterion was that the patient needed to be unquestionably schizophrenic. The diagnoses of patients in the study were made by two independent research personnel (clinical psychologists) with considerable experience in the treatment and diagnosis of schizophrenia, neither of whom was connected with treatment of the clinical patients. *DSM-II* criteria and medical screening for organic illnesses were utilized to guide the diagnostic process. Both experts had to agree with the schizophrenia diagnosis for the patient to be included. Further, the DPI Ward Chief as well as an independent psychiatric resident also had to concur with the diagnosis for the patient to be included in the project. Criteria two through six included: 2) onset of blatant psychotic symptoms within 3 months prior to admission; 3) first admission; 4) no history of ECT or insulin shock treatment; 5) no organic brain damage; and 6) no history of alcoholism or drug addiction.

Of particular relevance to this sample, Dunham (1965) found that the median time between onset of blatant psychotic symptoms and the first presentation to treatment was 34.5 months for poor, inner-city African-Americans. Since these patients tended to be ill for some time prior to admission at DPI, criterion three (i.e., “first admission”) is misleading as these patients were more severely impaired and chronic compared to other

first admissions derived from less impoverished communities. Severity of illness was best observed on the pre-treatment scores on the Drasgow-Feldman Visual Verbal Test (VVT; Feldman & Drasgow, 1951) for which 29 of 35 patients scored at or below the norms for chronic schizophrenic individuals. Importantly, VVT normative data were drawn from schizophrenic patients hospitalized for 3 or more years. Thus, across groups, these poor, generally African-American patients exhibited a very severe level of chronicity atypical for first admission schizophrenics. Exacerbating the severity was that initial screening failed to reveal 11 previous hospitalizations (Psychotherapy-only = 4, Mixed = 2, Medication-only = 5). Patients and their families reasoned correctly that a previous hospitalization leads to worse treatment in general and therefore they actively concealed this information.

Despite the attempt at careful medical screening, some medical problems were not accurately revealed during the initial screening process. Four dramatic instances occurred. Two catatonic patients died of embolisms. The first died before randomization had occurred and was therefore replaced without jeopardizing random assignment. The second patient (a female in the Psychotherapy group treated by a trainee) died following group assignment. She was therefore not replaced as such an assignment would not be purely random. A female patient being treated by the Psychotherapy group supervisor (Karon) was eventually diagnosed with multiple sclerosis despite being originally cleared by the neurology and internal medicine services. It was only after psychotherapy improved her thought disorder, reality testing and interpersonal distress that this patient's remaining motor impairments (this patient had a gait all along) were properly understood as symptomatic of multiple sclerosis. Since the patient was indeed clearly schizophrenic

and the two sets of symptoms followed independent courses, this patient was included in the data. Karon and Vandenbos (1981) found that analyses were not significantly altered by exclusion of this patient and so she will remain in the proposed study as well.

Another patient (a male in the Psychotherapy group treated by a trainee) initially denied a history of drug abuse. However, during the course of psychotherapy this patient's verbalizations revealed a possible history of addiction. Subsequent collateral contacts with the patient's girlfriend and family revealed that for many years he had taken seconal, Dexedrine, and nutmeg in daily irregularly large doses sufficient to produce brain damage. This patient was excluded from the data analyses in light of the inappropriate selection for the project. A third patient was excluded from the treatment because of grossly unprofessional staff interference described in detail in Karon and Vandenbos (1981). In sum, the Psychotherapy-only group lost three patients: one patient died from an embolism, one was excluded due to a previous addiction history, and a third was excluded because of gross staff interference. Therefore, the original analyses included nine subjects in the Psychotherapy-only group, and 12 in the Mixed and Medication-only groups (total $n = 33$). Thus, the proposed study utilizing only the pure groups will have a final n-size of 21 (Psychotherapy-only = 9; Medication-only = 12).

Assignment of patients to experienced and inexperienced therapists was on a rotation basis. Psychotherapy supervisors did not select which patients with whom they would work. Psychotherapy supervisors were assigned two research patients previous to assignment of patients to trainees. These cases served as training cases and were viewed on closed-circuit TV and subsequently discussed.

Therapists

There was one experienced supervisor for each of the psychotherapy treatment groups. Each supervisor ultimately treated four patients. 'Experienced', as used in this study, means approximately 10 years of experience specifically treating schizophrenic patients (i.e., not merely doing psychotherapy but treating schizophrenic patients). Both supervisors also had lengthy experience working with poor, inner-city African-American patients. The psychology interns and psychiatric residents had three months to one year of general practicum or residency experience before the initiation of the project. In addition to supplemental texts designed to inform (though not manualize) treatment, the trainees watched via closed circuit TV the beginning of the treatment of two patients by their respective group supervisor before they began to work with their own patients. To facilitate training, trainees' early sessions were viewed on closed circuit TV by the supervisor, as well as the other group trainees. In the latter phases of the study, conventional individual supervision sessions were held. Trainees were monetarily compensated throughout most of the study. At mid-project (approximately 12 months) payments were suspended due to an intra-professional rule change precluding students from accepting monetary compensation while matriculating. Trainees agreed to work for several months without pay until the project supervisors were able to reinstitute payments. It is possible that withheld payments may have affected motivation of trainee therapists and impaired the effectiveness of their performance during the period of withheld compensation. All project therapists were Caucasian, reflecting the ethnic composition of the hospital's professional staff and of the clinical psychology and medical students. Minority students were unavailable in the programs in which the

trainees matriculated. Medical and psychology students were evenly distributed among the psychotherapy treatment groups to minimize professional jealousies as a contaminating factor.

Explanation of Psychotherapeutic Treatment

Because the Mixed group will not be investigated in this proposed study, only the technique and theory of the Psychotherapy-only group will be presented. The Psychotherapy-only group used psychodynamic psychotherapy without medication. The derivative of psychodynamic therapy utilized with these patients was largely influenced by Karon's (1994; Karon & Vandenbos, 1981) interpretation of the theory and technique of Sullivan (1953, 1962) and Fromm-Reichmann (1950). Briefly, there are a few central tenets of Karon's approach largely in tune with the core psychodynamic principles discussed earlier. The first critical issue is how to come into meaningful contact with the patient. That is, at the very onset of treatment emotional contact must be established. Schizophrenic individuals are good at avoiding authentic contact with people, so in the initial sessions it is imperative for the therapist to establish his or her existence, desire to help, as well as convey understanding of the patient's confusing and frightening experience. The immense dread often experienced by the patient is diminished if the therapist provides attention, interest, and absolute seriousness in the meaning of the most frightening psychotic experience of the patient (e.g., projections, hallucinations, delusions). There are also two general principles of interpretation adhered to in this approach, both intended to generate insight promoting change.

The first principle is to interpret from the surface in the sense of exhausting reality factors and realistic explanations first. Few patients are willing to consider psychodynamic factors until commonsense explanations are found wanting. Driving this principle is the basic idea that a therapist interprets what he or she believes the patient can make use of at that time. The second general principle is that the therapist never does for a patient what the patient can do for him- or herself. An insight discovered by the patient is far more potent than a similar interpretation from the therapist. In other words, value the meaning-making processes inherent in a patient's tendency towards health. In terms of therapist activity, Karon and VandenBos (1981) assert that it is not *accurate* empathy or understanding that produces change in patients, but rather the therapist's concerted *attempts* at empathy and understanding (whether or not successful). Also, Karon's approach highlights the importance of the therapist's capacity to tolerate confusion, due not only to the often obscure and entangled thinking of the patient, but also because conveying confusion as a tolerable and all-too-human experience is an invaluable offering to a person suffering with schizophrenic processes. In sum, Karon's approach to psychotherapy with schizophrenic patients is to value and encourage the totality of patients' successful and troublesome encounters with self and world, to be tolerant and kind, create hope, to encourage self-understanding, and perhaps most of all, to be stubborn in not accepting therapeutic failure.

Measure

Social Cognition and Object Relations Scale (SCORS). Westen's (1995; Hilsenroth, Stein, & Pinsker, 2004) SCORS is a narrative-based object relations measure

designed to assess a variety of dynamic personality features beyond the overt presentation of the patient. The SCORS consists of eight clinician-rated dimensional variables that examine the affective and cognitive aspects of an individual's object relations. This multivariate approach allows clinicians to discern higher from lower areas of object relational functioning. Such an approach is clinically useful considering that people generally possess interpersonal strengths and weaknesses contingent upon the content of the schema certain social situations activate (e.g., emotional intimacy schema, authority schema). Each of the eight SCORS variables is scored on a 7-point anchored rating scale where lower scores (e.g., 1 or 2) indicate greater pathology and higher scores (e.g., 6 or 7) indicate greater psychological health.

The eight SCORS subscales include: 1) "Complexity of Representations" (COM) assesses the richness of a patient's representations of self and others, as well as his or her abilities to integrate both positive and negative attributes of self and others; 2) "Affect Tone of Representations" (AFF) assesses a patient's positive and/or negative expectations from others in relationships and how the patient describes relationships; 3) "Emotional Investment in Relationships" (EIR) identifies the patient's level of commitment and emotional sharing in relationships; 4) "Emotional Investment in Values and Moral Standards" (EIM) distinguishes between patients who show no remorse for selfish actions versus those who think about moral questions in genuinely compassionate and thoughtful ways; 5)"Understanding of Social Causality" (USC) assesses how well a patient understands why people do what they do; 6) "Experience and Management of Aggressive Impulses" (AGG) assesses a patient's ability to control and appropriately express aggression; 7) the "Self-Esteem" (SE) variable assesses the affective quality of patient's

self-representation; and 8) "Identity and Coherence of the Self" (ICS) assesses a patient's level of integration and goal-directed behavior.

Huprich and Greenberg (2003) and Stricker and Gooen-Piels (2004) provide an extensive review of previous SCORS research, therefore the following will be an illustrative rather than exhaustive presentation of the SCORS' reliability and validity. The SCORS has exhibited good to excellent inter-rater reliability when utilized to rate TAT narratives (Ackerman, Clemence, Weatherill, & Hilsenroth, 1999; Ackerman, Hilsenroth, Clemence, Weatherill, & Fowler, 2000; Fowler, Ackerman, Speanburg, Bailey, & Blagys, 2004), relational and self-statements expressed to clinicians in psychotherapy sessions (Peters, Hilsenroth, Eudell-Simmons, Blagys, & Handler, 2006), early memory narratives of adult clinical outpatients (Fowler, Hilsenroth, & Handler, 1995; Slavin, Stein, Pinsker, & Hilsenroth, in press), semi-structured interview data (Porcerelli, Cogan, & Hibbard, 1998), and stories elicited by the Picture Arrangement subtest of the WAIS-R (Segal, Westen, Lohr, & Silk, 1993).

Convergent validity has been demonstrated in a number of studies comparing the SCORS to validated research instruments such as the Rorschach Inkblot Method (Ackerman, Hilsenroth, Clemence, Weatherill, & Fowler, 2001; Hibbard, Hilsenroth, Hibbard, & Nash, 1995) and the *DSM-IV* Axis V scales (Peters et al., 2006). Previous studies have found the SCORS useful for understanding affective aspects of psychopathology (Hibbard et al., 1995), patterns of psychotherapy continuation (Ackerman et al., 2000), personality change in treatment-refractory inpatients (Fowler et al., 2004); interpersonal components of victims of childhood sexual abuse (Callahan, Price, & Hilsenroth, 2003; Slavin et al., in press), and differentiating borderline

personality disorder outpatients into subgroups (Tramantano, Javier, & Colon, 2003). The SCORS has also shown the capacity to discriminate between borderlines, major depressives, and normals (Westen, Lohr, Silk, Gold, & Kreber, 1990), MMPI diagnosed psychotics, sociopaths, and normals (Porcerelli et al., 1995), inpatient suicide attempters and non-attempters (Kaslow et al., 1997), as well as cluster B and cluster C personality disorders (Ackerman et al., 1999). The SCORS has also predicted treatment response for PTSD diagnosed inpatients (Ford, Fisher, & Larson, 1997) and psychosocial adjustment approximately two years after the death of a spouse (Field, Sturgeon, Puryear, Hibbard, & Horowitz, 2001).

Interrater reliability of each of the SCORS subscales was evaluated utilizing a one-way random effects model intraclass correlation coefficients (ICC [*1*,*1*]; Shrout & Fleiss, 1979) from 20 randomly selected pre- or post-treatment protocols across all three groups. Raters were two advanced doctoral students enrolled in an American Psychological Association approved clinical Ph.D program. Prior to rating the SCORS both raters participated in group training in which scoring guidelines were reviewed and supervised by a licensed Ph.D Clinical Psychologist. Upon completion of the reliability scoring, raters met to discuss any ratings that were separated by two or more points to attempt to bring the scores to at least within one point of each other. This additional step was necessary due to the degree of cognitive distortion and psychotic psychopathology inherent in this schizophrenic sample. All protocols were coded to mask group affiliation, name, gender, and time to ensure that ratings were blind throughout the entirety of scoring for both raters. Table 3 reports interrater reliability for each of the SCORS' eight subscales. ICCs are considered to be excellent if greater than .74, good from .60 to .74,

fair from .40 to .59, and poor if under .40 (Shrout & Fleiss, 1979; Fleiss, 1981). All SCORS subscales were in the good to excellent range.

Explanation of Analyses

SCORS variables with appropriate unrestricted variance will be utilized in all subsequent analyses. First, to assess the convergent and divergent validity of the SCORS for use with a schizophrenic sample, Pearson *r*s were computed between each of the pre-treatment SCORS variables and five criterion variables: 1) pre-treatment Clinical Status Interviews (CSI); 2) verbal IQ as measured by the WAIS (Weschler, 1955); 3) total days in hospital (controlling for treatment change) over the course of the 20 month investigation; 4) age; and 5) years of education.

CSIs were conducted with each patient at pre-treatment by Karon and Vandenbos (1981). The CSI is a clinician-rated indicator of overall health-sickness comparable to the Global Assessment of Functioning scale found in the *DSM-IV* (APA, 1994). Similar to the SCORS, higher CSI scores indicate healthier levels of functioning. Thus, it was hypothesized that the CSI and all SCORS variables would exhibit a positive and significant relationship.

Past research has found that variables COM and USC are the cognitive variables of the SCORS measure, as opposed to the other SCORS variables that are deemed the affective variables (Hibbard et al., 1995; Westen, 1995). Therefore, in regard to verbal IQ, it was predicted that the SCORS' cognitive variables COM and USC would be positively and significantly related to verbal acuity as measured by the WAIS, thereby indicating convergent validity. In contrast, in the service of assessing divergent validity,

it was hypothesized that AFF, EIR, SE, and ICS would not exhibit a positive and significant relationship, since these variables are designed to assess the affective components of object relations and are theoretically distinct from IQ. Exhibiting divergent validity of a narrative-based measure with verbal acuity is important to ensure that the quality of narratives is more related to the construct under investigation (i.e., object relations) rather than to verbal skills.

In regard to the SCORS and total days in the hospital, it was hypothesized that the relationship would be significant and in the *negative* direction. That is, the more days spent in the hospital due to poor psychiatric functioning, the lower the pre-treatment SCORS ratings. Lastly, in the service of divergent validity, it was hypothesized that age and years of education would not be related to object relations. Pearson *r* correlational coefficients are considered to represent a small effect from .1 to .3, a medium effect from .3 to .5, and a large effect if greater than .5 (Cohen, 1988).

Utilizing paired-samples *t* tests, pre-treatment SCORS variables with appropriate unrestricted variance were examined to determine within-group change. This analysis assessed whether or not Psychotherapy or Medication treatment led to significant change across each SCORS variable. *T* scores were converted to Cohen's *d* effect size to facilitate comparison between variables and groups and provide a commonly used magnitude of effect coefficient. Cohen's *d* values .2, .5, and .8 indicated small, medium, and large effects, respectively.

Following the paired-samples *t* tests, 'difference scores' for each SCORS variable were calculated for each SCORS variable at the group level (i.e., post-treatment minus pre-treatment). Independent-samples *t* tests will be conducted to assess between-group

differences for each SCORS variable. While these traditional outcome analyses allow for determination of gross differences between treatment groups, they are not capable of isolating individual patient change. To examine change at the *individual patient level* pre-treatment SCORS variables with appropriate unrestricted variance were subsequently examined using Clinical Significance (CS) methodology (Jacobson & Truax, 1991; Jacobson, Roberts, Berns, & McGlinchley, 1999). Utilization of CS methodology provides "valuable information on variability of outcome within each treatment condition and a way of determining the practical importance of statistically significant differences between groups" and will be used to "descriptively augment" group change examined with the independent-samples *t* tests of 'difference scores' (Jacobson et al., p. 301). Jacobson and colleagues (1999) write,

> Group means…do not in and of themselves indicate the proportion of participants who have improved or recovered as a result of treatment… Standard statistical comparisons between groups seldom determine the practical importance of the treatment effects… even effect sizes do not directly speak to clinical significance" (p. 300).

In the CS methodology an individual patient is considered 'Recovered' if he or she meets two criteria: 1) a Reliable Change Index (RCI) score that exceeds 1.96; and 2) achieves an outcome score (on the SCORS subscale) that is 2 *SD*s greater than the accepted normative data of the scale in question. Meeting only one or the other criterion precludes a researcher from labeling the individual 'Recovered'. Considering the acutely severe presentation of the current patient sample and the increased time it took for these patients to seek hospital admission, there are obvious concerns regarding regression to

the mean (Fowler et al., 2004). As a result, the Edwards-Nunnally formula (Speer, 1992), which was created to control for regression to the mean during the computation of RCI scores was applied to adjust all pre-treatment mean scores.

Criterion 1, calculation of the RCI, is computed in a fashion that controls for measurement error, therefore suggesting that the RCI score is both psychometrically reliable and reflective of real change, rather than standard error of measurement. For our study using the SCORS, a modification in the RCI formula was necessary because test-retest reliability, which is unavailable for the SCORS, is most commonly used to determine the standard error of difference (*Sdiff*) found in the denomination of the RCI formula (Wise, 2004). Jacobson et al. (1999) propose utilization of alternative measures of reliability for the calculation of *Sdiff*. Hilsenroth, Ackerman, Blagys, Baity, & Mooney, (2003) and Fowler et al. (2004) have made use of this suggestion in two recently published outcome studies by using ICCs as an alternative reliability coefficient in calculating *Sdiff*. For the purpose of this dissertation project ICCs will be used in a like manner.

In regard to criterion 2, just as there are no normative test-retest data for the SCORS, there are also no available normative means for its subscales. Fortunately, other authors have proposed (Bauer, Lambert, and Nielsen, 2004) and utilized (Fowler et al., 2004; Hilsenroth et al., 2003) an alternative method for computing a post-treatment outcome score cutoff point. This cutoff point is determined by taking the pooled pre-treatment sample mean of each target variable (i.e., the SCORS subscale) and adding two *SD*s. For example, if the pooled mean of the Complexity of Representations variable was

2.01 with a SD of .67, the outcome score of this variable for an individual patient must exceed 3.35 (i.e., 2.01 + [.67 * 2]) to meet the condition needed to satisfy criterion 2.

One last modification is necessary due to the severity of our sample. Wise (2004) observed that it is unrealistic to expect a sample of severely disturbed patients to achieve the clinically significant benchmarks of RCI > 1.96 and an outcome cutoff point two *SD*s greater than the pre-treatment mean. As such, Wise (2004) recommended additional criteria for both RCI scores and standard deviation cutoff points. Wise's (2004) alternative is termed 'Positive Response' and requires that a patient achieve an RCI > 1.28 (i.e., the 90% confidence interval for RCI) and an outcome cutoff point one *SD* greater than the pre-treatment mean. In their study of treatment-refractory inpatients, Fowler et al. (2004) utilized Wise's (2004) alternative criteria in addition to Jacobson and Truax' (1991) original classification (RCI > 1.96 and *SD* > 2.0). Based on our severely disturbed schizophrenic sample, the current evaluation has also focused on both change estimates classified as 'Recovered' and 'Positive Response'. Percentages of patients in each group meeting these two criteria were computed for all six SCORS variables. Rates of clinical regression follow the same criteria but in the opposite direction and they too are reported. Examining regression (if any) is particularly significant due to the widely held belief that psychodynamic psychotherapy of schizophrenia is dangerous and results in clinical regression (e.g., Lehman & Steinwachs, 1998).

An additional manner in which to understand how the patients of the current sample presented clinically at post-treatment can be accomplished by comparing post-treatment SCORS scores to alternative SCORS data derived from another study that investigated a less disturbed sample. For such a purpose, Table 4 reports pre-treatment

SCORS subscale data from a study investigating the object relations of a patient sample seeking psychotherapy from a university-based mental health center (Peters et al., 2006). Peters and colleagues reported that the "level of psychological/emotional distress of the patients in this treatment program was primarily in the mild to moderate range of impairment, as evidenced by the *DSM-IV* (American Psychiatric Association, 1994) diagnostic categories, clinician rating scales, and self-report measures" (p. 618). Thus, if the outcome SCORS ratings of any schizophrenic patients equal or exceed the SCORS ratings of this markedly healthier sample, it can be cautiously suggested that, at post-treatment, the object relations of these patients are comparable to patients entering psychotherapy with mild to moderate levels of distress. Providing this auxiliary information is another manner by which to show whether or not schizophrenics receiving medication–only or psychotherapy-only are capable of improving in a manner contrary to the expectations set forth by the biopsychiatric model.

While comparing percentages of those patients meeting 'Recovered' and 'Positive Response' criteria provide important clinical information, application of chi square analyses would provide a measure of statistical significance for group differences. However, to perform the necessary 12 chi square analyses for all six subscales testing for group differences using 'Recovered' and 'Positive Response' criteria would greatly increase the risk of making a Type I error. Ford and colleagues (1997) and Peters et al. (2006) have previously used a composite object relations score as a variable. This procedure was adopted for the present study to limit the number of performed analyses in an attempt to minimize the possibility of a Type I error. SCORS-Composite (SCORS-C) was calculated by simply summing each of the SCORS variables and dividing by the

number of variables summed to produce a single 7-point object relations total score. RCI scores and improvement status ('Recovered' and/or 'Positive Response') were calculated using the same procedures described above for the individual SCORS subscales. Chi square scores were converted to Cohen's *d* effect size to facilitate comparison between variables and provide a commonly used magnitude of effect coefficient.

Lastly, to statistically test for group differences in clinical change *after controlling for error measurement and regression to the mean*, independent-samples *t* tests were computed using RCI scores as the dependent variable and group assignment as the grouping variable. Again, because the RCI score is both psychometrically reliable and reflective of real change, rather than standard error of measurement or regression to the mean, it is a particularly useful indicator of clinical change and a statistical improvement over past outcome research that historically has not controlled for such artifacts. In this way, independent-samples *t* tests using RCI as the dependent variable might be a more reliable and accurate indicator of group change compared to the traditional 'difference score' analyses also conducted in this study. *T*-scores were converted to Cohen's *d* effect size to facilitate comparison between variables and provide a commonly used magnitude of effect coefficient.

III. RESULTS

The EIM (*SD* = .22) and AGG (*SD* = .24) variables (combined groups) did not exhibit suitable unrestricted variance at pre-treatment and were thus removed from subsequent analyses. The *SD*s of the other six SCORS variables ranged from .49 - .81, thus the *SD*s for EIM and AGG were observably restricted. As a result, only six of the eight SCORS subscales will be utilized in all subsequent analyses (COM, AFF, EIR, USC, SE, and ICS). There were no significant between-group differences for any of the six pre-treatment SCORS variables or demographic variables (education, age, gender, race) confirming the randomness of group assignments.

Convergent and Divergent Validity

Table 5 reports all convergent and divergent validity analyses utilizing the pre-treatment SCORS ratings. As expected, the WAIS Verbal Scale IQ ($n = 17$) and the SCORS' COM ($r = .52$, $p = .03$) and USC ($r = .45$, $p = .01$) exhibited significant and moderate to large effects, indicating that the SCORS' cognitive variables were appropriately related to verbal intelligence. In regard to divergent validity, as expected, the SCORS' affective variables were not related to verbal IQ, indicating that the SCORS affective variables measure something distinct from verbal functioning. In regard to convergent validity, the CSI ($n = 21$) and the pre-treatment SCORS variables COM ($r = .79$, $p < .0001$), USC ($r = .71$, $p < .0001$), and ICS ($r = .59$, $p = .005$) all exhibited significant and large effects. Pearson *r*s of the CSI and the pre-treatment SCORS variables AFF ($r = .50$, $p = .02$) and SE ($r = .48$, $p = .03$) exhibited significant and moderate effects. SCORS variable EIR ($r = .42$, $p = .06$) approached significance with a

moderate effect. Associations between the pre-treatment SCORS subscales and total days in the hospital (n = 21) further substantiate the convergent validity of the SCORS for use with this uniquely disturbed cohort. Pearson r partial correlations for total days in hospital and the pre-treatment SCORS variables COM (r = -.57, p = .009), USC (r = -.66, p = .002), and ICS (r = -.68, p = .001) all exhibited significant and large effects in the appropriate negative direction. Pearson r partial correlations for total days in hospital and the pre-treatment SCORS variables AFF (r = -.38, p = .10) and EIR (r = -.39, p = .09) approached significance with moderate effects in the appropriate negative direction.

Within-Group Analyses of Change

Paired-samples t tests illustrate pre- to post-treatment within-group change across all six SCORS variables for both the Psychotherapy (Table 6) and Medication (Table 7) groups. Patients in the Psychotherapy group exhibited significant change with robust effects across all six object relations constructs (COM t[8] = 4.46, p = .002, d = 3.15; AFF t[8] = 3.89, p = .005, d = 2.75; EIR t[8] = 2.39, p = .04, d = 2.39; USC t[8] = 6.53, p = .0001, d = 4.62; SE t[8] = 4.53, p = .002, d = 3.20; and ICS t[8] = 5.01, p = .001, d = 3.54). Overall mean Cohen's d effect size for the Psychotherapy group was large at 3.28. Comparatively, patients of the Medication group did not fare as well relative to patients receiving psychodynamic psychotherapy. Only two of six variables in the Medication group reached statistical significance for pre- to post-treatment change (COM t[11] = 2.67, p = .02, d = 1.61 and EIR t[11] = 3.29, p = .007, d = 1.98). SCORS variables USC t[11] = 2.04, p = .07, d = 1.23 and ICS t[11] = 1.74, p = .11, d = 1.05 approached significance. Considering the small group size (and thus limited power), the results for

the latter two variables may be indicative of a significant change. Liberally, these effect sizes will be included in the Medication group's mean effect size. The effect sizes of the non-significant variables will be set to zero for this calculation. As such, overall mean Cohen's *d* effect size for the Medication group was .98.

Traditional Analyses of Between-Groups Change Using 'Difference Scores'

Traditional analyses of between-groups change found that psychodynamic psychotherapy for schizophrenia is superior to medication-only for treating schizophrenia across multiple object relations domains (Table 8). Differences between treatments were significant for three of six SCORS variables with large effects (COM $t[19] = 2.27$, $p = .04$, $d = 1.05$; USC $t[19] = 2.61$, $p = .02$, $d = 1.21$; and SE $t[19] = 2.40$, $p = .03$, $d = 1.13$). SCORS variables AFF $t[19] = 1.94$, $p = .07$, $d = .90$ and ICS $t[19] = 1.65$, $p = .12$, $d = .77$ both approached significance with a large and medium effect, respectively. Considering the small sample size (and thus limited power) used in these analyses, the results for the latter two variables may be indicative of significant between-groups treatment effects. The SCORS variable EIR did not exhibit a statistically significant between-groups effect ($p = .86$).

'Recovered' and/or 'Positive Response' to Treatment

Table 9 reports improvement and deterioration rates of the Psychotherapy and Medication patients for the SCORS subscale Complexity of Representation (COM). Utilizing Jacobson and Truax' (1991) conservative estimates of recovery (i.e., RCI > 1.96 and *SD* > 2.0), 56% (5/9) of Psychotherapy patients met criteria for recovery in regard to a multi-dispositional and differentiated view of self and others. An even more

conservative estimate of recovery would be the 22% (2/9) of patients who achieved an RCI > 1.96 and reached the pre-treatment mean of the patients represented in Peters et al. (2006). In comparison, 42% (5/12) of the Medication patients met the conservative estimate of recovery for COM. However, unlike the Psychotherapy group, no medicated patients achieved the RCI > 1.96 score and pre-treatment mean of the patients represented in Peters et al. (2006).

Table 10 reports improvement and deterioration rates of the Psychotherapy and Medication patients for the SCORS subscale Affect Tone of Representations (AFF). Conservative estimates of recovery found that 11% (1/9) of Psychotherapy patients met criteria for improvement in their expectations of other as benevolent and helpful. Interestingly, 33% (3/9) of the Psychotherapy patients achieved an RCI > 1.96 and reached the pre-treatment mean of the patients in Peters et al. (2006), indicating that a third of the Psychotherapy group ended treatment comparable to an average outpatient in regard to affective expectations of others. No Medication patients met the Jacobson and Truax' (1991) conservative estimates of recovery or the RCI > 1.96 in combination with the pre-treatment mean of the patients represented in Peters et al. (2006).

Table 11 reports improvement and deterioration rates of the Psychotherapy and Medication patients for the SCORS subscale Emotional Investment in Relationships (EIR). Conservative estimates of recovery found that 33% (3/9) of Psychotherapy patients met criteria for improvement in regard to their desire to invest themselves emotionally in interpersonal relationships. More conservatively, only 11% (1/9) of psychotherapy patients achieved an RCI > 1.96 and reached the pre-treatment mean of the patients in Peters et al. (2006). In comparison, 17% (2/12) of the Medication patients

met the conservative estimate of recovery, and only 8% (1/12) of the medicated patients achieved an RCI > 1.96 and reached the pre-treatment mean of the patients in Peters et al. (2006).

Table 12 reports improvement and deterioration rates of the Psychotherapy and Medication patients for the SCORS subscale Understanding of Social Causality (USC). Conservative estimates of recovery found that 44% (4/9) of Psychotherapy patients met the criteria for improvement in regard to their capacity to determine why people behave the way they do in the interpersonal field. That is, nearly half of the Psychotherapy patients by treatment's end were significantly better equipped to read the motivation and behavior of others, largely ameliorating one of the defining features of schizophrenic suffering: the inability to realistically assess one's social world. An even more conservative estimate of recovery would be the 22% (2/9) of patients who achieved an RCI > 1.96 and reached pre-treatment mean of the patients in Peters et al. (2006). In comparison, 25% (3/12) of Medication patients met the conservative estimate of recovery. Unlike the Psychotherapy group, no Medication patients achieved an RCI > 1.96 and reached pre-treatment mean of the patients in Peters et al. (2006).

Table 13 reports improvement and deterioration rates of the Psychotherapy and Medication patients for the SCORS subscale Self-Esteem. Conservative estimates of recovery found that 22% (2/9) of Psychotherapy patients met criteria for improvement in their experience of self-esteem. Of note, 56% (5/9) of Psychotherapy patients achieved an RCI > 1.96 and reached the pre-treatment mean of the patients in Peters et al. (2006). That is, by treatment's end, over half of the Psychotherapy patients had as much self-esteem as a mild to moderately severe clinical sample typical of an outpatient, university-

based psychotherapy clinic. In comparison, no medication patients met Jacobson and Truax' (1991) conservative estimates of recovery for Self-Esteem nor did any medicated patients achieve an RCI > 1.96 and reach the pre-treatment mean of the patients in Peters et al. (2006).

Table 14 reports improvement and deterioration rates of the Psychotherapy and Medication patients for the SCORS subscale Identity and Coherence of the Self (ICS) Conservative estimates of recovery found that 11% (1/9) of Psychotherapy patients met criteria for improvement in their experience of themselves as moving forward in the world with goals and a coherent, temporally stable sense of self. Of note, 67% (6/9) of Psychotherapy patients achieved an RCI > 1.96 and reached the pre-treatment mean of the patients in Peters et al. (2006). That is, by treatment's end, two-thirds of the Psychotherapy patients subjectively experienced themselves as an individual with a personality as well integrated and as non-psychotic as patients seeking outpatient psychotherapy. In comparison, no medication patients met Jacobson & Truax' (1991) conservative estimates of recovery while 25% (3/12) achieved an RCI > 1.96 and reached the pre-treatment mean of the patients in Peters et al. (2006).

Table 15 reports improvement and deterioration rates of the Psychotherapy and Medication patients for the SCORS composite variable (SCORS-C). Conservative estimates of recovery found that 44% (4/9) of Psychotherapy patients met criteria for improvement in their overall object relations. In comparison, only 8% (1/12) of medication patients met Jacobson & Truax' (1991) conservative estimates of recovery in regard to overall object relations change.

As can be viewed in Tables 9-15, for all six SCORS variables and SCORS-C, rates of improvement rose for the Psychotherapy patients when the criteria were relaxed according to Wise's (2004) alternative 'Positive Response' criteria (RCI > 1.28 and *SD* > 1.0). Rates of 'Positive Response' to psychodynamic psychotherapy were as follows: Complexity of Representations (7/9, 78%); Affect Tone of Representations (3/9, 33%); Emotional Investment in Relationships (5/9, 56%); Understanding of Social Causality (7/9, 78%); Self-Esteem (4/9, 44%); Identity and Coherence of the Self (6/9, 67%); and SCORS-C (7/9, 78%).

In regard to the Medication group, as can be viewed in Tables 9-15, rates of improvement *generally* - though not across the board - rose when the criteria were relaxed according to Wise's (2004) alternative 'Positive Response' criteria (RCI > 1.28 and *SD* > 1.0). Rates of 'Positive Response' to medication were as follows: Complexity of Representations (5/12, 42%); Affect Tone of Representations (1/12, 8%); Emotional Investment in Relationships (5/12, 42%); Understanding of Social Causality (6/12, 50%); Self-Esteem (3/12, 25%); Identity and Coherence of the Self (4/12, 33%); and SCORS-C (4/12, 33%).

Contrary to the PORTs negative expectations, it is important to note that whereas *no Psychotherapy patients exhibited clinically significant regression* either at the conservative or more relaxed criteria, there were in fact Medication patients who exhibited clinically significant regression when inverting Wise's (2004) 'Positive Response' criteria (i.e., 'Negative Response'). For Medication patients, 8% (1/12) exhibited a 'Negative Response' for COM, AFF, EIR, ICS, SE, and SCORS-C. Similar to conservative rates of improvement using Jacobson and Truax' (1991) 'Recovered'

criteria, the Psychotherapy patients also fared better (percentage wise) on *all* variables when using Wise's (2004) 'Positive Response' criteria.

In regard to significance tests for SCORS-C, chi square analyses examining group differences indicated results approaching significance with a large effect for number of 'Recovered' patients ($\chi^2 = 3.70$, $p = .12$, $d = .93$) as well as 'Positive Response' patients ($\chi^2 = 4.07$, $p = .08$, $d = .98$). Considering the small sample size (and thus limited power) of the present study, these results may be indicative of a significant and clinically relevant difference between groups.

As mentioned, no patients receiving psychodynamic psychotherapy exhibited clinically significant regression. In fact, any sign of regression indicated by a negative RCI score was relatively rare for patients receiving psychodynamic psychotherapy. Out of 63 computed RCI scores (i.e., nine patients multiplied by six SCORS variables and SCORS-C) only 4 had a negative value (6%). In fact, for SCORS-C, not only were there no psychotherapy patients with a negative RCI score, only one patient had a SCORS-C RCI score below 1.00. This degree of consistent positive performance was not true of the Medication group for which 25 of 84 RCI scores (30%) exhibited clinical regression. Furthermore, 25% (4/12) of Medication patients had a negative RCI score for SCORS-C, and, as mentioned, one Medication patient exhibited *clinically significant regression* for the inversion of Wise's 'Positive Response'. A *post hoc* chi square analysis found that the between-group difference in regard to presence of a *negative treatment response* as indicated by a negative SCORS-C RCI was approaching significance with a large effect ($\chi^2 = 3.71$, $p = .10$, $d = .93$). Considering the small sample size (and thus limited power)

of the present study, these results may be indicative of a significant and clinically relevant difference between groups.

Group Differences for RCI Scores

To maintain the optimal clinical utility of the present study, it was important to also test for group differences on each of the six SCORS variables while controlling for error measurement and regression to the mean. Providing data on these individual variables offers practicing clinicians tangible, real-world information applicable to clinical practice and therefore maintains the generalizability of the study. That is, it is clinically useful to know if one treatment or the other specifically impacts a particular dimension of object relations after controlling for artifacts. Table 16 reports the results of independent-samples *t* tests conducted for all six SCORS variables to determine if the Psychotherapy and Medication groups differed in regard to degree of change as measured by the RCI. Again, because the RCI is reflective of real change and not the product of measurement error it is a particularly useful indicator of clinical progress. Three of six SCORS RCI values exhibited statistically significant and large differences between the Psychotherapy and Medication groups. As can be seen by the RCI means in Table 16, all results favored the Psychotherapy patients (COM, $t[19] = 2.08$, $p = .05$, $d = .92$; USC $t[19] = 2.44$, $p = .03$, $d = 1.08$; and SE $t[19] = 2.45$, $p = .02$, $d = 1.08$). Two of six SCORS RCI values approached statistical significance with large effects between the Psychotherapy and Medication groups (AFF, $t[19] = 2.01$, $p = .06$, $d = .89$; ICS $t[19] = 1.81$, $p = .09$, $d = .80$). Again, considering the small sample size (and thus limited power) of the present study, the results for the latter two variables may be indicative of a

significant and clinically relevant difference between groups. The SCORS variable EIR did not exhibit a statistically significant between-groups effect ($p = .71$). These results are consistent with the traditional 'difference score' analyses presented in Table 8, though after controlling for error measurement and regression to the mean, as can be seen by comparison of effect sizes, results favoring the Psychotherapy group were even more robust.

IV. CONCLUSION

For far too long the empirical evidence supporting the positive effects of psychodynamic psychotherapy for the treatment of schizophrenia has been neglected due to a few core assumptions. As a result, there has been little space for its regular inclusion in research; furthermore, its clinical application in standard treatment has been diminishing for years.

First, the degenerative brain disease assumption has inculcated the idea in the mental health profession that schizophrenia can only be managed rather than cured. As extensively presented above, substantial evidence contradicts this notion on the grounds that 1) the 19th century derived schizophrenia concept is grounded in often unacknowledged non-epistemic ideas such as degeneration theory and philosophical realism that serve to distort current conceptualization and treatment approaches; 2) research investigating the effects of the duration of untreated schizophrenia has *not* reliably shown a psychotoxic effect on the brain, nor has it been related to clinical regression; and 3) ample longitudinal, cross-cultural data exist that document significant improvement and recovery for people suffering with schizophrenia.

The second assumption is the supposed superiority and safety of medication treatment for schizophrenia. Despite limited empirical backing, this assumption has been reproduced continuously since the 1950s. In contrast, substantial evidence presented earlier suggests that medications are of limited effectiveness, undeniably dangerous in regard to an array of side-effects and may even preclude clinical progress/recovery.

The third assumption, that is, the alleged ineffectiveness of psychodynamic psychotherapy, has been directly stated in the schizophrenia Patient Outcome Research Team study (PORT; Lehman & Steinwachs, 1998), a recent schizophrenia treatment guideline published by biologically-based clinicians and researchers. PORTs Recommendation 22 reads,

> Individual and group psychotherapies adhering to a psychodynamic model (defined as therapies that use interpretation of unconscious material and focus on transference and regression) should not be used in the treatment of persons with schizophrenia… there is a consensus that psychotherapy that promotes regression and psychotic transference can be *harmful* to persons with schizophrenia (pp. 7-8; my emphasis).

Since the PORT project was convened to review the clinical outcome literature to create treatment guidelines for practicing psychiatrists and psychologists, this misperception is extremely significant. Ver Eecke (2003), Gottdiener and Haslam (2003), and Silver (2003) provide a clear, empirically- and theoretically-based critique of the mistaken conclusions of the PORT. The results of the current investigation add weight to their important criticism and directly challenge this flawed and widespread assumption.

Speaking directly to the empirical results of this dissertation project, there are four major findings. First, the SCORS exhibits satisfactory inter-rater reliability for use with a schizophrenic sample. Second, the SCORS, in a preliminary fashion, exhibits adequate convergent and divergent validity for use with a schizophrenic sample. Third, contrary to the PORTs admonishments that psychodynamic psychotherapy for the treatment of schizophrenia is “harmful”, the results of this study indicate that psychodynamic

psychotherapy is capable of providing reliable clinical change in the object relations of schizophrenic patients. Fourth, in comparison to medication-only treatment, psychodynamic psychotherapy results in significantly greater improvement in a variety of object relations domains for patients suffering with schizophrenia.

Reliability estimates for the current investigation are consistent with past research regarding the interrater reliability of the SCORS variables (e.g., Fowler et al., 2004). Importantly, although the SCORS has been used to rate a diverse array of clinical and non-clinical samples, as far as can be determined this is the first study to apply the SCORS to a strictly schizophrenic sample. Thus, this study extends previous research since the current findings indicate the SCORS can reliably measure TAT narratives collected from severely psychotic individuals.

Correlational analyses helped illuminate preliminary divergent and convergent validity of the SCORS for use with a psychotic sample. As expected, the affective SCORS variables were unrelated to verbal functioning. This finding is important because it is imperative that a narrative-based measure maintains its independence from verbal acuity so that an individual's capacity to verbalize him- or herself does not disproportionately obscure accurate assessment of other constructs under investigation. Further, support for the SCORS' independence from verbal IQ is important due to the fact that Karon and Vandenbos (1981) have already reported group differences for IQ in their original study. Thus, the divergent validity observed in the present investigation allows for analysis and reporting of non-redundant clinical outcome data. Divergent validity was further supported since there were no significant correlations between the

SCORS and age or education, as hypothesized. In sum, results provide preliminary divergent validity evidence for use of the SCORS with a strictly psychotic sample.

As hypothesized, the SCORS' cognitive variables (COM and USC) were related to a commonly used measure of verbal IQ, suggesting that the SCORS can provide valid measurement of cognitive processes such as verbal production. Also consistent with the theoretical nature of the SCORS, correlational results with clinical status interviews (CSI) and days spent in the hospital throughout the duration of the 20-month study exhibited significant and moderate to large effect sizes. Determining SCORS object relations variables capable of predicting inpatient stays may aid treatment planning by highlighting particularly important areas of functioning worth focusing on to avoid hospitalizations. The results of this study suggest that clinical attention to patients' accurate appraisal of social interactions (USC), degree of complex understanding of self and others (COM), goal-directed behavior across time (ICS), affective expectations of others (AFF), emotional investment in interpersonal relationships (EIR), as well as issues of self-esteem are all potentially important areas of treatment focus that might prevent or decrease the need for hospitalization.

Previous studies have found the SCORS to be a useful indicator of process variables such as treatment continuation (Ackerman et al., 2000), suicidality (Kaslow et al., 1997), childhood sexual abuse (Callahan, Price, & Hilsenroth, 2003; Slavin, Stein, Pinsker, & Hilsenroth, 2007), social, vocational, and symptomatic functioning (Peters et al., 2006), and mourning and loss (Field, Sturgeon, Puryear, Hibbard, & Horowitz, 2001), all of which are highly relevant to the treatment of schizophrenia. Thus, the reliability and validity results of this investigation represent a promising beginning for use of the

SCORS in future process and outcome schizophrenia research. Inclusion of this widely validated instrument capable of measuring personality processes beyond overt symptomatic distress would also be a significant improvement over the majority of schizophrenia outcome studies that overwhelmingly rely on self-report measures of positive and negative symptoms, or, simply, medication compliance.

In regard to outcome, the current investigation replicates past results documenting the effectiveness of psychodynamic psychotherapy for the treatment of schizophrenia (see for a review Gottdiener & Haslam, 2002). However, this study is fairly unique in contemporary research in that data for a *completely non-medicated group* is provided for comparative purposes with a medication-only group. As a result, this study has been able to test and, in the end, *contest* two important assumptions of the recent PORT study, namely: 1) that psychodynamic psychotherapy of schizophrenia is "harmful"; and 2) that medication treatments are preferable.

First, in regard to the psychotherapy of schizophrenia being "harmful", the present findings clearly indicate that psychotherapy was *not* harmful, but, to the contrary, resulted in clinically reliable and valid change in both individual and group analyses across all of the object relations constructs measured including a patients' understanding of the internal motivations, thoughts, and feelings of self and others (COM), emotional investment in relationships (EIR), accurate appraisal of social interactions (USC), more benevolent affective expectations of others (AFF), greater levels of self-esteem (SE), more coherent sense of identity across time (ICS), and healthier global object relations (SCORS-C). Far from being harmful, CS methodology assessing individual patients' progress revealed that an impressive percentage of psychodynamic psychotherapy

patients across all variables met 'Positive Response' (33-78%) and/or 'Recovered' (11-56%) criteria. These results extend Karon & Vandenbos' (1981) original findings that found psychodynamic psychotherapy to be effective for treating the thought disorder component of schizophrenia into the broader realm of interpersonal and self functioning as measured by the SCORS. *These within-group positive outcome results stand with or without comparison to a medication-only group.* This is a highly significant point because a likely critique of the current design was the use of the older, typical medications as opposed to the newer atypicals. However, as discussed above, such a critique is empirically untenable due to the majority of clinical research that has found no particular clinical advantages of the newer neuroleptic medications (e.g., the CATIE study).

An additional empirical test of the PORT's claim that psychodynamic psychotherapy is harmful was carried out via a *post hoc* analysis of group differences testing an indicator of clinical regression (i.e., negative RCI scores). In direct contrast to the PORT's claim, this analysis found that the number of Medication patients exhibiting a negative RCI score on a measure of global object relations (SCORS-C) was significantly greater than Psychotherapy patients. That is, regarding object relations change the medication-only approach and *not* psychodynamic psychotherapy appeared to place patients at greater risk for clinical regression and of course physiological side-effects for which there are none for psychotherapy.

Unlike the psychotherapy patients, results for medication patients were inconsistent as indicated by within-group and individual analyses. When assessed as a group (Table 7), Medication patients failed to exhibit group change in regard to Affect

Tone of Representations and Self-Esteem, two areas that were significantly and positively impacted by psychodynamics psychotherapy. In regard to individual analyses using CS methodology, while nearly every patient in the Psychotherapy group showed some degree of positive improvement across *all* SCORS variables (i.e., as indicated by a positive RCI score), this cannot be said of the Medication patients. This is to say, some Medication patients did well on a few variables but not all, and, more problematically, some Medication patients simply did poorly across the board. Empirically, this helps explain statistically significant group differences for both 'difference scores' (Table 8) and RCI scores (Table 16) favoring the psychodynamic psychotherapy patients for SCORS variables COM, USC, SE, AFF, and ICS, in addition to the overall number of patients exhibiting 'Recovered' and 'Positive Response' criteria for SCORS-C.

The less consistent outcome of the Medication patients suggests that while medication treatment can be helpful for some patients, its generalized, non-specific qualities might suggest a certain 'hit or miss' phenomenon. This seems to be the case in this sample despite psychiatrists who made numerous medication adjustments based on their expert, biological-based understanding of schizophrenia. In fact, because medication treatments are of course not capable of handling the idiosyncratic thematic issues troubling a patient, it would be unreasonable to expect medication to provide an ameliorative effect for a patient's interpersonal, self-esteem, and identity concerns among a multitude of other complex issues gravely impacting a person suffering with schizophrenia. However, in stark contrast and as many have theoretically suggested (e.g., Fromm-Reichmann, 1950), the capacity for a therapist to engage a patient as a complex

person with unique difficulties and a life-story to share may be the driving curative factor in psychotherapeutic work.

The erratic outcome and more significant regression observed for the Medication patients are not particularly surprising. Writing in the *New England Journal of Medicine* Goldstein (2003 notes, "One of the most striking features of modern medicines is how often they fail to work. Even when they do work, they are often associated with serious adverse reactions" (p. 553). The relatively high regression of the Medication patients found in the present study is also consistent with the extraordinarily high early discontinuation rates found in contemporary medication research (e.g., 74%) which stands as another indicator of medication intolerability and ineffectiveness (Lieberman et al., 2005). Of course, the empirically invalid assumption that safe and effective alternatives such as psychodynamic psychotherapy do not exist limits treatment choices and fosters the pessimistic submission that medication and their associated drawbacks are, so to speak, 'all that's out there'. The results of this study, as well as other empirical investigations of the effectiveness of psychodynamic psychotherapy strongly suggest otherwise.

Investigating the results more closely, a few more important issues surface which are highly relative to the comparative effectiveness of the interventions in question. To a degree, and usually on a temporary basis, medication treatments have been shown to minimize the positive symptoms of schizophrenia such as disorganized and delusional thinking as well as hallucinations. Though, as presented above, such treatment successes are often overestimated due to uncontrolled-for statistical confounds such as abrupt drug withdrawal, clinically impractical exclusion criteria, multiple prescribing practices,

excessive early discontinuation, and psychological and physiological impact of side-effects (Karon, 1989; Jackson, 2005; Thornley & Adams, 1998). Nonetheless, despite such confounds, decrease of positive symptoms via medication seems to be the one fairly consistent and positive result of medication research and treatment. Indeed, in the present study, most medication patients fared acceptably according to the SCORS cognitive variables COM and USC. Nonetheless, the psychotherapy patients fared significantly better for these variables. That is, psychotherapy outperformed medications in the very area medications are expected to function most effectively. This is a key finding considering that there is also considerable evidence that long- and short-term neuroleptic use may actually harm cognitive functioning in a variety of domains (Kasper & Resinger, 2003; Minzenburg et al., 2004; Gilbertson & van Kammen, 1997; Mehta, Manes, Magnolfi, Sahakian, & Robbins, 2004; Papageorgiou et al., 2003; Sweeney, Keilp, Haas, Hill, & Weiden, 1991; Vitiello et al., 1997; Cassens, Inglis, Appelbaum, & Gutheil, 1990; King, 1994; Ridout & Hindmarch, 2003; Brebion, Amador, Smith, & Gorman, 1998; Cleghorn, Kaplan, Szechtman, Szechtman, & Brown, 1990; Rammsayer, 1997; McCartan et al., 2001; Scherer et al., 2004), thus making viable non-biological alternatives an imperative.

Unlike the partial success of medications for improving the positive symptoms associated with schizophrenia, it is well documented that neuroleptic use – typical or atypical – does little to ameliorate the debilitating negative symptoms associated with schizophrenia (Jackson, 2005). This study builds upon this widely accepted empirical fact and provides additional evidence that it might be unrealistic to expect medication treatment to positively impact object relations domains such as self-esteem, affective

expectation of others, and identity and coherence of self, all of which are dramatically impacted by negative symptoms such as social withdrawal, depressive collapse, anhedonia, alogia, and avolition. Indeed, in these object relations domains psychodynamic psychotherapy outperformed the medication group on *all* accounts. Considering convergent validity results suggesting that such factors may also be potential predictors of hospitalization, the role of psychodynamic psychotherapy for schizophrenia may not only be clinically valuable in regard to mental health and overall quality of life, but economically beneficial as well (Warner, 2004).

It should be recalled that there were a number of factors working against the psychotherapy patients that may render the positive and comparably superior results of this type of treatment an *underestimation* of the potential effectiveness of psychodynamic psychotherapy. These factors include inexperienced therapist trainees, Caucasian therapists treating and attempting to relate/engage generally inner-city African-American patients during the time of the Detroit race riots in the 1960s, fairly chronic rather than early episode patients and, on average, only one session per week as opposed to the 2-3 times per week recommended by most clinicians experienced in the psychotherapy of schizophrenia (e.g. Sullivan, 1962). Therefore, even under these difficult circumstances psychodynamic interventions appear quite capable of providing substantial clinical benefit over and above the effects of medication-only treatment.

Due to the growing evidence of the danger and ineffectiveness of neuroleptic treatment for schizophrenia, many psychologists, and perhaps even more importantly, psychiatrists and the news media are beginning to question the degenerative as well as the 'medicate continuously and as early as possible' assumptions. In a 2006 *New York Times*

(*NYT*) interview, Dr. William Carpenter, director of the University of Maryland's Psychiatric Research Center and editor of the journal *Schizophrenia Bulletin* reports, "My personal view is that the pendulum has swung too far, and there's a knee-jerk reaction out there that says that any period off medication, *even for research*, is on the face of it unethical" (my emphasis). Oddly, in this same *NYT* interview, despite his own co-authored meta-analysis that found *no* association between untreated schizophrenia with neurocognitive deficits, morphological changes in the brain, or symptom relapse (i.e., Perkins et al., 2005), Dr. Jeffrey Lieberman responds,

> I am usually a pretty moderate person. But on this I am 110 percent emphatic: If the diagnosis is clear, not treating with medication is a huge mistake that risks the person's best chance at recovery. It's just flat out nuts.

Beyond his own meta-analytic findings refuting his comments, there are many longitudinal and psychotherapy outcome studies indicating that *not* medicating can be equally and even more helpful than medication treatments. In fact, as shown above, reviews of the schizophrenia outcome literature suggest that exposure to neuroleptic medications may *negatively* impact treatment outcome and even preclude recovery.

It is clear that due to the complexity inherent in any treatment of schizophrenia a psychiatry-psychology alliance might be greatly welcomed by frontline care providers who are often frustrated and dismayed by the limited effectiveness and all-too-prevalent side-effects of medication treatments (Breggin, 1991; Jackson, 2005; Whitaker, 1992). It has been the explicit intention of the current theoretical and empirical investigation to open a space within which biopsychiatry *and* psychology can work together, each providing the unique tools and insights of their distinct professions.

Mirroring the main premise of this dissertation project, Bola (2006) implored researchers to consider that psychotherapy outcome research – with and without adjunctive medications – should *not* be prohibited based on the assumptions deconstructed throughout this dissertation project. He writes,

> In the absence of substantive evidence of long-term harm from short periods of medication-free research in schizophrenia, a categorical prohibition of medication-free research in early episode schizophrenia on the ethical grounds of harm to human subjects should probably be reconsidered (p. 294).

Methodological designs for this type of inclusive, non-biased research have been provided by Emanuel and Miller (2001), Alanen, (1997), Seikkula et al. (2003) and Deikman and Whitaker (1979) among others to help clinicians and researchers satisfactorily test the effectiveness of drug *and* non-drug treatments whether independent of each other or combined. Indeed, the time is ripe for researchers and clinicians to respond positively to the call for fresh approaches and insights into schizophrenic suffering minus the suffocating biases that have held the field back for over half a century.

There are a few significant limitations of the present study. First, although the sample size was intentionally limited by the original investigators to control for quality of psychotherapy provided to patients, its restricted power necessitates a degree of caution when interpreting the results. Future studies might consider using external raters to assess treatment fidelity and quality rather than relying on restricted *n*-size. Such covariates would not only allow for increased sample size, but would also provide potentially interesting clinical information. Second, patients in this sample were generally urban

minorities of a low SES. Studies with a more diverse sample might provide useful information pertaining to the effects of these demographic variables. Third, future studies should include a mixed treatment group to investigate the effectiveness of combined treatments. Due to lost data, the mixed group originally included in Karon and Vandenbos (1981) could not be assessed for object relations change. Fourth, the Medication patients in this study received older, typical neuroleptics. At first glance, this might be deemed a fatal flaw of this study. However, as the CATIE study and other recent investigations reported above clearly suggest, the differences between the older and newer neurolpetics have been greatly overstated. This is true of clinical improvement as well as of troubling side-effects. Most importantly, many of these limitations will only be amenable to change if studies such as this one are received with openness and a level of scientific inquiry greatly needed if the schizophrenia research and clinical community is to accurately assess the quality of its different treatment approaches.

LIST OF REFERENCES

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Ackerman, S., Clemence, J., Weatherill, R., & Hilsenroth, J. (1999). Use of the TAT in the assessment of *DSM-IV* cluster B personality disorders. *Journal of Personality Assessment, 73*, 422-442.

Ackerman, S., Hilsenroth, M., Clemence, A., Weatherill, R., & Fowler, J. C. (2000). The effects of social cognition and object representation on psychotherapy continuation. *Bulletin of the Menninger Clinic, 64*, 386-408.

Ackerman, S., Hilsenroth, M., Clemence, A., Weatherill, R., & Fowler, J. C. (2001). Convergent validity of Rorschach and TAT scales of object relations. *Journal of Personality Assessment*, 77, 295-306.

Addonizio, G, Susman, V., & Roth, S. (1987). Neuroleptic malignant syndrome: review and analysis of 115 cases. *Biological Psychiatry, 22*, 1004-1020.

Alanen, Y. (1980). In search of the interactional origin of schizophrenia. In C. K. Hofling & J. M. Lewis (Eds.), *The family, evaluation, and treatment.* New York: Brunner/Mazel.

Alanen, Y. (1997). *Schizophrenia: Its origins and need-adapted treatment.* London: Karnac Books.

Alexiadis, M., Whitehorn, D., Woodley, H., & Kopala, L. (2002). Prolactin elevation with quetiapine. *American Journal of Psychiatry, 59*, 1608-1609.

Alliance for Human Research Protection (2006). *UK-MHRA drug-related suicide reports_yellow card.* Retrieved January 21, 2007, from www.ahrp.org/cms/content/view/90/28/).

American Psychiatric Association. (1994). *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.). Washington, DC: Author.

Antonuccio, D., Danton, W., & McClanahan, T. (2003). Psychology in the prescription era: Building a firewall between Marketing and science. *American Psychologist, 58*, 1028-1043.

Arieti, S. (1974). *Interpretation of schizophrenia*. New York: Basic Books.

Ayres, A., Busatto, G., Menezes, P., Schaufelberger, M., Coutinho, L., Murray, R., et al. (in press). Cognitive deficits in first-episode psychosis: A population-based study in Sao Paulo, Brazil. *Schizophrenia Research*.

Bachmann, S., Resch, F. & Mundt, C. (2003). Psychological treatment for psychosis: History and overview. *Journal of the American Academy of Psychoanalysis and Dynamic Psychiatry, 31*, 155-176.

Barrett, R. (1996). The psychiatric team and the social definition of schizophrenia: An anthropological study of person and illness. Cambridge, MA: Cambridge University Press.

Bauer, S., Lambert, M., and Nielsen, S. (2004). Clinical significance methods: A comparison of statistical techniques. *Journal of Personality Assessment, 82*, 60-70.

Bentall, R. (1998). Why there will never be a convincing theory of schizophrenia. In S. Rose (ed.) *From brains to consciousness: Essays on the new sciences of the mind*. London: Allen Lane, The Penguin Press.

Bezchlibynk-Butler, K., & Jeffries, J. (2002). *Clinical handbook of psychotropic drugs*. Seattle, WA: Hogrefe & Huber Publishers.

Bleuler, E. (1911). Dementia Praecox or the group of the schizophrenias. New York: International Universities Press.

Bleuler, M. (1978). The schizophrenic disorders: Long term patient and family studies. New Haven, CT: Yale University Press.

Blom, J. (2003). *Deconstructing schizophrenia*. Holland: Boom.

Bobes, J., Garcia-Portilla, M., Rejas, J., Hernandez, G., Garcia-Garcia, M., Rico-Villademoros, F., et al. (2003). Frequency of sexual dysfunction and other reproductive side-effects in patients with schizophrenia treated with risperidone, olanzapine, quetiapine, or Haloperidol: The results of the EIRE study. *Journal of Sex & Marital Therapy, 29*, 125-147.

Bockoven, J., & Solomon, H. (1975). Comparison of two five-year follow-up studies: 1947 to 1952 and 1967 to 1972. *American Journal of Psychiatry, 132*, 796-801.

Bola, J. (2006). Medication-free research in early episode schizophrenia: Evidence of long-term harm? *Schizophrenia Bulletin, 32*, 288-296.

Bonelli, R., Hofmann, P., Aschoff, A., Niederwieser, G., Heuberger, C., Jirikowski, G. et al. (2005). The influences of psychotropic drugs on cerebral cell death: female neurovulnerability to antipsychotics. *International Clinical Psychopharmacology, 20*, 145-149.

Boyer, L. & Giovacchini, P., & Hoedemaker, E. (1967). Psychoanalytic treatment of schizophrenic and characterological disorders. New York: Science House.

Boyle, M. (2002). *Schizophrenia: A scientific delusion* (2nd ed.). London: Routledge.

Bralet, M., Yon, V., Loas, G., & Noisette, C. (2000). Cause of mortality in schizophrenic

patients: Prospective study of years of a cohort of 150 chronic schizophrenic patients. *Encephele, 26*, 32-41.

Brebion, G., Amador, X., Smith, M., & Gorman, J. (1998). Memory impairment and schizophrenia: the role of processing speed. *Schizophrenia Research, 27*, 31-39.

Breggin, P. (1991). *Toxic psychiatry*. New York: St. Martin's Press.

Breggin, P. (1997). *Brain-Disabling Treatments in Psychiatry: Drugs. Electric Shock and the Role of the FDA*. New York: Springer.

Breggin, P., & Cohen, D. (1999). *Your drug may be your problem: How and why to stop taking psychiatric medication*. Boston, MA: Perseus Books.

Callahan, K., Price, J., & Hilsenroth, M. (2003). Psychological assessment of adult survivors of childhood sexual abuse within a naturalistic clinical sample. *Journal of Personality Assessment, 80,* 173-184.

Carey, B. (2006). Revisiting Schizophrenia. *New York Times*, March 21, 2006.

Carpenter, W. (1999). Diazepam treatment of early signs of exacerbation in schizophrenia. *American Journal of Psychiatry, 156*, 299-303.

Carpenter, W., McGlashan, T., and Strauss, J. (1977). The treatment of acute schizophrenia without drugs: aninvestigation of some current assumptions. A*merican Journal of Psychiatry, 134*, 14-20.

Cassens, G., Inglis, A., Appelbaum, P., & Gutheil, T. (1990). Neuroleptics: Effects on neuropsychological function in chronic schizophrenic patients. *Schizophrenia Bulletin, 16*, 477-500.

Chakos, M., Lieberman, J., Hoffman, E., Bradford, D., & Sheitman, B. (2001).

Effectiveness of second-generation antipsychotics in patients with treatment-resistant schizophrenia: A review and meta-analysis of randomized trials. *American Journal of Psychiatry, 158*, 518-526.

Chouinard, G., Jones, B., & Annable, L. (1978). Neuroleptic-induced supersensitivity psychosis. *American Journal of Psychiatry, 135,* 1409-1410

Chouinard, G., & Jones, B. (1980). Neuroleptic-induced supersensitivity psychosis: clinical and pharmacologic characteristics. *American Journal of Psychiatry, 137*, 16-20.

Ciompi, L. (1980). The natural history of schizophrenia in the long term. *British Journal of Psychiatry, 136,* 413-420.

Clarke, M., Whitty, P., Browne, S., McTigue, O., Kamali, M., Gervin, M., et al. (2006). *British Journal of Psychiatry*, *189*, 235-40.

Cleghorn, J., Kaplan, R., Szechtman, B., Szechtman, H., & Brown, G. (1990). Neuroleptic drug effects on cognitive function in schizophrenia. *Schizophrenia Research, 3*, 211-219.

Cohen, J. (1988). *Statistical Power Analysis for the Behavioral Sciences* (2nd ed.). New York: Academic Press.

Cormac, I., Jones, C., & Campbell, C. (2002). *Cognitive behaviour therapy for schizophrenia (Cochrane Review)*. Retrieved January 11, 2007, from www.cochrane.org.

Craig, T., Bromet, E., Fennig, S., Tanenberg-Karant, M., Lavelle, J., & Galambos, N.

(2000). Is there an association between duration of untreated psychosis and 24-month clinical outcome in a first admission series? *American Journal of Psychiatry, 157*, 60-66.

Cullberg, J. (1991). Integrating intensive psychosocial therapy and low dose medical treatment in a total material of first episode psychotic patients compared to "treatment as usual" a 3 year follow-up. *Acta Psychiatry Scandinavia*, *83*, 363-72.

David, A. (2004). Is early intervention a waste of valuable resources? In C. McDonald (Ed.). *Schizophrenia: Challenging the orthodox*. New York: Taylor & Francis.

Deikman, A., & Whitaker, L. (1979). Humanizing a psychiatric ward: Changing from drugs to psychotherapy. *Psychotherapy: Theory, Research, and Practice, 16*, 204-214.

de Leon, J. (in press). The effect of atypical versus typical antipsychotics on tardive dyskinesia : A naturalistic study. *European Archives of Psychiatry and Neuroscience.*

Dixon, L., Weiden, P., Delahanty, J., Goldberg, R., Postrado, L., Lucksted, A., et al. (2000). Prevalence and correlates of diabetes in national schizophrenia samples. *Schizophrenia Bulletin, 26*, 903-912.

Drake, E. & Sederer, L. (1986) The adverse effects of intensive treatment of chronic schizophrenia. *Comprehensive Psychiatry 27*, 313-326.

Dunham, H. (1965). Community and schizophrenia: An epidemiological analysis. *Lafayette Clinical Monographs in Psychiatry.* Detroit, MI: Wayne St. University Press.

Emanuel, E., & Miller, F. (2001). The ethics of placebo-controlled trials: A middle

ground. *New England Journal of Medicine, 345*, 915-919.

Epstein, L., Morgan, R., & Reynolds, L. (1962). An Approach to the Effect of Ataraxic Drugs on Hospital Release Rates. *American Journal of Psychiatry, 119*, 36-47.

Faries, D., Heiligenstein, J., Tollefson, G., & Potter, W. (2001). The double-blind variable placebo lead-in period: Results from two antidepressant clinical trials. *Journal of Clinical Psychopharmacology, 21*, 561-568.

Feldman, M., & Drasgow, J. (1951). A visual-verbal test for schizophrenia. *Psychiatric Quarterly Supplement, 25*, 55-64.

Field, N., Sturgeon, S., Puryear, R., Hibbard, S., & Horowitz, M. (2001). Object relations as a predictor of adjustment in conjugal bereavement. *Development and Psychopathology, 13*, 399-412.

Fleiss, J. (1981). *Statistical methods for rates and proportions* (2nd ed.). New York: John Wiley & Sons.

Ford, J., Fisher, P., & Larson, L. (1997). Object relations as a predictor of treatment outcome with chronic Post Traumatic Stress Disorder. *Journal of Consulting and Clinical Psychology, 65*, 547-559.

Fowler, J. C., Ackerman, S., Speanburg, S., Bailey, A., & Blagys, M. (2004). Personality and symptom change in treatment-refractory inpatients:Evaluation of the phase model of change using Rorschach, TAT, and *DSM-IV* Axis V. *Journal of Personality Assessment, 83*, 306-322.

Fowler, J. C., Hilsenroth, M. J., Handler, L. (1995). Early memories: An exploration of theoretically derived queries and their clinical utility. *Bulletin of the Menninger Clinic*, *52*, 31-52.

Fromm-Reichmann, F. (1950). Transference problems in schizophrenics. *Psychoanalytic Quarterly, 8*, 412-426.

Gabbard, G., & Freedman, R. (2006). Psychotherapy in the Journal: What's missing? *American Journal of Psychiatry, 163*, 182-184.

Gardos, G., & Cole, J. (1976). Maintenance antipsychotic therapy: Is the cure worse than the disease. *American Journal of Psychiatry, 133*, 32-36.

Geddes, J., Freemantle, N., Harrison, P., & Bebbington, P. (2000). Atypical antipsychotics in the treatment of schizophrenia: Systematic overview and met-regression analysis. *British Medical Journal, 321*, 1371-1376.

Gilbertson, M. & van Kammen, D. (1997). Recent and remote memory dissociation: Medication effects and hippocampal function in schizophrenia. *Biological Psychiatry, 42*, 585-595.

Gilman, S. (1982). *Seeing the insane*. New York: Wiley and Brunner/Mazel Publishers.

Gilmore, J., & Murray, R. (2006). Prenatal and perinatal factors. In J. Lieberman, T. Stroup, & D. Perkins (Eds.). *Textbook of schizophrenia*. Washington, DC: American Psychiatric Publishing, Inc.

Glazer, W. (2000). Extrapyramidal side effects, tardive dyskinesia, and the concept of atypicality. *Journal of Clinical Psychiatry, 61*, 16-21.

Goldstein, D. (2003) Pharmacogenetics in the laboratory and the clinic. *New England Journal of Medicine, 348*, 553-556.

Gottdiener, W., & Haslam, N. (2002). The benefits of individual psychotherapy for

people diagnosed with schizophrenia: A meta-analytic review. *Ethical Human Sciences and Services, 4*, 163-187.

Gottdiener, W., & Haslam, N. (2003). A critique of the methods and conclusions in the Patient Outcome Research Team (PORT) Report on psychological treatments for schizophrenia. *Journal of the American Academy of Psychoanalysis and Dynamic Psychiatry, 31*, 191-208.

Gottesman, I. (1999). *Schizophrenia Genesis: The origins of madness*. New York: Freeman.

Grinspoon, L., Ewalt, J., & Shader, R. (1972). *Schizophrenia, pharmacology, and psychotherapy*. Bethesda, MD: Williams and Wilkins.

Gur, R., Maany, V., Mozley, D., Swanson, C., Bilker, W., & Gur, R. (1998). Subcortical MRI volumes in neuroleptic-naïve and treated patients with schizophrenia. *American Journal of Psychiatry, 155*, 1711-1717.

Harding, C. (1995). The interaction of biopsychosocial factors, time, and course of schizophrenia. In C. L. Shiriqui & H. A. Nasrallah (Eds.), *Contemporary issues in the treatment of schizophrenia* (pp. 653-681). Washington, DC: American Psychiatric Press.

Harding, C., & Brooks, G., Ashikaga, T., Strauss, J., & Breier, A. (1987a). The Vermont longitudinal study of persons with severe mental illness, I: Methodology, study sample, and overall status 32 years later. *American Journal of Psychiatry, 144*, 718-726.

Harding, C., & Brooks, G., Ashikaga, T., Strauss, J., & Breier, A. (1987b) The Vermont

longitudinal study of persons with severe mental illness, II: Long-term outcome of subjects who retrospectively met DSM-III criteria for schizophrenia. *American Journal of Psychiatry, 144*, 727-7235.

Harding, C., & Zahniser, J. (1994). Empirical correction of seven myths about schizophrenia with implications for treatment. *Acta Psychiatry Scandanavia Supplement, 384*, 140-146.

Harding, C., Zubin, J., & Strauss, J. (1992). Chronicity in schizophrenia: Revisited. *British Journal of Psychiatry, 161*, 27-37.

Harman, C. (2003). *The diagnosis and stigma of schizophrenia*. Brookings, OR: Old Court Press.

Harrison, G., Hopper, K., Craig, T., Laska, E., Wanderling, J., Dube, K., et al. (2001). Recovery from psychotic illness: A 15- and 25-year international follow-up study. *British Journal of Psychiatry, 178*, 506-517.

Hayashi, T., Nishikawa, T., Koga, I., Uchida, Y. & Yamawaki, S. (1996). Prevalence of and risk factors for respiratory dyskinesia. *Clinical Neuropharmacology, 19*, 390-398.

Healy, D. Harris, M., Michael, P., Catell, D. Savage, M., Chalasani, P., et al. (2005). Service utilization in 1896 and 1996: Morbidity and mortality data from North Wales. *History of Psychiatry, 16*, 27-41.

Healy, D., Harris, M., Tranter, R., Gutting, P., Austin, R., Jones-Edwards, G., et al. (2006). Lifetime suicide rates in treated schizophrenia: 1875–1924 and 1994–1998 cohorts compared. *The British Journal of Psychiatry*, *188*, 223-228.

Hegarty, J., Baldessarini, R., Tohen, M., Waternaux, C., & Oepen, G. (1994). One

hundred years of schizophrenia: A meta-analysis of the outcome literature. *American Journal of Psychiatry, 151*, 1409-1416.

Henderson, D., Nguyen, D., Copeland, P., Hayden, D., Borba, C., Louie, P., et al. (2005). Clozapine, diabetes mellitus, hyperlipidemia, and cardiovascular risks and mortality: Results of a 10-year naturalistic study. *Journal of Clinical Psychiatry, 66*, 1116-1121.

Hibbard, S., Hilsenroth, M., Hibbard, J., & Nash, M. (1995). A validity study of two projective object representations measures. *Psychological Assessment, 7,* 432-439.

Hilsenroth, M., Ackerman, S., Blagys, M., Baity, M., & Mooney, M. (2003). Short-term psychodynamic psychotherapy for depression: An examination of statistical, clinically significant, and technique specific change. *Journal of Nervous and Mental Disease, 191,* 349-357.

Hilsenroth, M., Stein, M., & Pinsker, J. (2004). *Social Cognition and Object Relations Scale: Global Rating Method (SCORS-G)*. Unpublished manuscript, The Derner Institute of Advanced Psychological Studies, Adelphi University, Garden City, NY.

Ho, B., Alicata, D., Ward, J., Moser, D., O'Leary, D., Arndt, S., et al. (2003). Untreated initial psychosis: Relation to cognitive deficits and brain morphology in first-episode schizophrenia. *American Journal of Psychiatry, 160*, 142-148.

Hoff, A., Sakuma, M., Razi, K., Heydebrand, G., Csernansky, J., & DeLisi, L. (2000). Lack of association between duration of untreated illness and severity of cognitive and structural brain deficits at the first episode of schizophrenia. *American*

Journal of Psychiatry, 157, 1824-1828.

Hopper, K., Harrison, G., Aleksander, J. & Sartiorius, N. (2007) *Recovery from Schizophrenia: An International Perspective: A Report from the WHO Collaborative Project, the International Study of Schizophrenia*. London: Oxford University Press.

Hopper, K., & Wanderling, J. (2000). Revisiting the developed versus developing country distinction in course and outcome in schizophrenia: Results from ISoS, the WHO collaborative follow-up project. International Study of Schizophrenia. *Schizophrenia Bulletin, 26, 835-846.*

Hubbard, J., Ganes, D., & Midha, K. (1987). Prolonged pharmacologic activity of neuroleptic drugs. *Archives of General Psychiatry, 44*, 99-100.

Huber, G., Gross, G., & Schuttler, R. (1975). A long-term follow-up study of schizophrenia: Psychiatric course of illness and prognosis. *Acta Psychiatrica Scandinavica, 52*, 49-57.

Huprich, S., & Greenberg, R. (2003). Advances in the assessment of object relations in the1990s. *Clinical Psychology Review, 23*, 665-698.

Irwin, M. (2004a). Reversal of schizophrenia without neuroleptics. *Ethical Human Psychology and Psychiatry, 6*, 53-68.

Irwin, M. (2004b). Treatment of schizophrenia without neuroleptics: Psychosocial interventions versus neuroleptic treatment. *Ethical Human Psychology and Psychiatry, 6,* 99-110.

Jablensky, A., Sartorius, N., Ernberg, G., Anker, M., Korten, A., Cooper, J., et al. (1992).

Schizophrenia: Manifestations, incidence, course in different cultures, a World Health Organization ten country study. *Psychological Medicine Monograph Supplement, 20*, 1-95.

Jackson, G. (2005). Rethinking psychiatric drugs: A guide for informed consent. Bloomington, IN: Author House.

Jacobson, N., Roberts, L., Berns, S., & McGlinchey, J. (1999). Methods for defining and determining the clinical significance of treatment effects: Description, application, and Alternatives. *Journal of Consulting and Clinical Psychology, 67,* 300-307.

Jacobson, N., & Truax, P. (1991). Clinical significance: A statistical approach to defining meaningful change in psychotherapy research. *Journal of Consulting and Clinical Psychology, 59,* 12-19.

Jeste, D., Lacro, J., Palmer, B., Rockwell, E., Harris, M., & Caliguri, M. (1999). Incidence of tardive dyskinesia in early stages of low-dose treatment with typical neuroleptics in older patients. *American Journal of Psychiatry, 156*, 309-311.

Joukamaa, M., Heliovaara, M., Knekt, P., Aromaa, A., Raitasalo, R., & Lehtinen, V. (2006). Schizophrenia, neuroleptic medication and mortality. *The British Journal of Psychiatry*, *188*, 122-127.

Karon, B. (1989). Psychotherapy versus medication for schizophrenia: Empirical Comparisons. In S. Fisher & R. Greenberg (Eds.), *The limits of biological treatments for psychological distress* (pp. 106-149). Hilsdale, NJ: Erlbaum.

Karon, B. (1994). Effective Psychoanalytic Therapy of Schizophrenia and Other Severe Disorders (videotape and supporting written material). *The APA Psychotherapy Videotape Series.* Washington, DC: American Psychological Association.

Karon, B., & VandenBos, G. (1981). *Psychotherapy of Schizophrenia: The Treatment of Choice*. New York: Aronson.

Karon, B. (2003). The tragedy of schizophrenia without psychotherapy. *Journal of the American Academy of Psychoanalysis and Dynamic Psychiatry,* 31, 89-118.

Kaslow, N., Reviere, S., Chance, S., Rogers, J., Hatcher, C., Wasserman, F., et al. (1997). An empirical study of the psychodynamics of suicide. *Journal of the American Psychoanalytic Association, 46,* 777-796.

Kasper, S., & Resinger, E. (2003). Cognitive effects and antipsychotic treatment. *Psychoneuroendicrinology, 28*, 27-38.

Keck, P. (1989). Time course of antipsychotic effects of neuroleptic drugs. *American Journal of Psychiatry, 146*, 1289-1292.

Khan, A., Khan, S., Leventhal, R., & Brown, W. (2001). Symptom reduction and suicide risk among patients treated with placebo in antipsychotic clinical trials: An analysis of the Food and Drug Administration Database. *American journal of Psychiatry, 158*, 1449-1454.

Khan, A., Warner, H., & Brown, W. (2000). Symptom reduction and suicide risk in patients treated with placebo in antidepressant clinical trials. *Archives of general Psychiatry, 57*, 311-317.

King, D. (1994). Psychomotor impairment and cognitive disturbances induced by neuroleptics. *Acta Psychiatrica Scandanavica, 89*, 53-58.

Koehler, B. (2006). *Genetics and the future of psychiatry*. Retrieved January 15, 2007, from www.isps-us.org/Koehler/genetics_future.htm.

Kornhuber, J., & Weller, M. (1994). Neuroleptic malignant syndrome. *Current Opinions in Neurology, 7*, 353-357.

Kua, J., Wong, K., Kua, E., & Tsoi, W. (2003). A 20-year follow-up study on schizophrenia in Singapore. *Acta Psychiatrica Scandinavica, 108*, 118-125.

Leff, J., Sartorius, N., Jablensky, A., Korten, A., & Ernberg, G. (1992). The international pilot study of schizophrenia: Five-year follow-up findings. *Psychological Medicine, 22*, 131-145.

Lehtinen, V., Aaltonen, J., Koffert, T., Rakkolainen, V., & Syvalahti, E. (2000). Two-year outcome in first episode psychosis treated according to an integrated model. Is immediate neuroleptisation always needed? *European Psychiatry, 15*, 312-20.

Lehman, A., & Steinwachs, D. (1998). At issue: Translating research in to practice: The schizophrenia patient outcomes research team (PORT) treatment recommendations. *Schizophrenia Bulletin, 24*, 1-10.

Lehrman, N. (1960). A state hospital population five years after admission: A yardstick for evaluative comparison of follow-up studies. *Psychiatric Quarterly, 34*, 658-681.

Leung, C., Chung, D., Kam, I., & Wat, K. (2000). Multiple rib fractures secondary to severe tardive dystonia and respiratory dyskinesia. *Journal of Clinical Psychiatry, 61*, 215-216.

Lewontin, R. (2000). *The triple helix: Gene, organism, and environment*. Cambridge, MA: Harvard University Press.

Lieberman, J., Tollefson, G., Tohen, M., Green, A., Gur, R., Kahn, R. et al. (2003). Comparative efficacy and safety of atypical and conventional antipsychotic drugs in first-episode psychosis: A randomized, double-blind trial of olanzapine versus haloperidol. *American Journal of Psychiatry, 160*, 1396-1404.

Lieberman, J., Stroup, T., McEvoy, J., Swartz, M., Rosenheck, R., Perkins, D., et al. (2005). Effectiveness of antipsychotic drugs in patients with chronic schizophrenia. *New England Journal of Medicine*, *353*, 1209-1223.

Malla, A., Norman, R., Scholten, D., Townsend, L., Manchanda, R., Takhar, J., et al. (2004). A comparison of two novel antipsychotics in first episode non-affective psychosis: One-year outcome on symptoms, motor side-effects, and cognition. *Psychiatry Research, 129*, 159-169.

Malmberg, L., & Fenton, W. (2001). *Individual psychodynamic psychotherapy and psychoanalysis for schizophrenia and severe mental illness (Cochrane Review).* Retrieved January 11, 2007, from www.cochrane.org.

Mathews, S. (1979). A non-neuroleptic treatment for schizophrenia: analysis of the two-year post-discharge risk of relapse. *Schizophrenia Bulletin, 5*, 322-332.

May, P., Tuma, H., Yale, C., Potepan, R., & Dixon, W. (1976). Schizophrenia: A follow -up study of results of five forms of treatment. *Archives of General Psychiatry, 33*, 481-486.

May, P., Tuma, H., Yale, C., Potepan, R., & Dixon, W. (1981). Schizophrenia: A follow -up study of results of treatment. *American Journal of Psychiatry, 38*, 776-784.

McCartan, D., Bell, R., Green, J., Campbell, C., Trimble, K., Pickering, A., et al. (2001).

The differential effects of chlorpromazine and haloperidol on latent inhibition in healthy volunteers. *Journal of Psychopharmacology, 15*, 96-104.

McGlashan, T. (2006). Is active psychosis neurotoxic? *Schizophrenia Bulletin, 32*, 609-613.

McEvoy, J., Meyer, J., Goff, D., Nasrallah, H., Davis, S., & Sullivan, L. (2005). Prevalence of the metabolic syndrome in patients with schizophrenia: Baseline results from the Clinical Antipsychotic Trials of Intervention Effectiveness (CATIE) schizophrenia trial and comparison with national estimates from NAHNES III. *Schizophrenia Research, 80*, 19-32.

Mehta, M, Manes, F., Magnolfi, G. Sahakian, B. & Robbins, T. (2004). Impaired set-shifting and dissociable effects on tests of spatial working memory following the dopamine D2 receptor antagonist sulpride in human volunteers. *Psychopharmacology, 176*, 331-342.

Menezes, N., Arenovich, T., & Zipursky, R. (2006). A systematic review of longitudinal studies of first-episode psychosis. *Psychological Medicine, 36*, 1349-1362.

Meyer, J. (2001). Effects of atypical antipsychotics on weight and serum lipid levels. *Journal of Clinical Psychiatry, 62*, 27-34 (discussion 40-41).

Mojtabai, R., Nicholson, R., & Carpenter, B. (1998). Role of psychosocial treatments in management of schizophrenia: A meta-analytic review of controlled outcome studies. *Schizophrenia Bulletin, 24*, 569-587.

Moncrieff, J. (2001). Are antidepressants overrated? A review of methodological problems in antidepressant trials. *Journal of Nervous and Mental Disease, 189*, 288-295.

Moncrieff, J. (2006). Does antipsychotic withdrawal provoke psychosis? Review of the literature on rapid onset psychosis (supersensitivity psychosis) and withdrawal-related relapse. *Acta Psychiatrica Scandinavica, 114*, 3-13.

Morgan, M., Scully, P., Youssef, H., Kinsella, A., Owens, J., & Waddington, J. (2003). Prospective analysis of premature mortality in schizophrenia in relation to health service engagement: a 7.5-year study within an epidemiologically complete, homogeneous population in rural Ireland. *Psychiatry Research, 117*, 127-135.

Minzenberg, M., Poole, J., Benton, C., & Vinogradov, S. (2004). Association of anticholinergic load with impairment of complex attention and memory in schizophrenia. *American Journal of Psychiatry, 161*, 116-124.

Moncrieff, J. (2006). Does antipsychotic withdrawal provoke psychosis? Review of the literature on rapid onset psychosis (supersensitivity psychosis) and withdrawal-related relapse. *Acta Psychiatrica Scandanavica, 1*, 1-11.

Mosher, L., Vallone, R., & Menn, A. (1979). Community residential treatment for schizophrenia: two year follow-up. *Hospital and Community Psychiatry*, *29*, 715-723.

Miller, D., McEvoy, J., Davis, S., Caroff, S., Saltz, B., Chakos, M. et al. (2005). Clinical correlates of tardive dyskinesia in schizophrenia: Baseline data from the CATIE schizophrenia trial. *Schizophrenia Research, 80*, 33-43.

Muller, P., & Seeman, P. (1978). Dopaminergic Supersensitivity after Neuroleptics: Time-Course and Specificity. *Psychopharmacology, 60*, 1-11.

Murphy, H., & Raman, A. (1971). The chronicity of schizophrenia in indigenous

Tropical peoples: Results of a 12 year follow-up survey in Mauritius. *British Journal of Psychiatry, 118*, 489-497.

Murray, H. (1943). Manual for the Thematic Apperception Test. Cambridge, MA: Harvard University Press.

Ogawa, K., Miya, M., Watarai, A., Nakazawa, M., Yuasa, S., & Utena, H. (1987). A long-term follow-up study of schizophrenia in Japan. *British Journal of Psychiatry, 151*, 758-765.

Oosthuizen, P., Emsley, R., Maritz, J., Turner, J., & Keyter, N. (2003). Incidence of tardive dyskinesia in first-episode psychosis patients treated with low-dose haloperidol. *Journal of Clinical Psychiatry, 64*, 1075-1080.

Osby, U., Correia, N., Brandt, L., Ekbom, A., & Sparen, P. (2000). Mortality and causes of death in schizophrenia in Stockholm county Sweden. *Schizophrenia Research, 45*, 21-28.

Osby, U., Correia, N., Ekbom, A. & Sparen, P. (2000). Time trends in schizophrenia mortality in Stockholm County, Sweden: Cohort Study. *British Medical Journal, 321*, 483-484.

Owens, D. (1999). Guide to the extrapyramidal side-effects of antipsychotic drugs New York: Cambridge University Press.

Papageorgiou, C., Oulis, P., Vasios, C., Matsopoulos, G., Uzunoglu, A., Rabavilas, A. et al., (2003). Do atypical antipsychotics fail to exert cognitive sparing effects. *Neuroreport, 14,* 505-509.

Paul, G., & Lentz, R. (1977). *Psychosocial treatment of chronic mental patients: Milieu vs. social learning programs*. Cambridge, MA: Harvard University Press.

Paul, G., Tobias, L., & Holly, B. (1972). Maintenance psychotropic drugs in the presence of active treatment programs. A "triple-blind" withdrawal study with long0term mental patients. *Archives of General Psychiatry, 27*, 106-115.

Perkins, D., Gu, H., Boteva, K., & Lieberman, J. (2005). *American Journal of Psychiatry, 162*, 1785-1804.

Peters, E., Hilsenroth, M., Eudell-Simmons, E., Blagys, M., & Handler, L. (2006). Reliability and validity of the SCORS in clinical use. *Psychotherapy Research*, *16*, 606-614.

Porcerelli, J., Cogan, R., & Hibbard, S. (1998). Cognitive and affective representations of people and MCMI-II personality psychopathology. *Journal of Personality Assessment 70,* 535-540.

Prien, R., Cole, J., & Belkin, N. (1968). Relapse in chronic schizophrenics following abrupt withdrawal of tranquillizing medication. *British Journal of Psychiatry*, *115*, 679-686.

Prien, R., Levine, J., & Switalski, R. (1971). Discontinuation of chemotherapy for chronic schizophrenics. *Hospital and Community Psychiatry*, *22*, 20-23.

Rachlin, H., Goldman, G., Guritz, M., Lurie, A., and Rachlin, L. (1956). Follow-Up study of 317 patients discharged from Hillside Hospital in 1950, *Journal of Hillside Hospital, 5*, 17- 40.

Rammsayer, T. (1997). Are there dissociable roles of the mesostriatal and mesolimbocortical dopamine systems on temporal information processing in humans. *Neuropsychobiology, 35*, 36-45.

Rappaport, M., Hopkins, H., Hall, K., Belleza, T., & Silverman, J. (1978). Are there

schizophrenics for whom drugs may be unnecessary or contraindicated? *International Pharmacopsychiatry*, *13*, 100-111.

Read, J. & Ross, C. (2003). Psychological trauma and psychosis: Another reason why people diagnosed schizophrenic must be offered psychological therapies. *Journal of the American Academy of Psychoanalysis and Dynamic Psychiatry, 31*, 247-268.

Read, J., Perry, B., Moskowitz, A., & Connolly, J. (2001). The contribution of early traumatic events to schizophrenia in some patients: A traumagenic neurodevelopmental model. *Psychiatry, 64*, 319-345.

Reilly, J., Ayis, S., Ferrier, I., Jones, S., & Thomas, S. (2002). Thioradizine and sudden unexplained death in psychiatric in-patients. *British Journal of Psychiatry, 180*, 515-522.

Ridout, F., & Hindmarch, I. (2003). The effects of acute doses of fexofenadine, promethazine, and placebo on cognitive and psychomotor function in healthy Japanese volunteers. *Annals of Allergy, Asthma, and Immunology, 90*, 404-410.

Rogers, C., Gendlin, E., Kiesler, D., & Traux, C. (1967). The therapeutic relationship and its impact: A study of psychotherapy with schizophrenics. Madison, WI: University of Wisconsin Press.

Rosenheck, R., Perlick, D., Bingham, S., Liu-Mares, W., Collins, J. & Stuart, W. (2004). Effectiveness and cost of olanzapine and haloperidol in the treatment of schizophrenia. *Journal of the American Medical Association, 290*, 2693-2702.

Ross, C., & Pam, A. (1995). *Pseudoscience in biological psychiatry: Blaming the body.* Oxford, England: John Wiley & Sons.

Safer, D. (2002). Design and reporting modifications in industry sponsored comparative psychopharmacology trials. *Journal of Nervous and Mental Disease, 190*, 583-592.

Scherer, H., Bedard, M., Stip, E., Paquet, F., Richer, F., Beriault, M., et al. (2004). Procedural learning in schizophrenia can reflect the pharmacologic properties of the antipsychotic treatments. *Cognitive and behavioral Neurology, 17*, 32-40.

Schindler, R. (1980). The change of long term progress of psychotics after psychotherapy. *Psychiatra Clinica, 13,* 206-216.

Schooler, N., Goldberg, S., Boothe, H., & Cole, J. (1967). One year after discharge: community adjustment of schizophrenic patients. *American Journal of Psychiatry, 123*, 986-995.

Searles, H. (1960). *The nonhuman environment in normal development and in schizophrenia*. New York: International Universities Press.

Searles, H. (1965). *Collected Papers on Schizophrenia and Related Subjects*. New York: International Universities Press.

Segal, H., Westen, D., Lohr, N., & Silk, K. (1993). Clinical assessment of object relations and social cognition using stories told to the picture arrangement subtest of the WAIS-R. *Journal of Personality Assessment, 61*, 58-80.

Segarra, R., Gutierrez, M., Eguiluz, I., Ojeda, N., & Fernandez, C. (2006). Controversies about duration of untreated psychosis as independent prognostic variable of the evolutive course of schizophrenic psychoses. *Actas Españolas de Psiquiatría, 34*, 377-385.

Seikkula, J., Alakare, B., & Aaltonen, J. (2000). A two-year follow-up on open dialogue

treatment in first episode psychosis: need for hospitalization and neuroleptic medication decreases. *Social and Clinical Psychiatry, 10*, 20-29.

Seikkula, J., Alakare, B., Aaltonen, J., Holma, J., Rasinkangas, A., & Lehtinen, V. (2003). Open Dialogue Approach: Treatment principles and preliminary resulats of a two-year follow-up on first episode schizophrenia. *Ethical and Human Sciences and Services, 5,* 163-182.

Shrout, P., & Fleiss, J. (1979). Intraclass correlations: Uses in assessing rater reliability. *Psychological Bulletin, 8,* 420-428.

Siebert, A. (1999). Unethical psychiatrists misrepresent what is known about schizophrenia. *Journal of Ethical Human Sciences and Services, 1*, 179-189.

Silver, A. (2003). The psychotherapy of schizophrenia: Its place in the modern world. *Journal of the American Academy of Psychoanalysis and Dynamic Psychiatry, 31*, 325-241.

Silverstein, S., Spaulding, W., & Menditto, A. (2006). *Schizophrenia.* Cambridge, MA: Hogrefe & Huber Publishers.

Slavin, J., Stein, M., Pinsker-Aspen, J., & Hilsenroth, M. (in press). Early memories from outpatients with and without a history of childhood sexual abuse. *Journal of Loss and Trauma.*

Smith, M., Glass, G., & Miller, T. (1980). *The benefits of psychotherapy*. Baltimore, MD: The Johns Hopkins University Press.

Speer, D. (1992). Clinically significant change: Jacobson and Truax (1991) revisited. *Journal of Consulting and Clinical Psychology, 60*, 402-408.

Stip, E. (2002). Happy birthday neuroleptics! 50 years later: la folie du doute. *European*

Psychiatry, 17, 1-5.

Straus, S., Bleumink, G., Dieleman, J., van der Lei, J., Jong, G., Kingma, J., et al. (2004). Antipsychotics and the risk of sudden cardiac death. *Archives of Internal Medicine, 164*, 1293-1297.

Stricker, G., & Gooen-Piels, J. (2004). Projective assessment of object relations. In M. J. Hilsenroth and D. L. Segal (Eds.), *Handbook of psychological assessment: Vol. 2 Personality Assessment* (pp. 449-465). Hillsdale, NJ: Wiley.

Sullivan, H.S. (1953). *The interpersonal theory of psychiatry*. New York: W.W. Norton & Co.

Sullivan, H. S. (1962). Schizophrenia as a human process. New York: W.W. Norton & Co.

Sullivan, P., Owen, M., O'Donovan, M., & Freedman, R. (2006). In J.Lieberman, T. Stroup, & D. Perkins (Eds.). *Textbook of schizophrenia*. Washington, DC: American Psychiatric Publishing, Inc.

Summers, F. (1994). *Object Relations Theories and Psychopathology*. Hillsdale, NJ:Analytic Press.

Sweeney, J., Keilp, J., Haas, G., Hill, H., & Weiden, P. (1991). Relationships between medication treatments and neuropsychological test performance in schizophrenia. *Psychiatry Research, 37*, 297-308.

Tienari, P., Sorri, A., Lahti, I., Naarala, M., Wahlberg, K., Ronkko, T., et al. (1985). The Finnish adoptive study of schizophrenia. *Yale Journal of Biology and Medicine, 58*, 227-237.

Thornley, B., & Adams, C. (1998). Content and quality of 2000 controlled trials in

schizophrenia over 50 years. *British Medical Journal, 317*, 1181-1184.

Tramantano, G., Javier, R., & Colon, M. (2003). Discriminating subgroups of borderline personality disorder: An assessment of object representations. *The American Journal of Psychoanalysis, 63*, 149-175.

Tsuang, M., Woolson, R., & Fleming, J. (1979). Long-term outcome of major psychoses. *Archives of General Psychiatry, 36*, 1295-1301.

Turkington, D., Kingdon, D., Rathod, S., Hammond, K., Pelton, J., & Mehta, R. (2006). Outcomes of an effectiveness trial of cognitive-behavioural intervention by mental health nurses in schizophrenia. *British Journal of Psychiatry, 189*, 36-40.

ver Eecke, W. (2003). The role of psychoanalytic theory and practice in understanding and treating schizophrenia: A rejoinder to the PORT report's condemnation of psychoanalysis. *Journal of the American Academy of Psychoanalysis and Dynamic Psychiatry, 31*, 11-30.

Viguera, A., Baldessarini, R., Hegarty, J., van Kammen, D., & Tohen, M. (1997). Clinical risk following abrupt and gradual withdrawal of maintenance neuroleptic treatment. *Archives of General Psychiatry, 54*, 49-55.

Vitiello, B., Martin, A., Hill, J., Mack, C., Molchan, S., Martinez, R., et al. (1997) Cognitive and behavioral effects of cholinergic, dopaminergic, and serotonergic blockade in humans. *Neuropsychopharmacology, 16*, 15-24.

Volkan, V. (1995). *The infantile psychotic self and its fates: Understanding and treating schizophrenics and other difficult patients*. Northvale, NJ: Jason Aronson Inc.

Waddington, J., & Youssef, H. (1986). An unusual cluster of tardive dyskinesia in

schizophrenia: association with cognitive dysfunction and negative symptoms. *American Journal of Psychiatry, 143*, 1162-1165.

Waddington, J., Youssef, H., & Kinsella, A. (1998). Mortality in schizophrenia: antipsychotic polypharmacy and absence of adjunctive anticholinergics over the course of a 10-year prospective study. *British Journal of Psychiatry, 173*, 325-329.

Warner, R. (2004). *Recovery from schizophrenia: Psychiatry and political economy* (3rd ed.). New York: Brunner-Routledge.

Wechsler, D. (1955). *Manual for the Wechsler Adult Intelligence Scale*. Oxford, England: Psychological Corporation.

Westen, D. (1995). *Social Cognition and Object Relations Scale: Q-Sort for Projective Stories*. Unpublished manuscript, Harvard University Medical School, Cambridge, Massachusetts.

Westen, D., Lohr, N., Silk, K., Gold, L., & Kreber, K. (1990). Object relations and social cognition in borderlines, major depressives, and normals: A Thematic Apperception Test analysis. *Psychological Assessment, 2*, 355-36.

Westen, D., Novotny, C., & Thompson-Brenner, H. (2004). The empirical status of empirically supported psychotherapies: Assumptions, findings, and reporting in controlled clinical trials. *Psychological Bulletin, 130*, 631-663.

Westen, D., & Gabbard, G. (1999). Psychoanalytic approaches to personality. In L. Pervin & O. John (Eds.), *Handbook of personality: Theory and research* (pp. 57-101). New York: Guilford Press.

Whitaker, L. (1992). *Schizophrenic Disorders; Sense and Nonsense in Conceptualization,*

Assessment, and Treatment. New York: Plenum Press.

Whitaker, R. (2002). *Mad in America*. Boston, MA: Perseus.

Whitaker, R. (2005). Anatomy of an epidemic: psychiatric drugs and the astonishing rise of mental illness in America. *Ethical Human Psychology and Psychiatry, 7*, 23-35.

Will, O. (1968). Schizophrenia nad psychotherapy. In J. Marmor (Ed.). *Modern psychoanalysis: New directions and perspectives*. New York: Basic Books.

Wise, E. (2004). Methods for analyzing psychotherapy outcomes: A review of clinical significance, reliable change, and recommendations for future directions. *Journal of Personality Assessment, 82*, 50-59.

Wolkowitz, O. (1991). Benzodiezapines in the treatment of schizophrenia: A Review and reappraisal. *American Journal of Psychiatry, 148*, 714-726.

Wynne, L., & Singer, M. (1963). Thought disorder and family relations of schizophrenics. II: A classification of forms of thinking. *Archives of General Psychiatry, 9*, 199-206.

APPENDIX A

Table 1. Most Commonly Prescribed Neuroleptic Medications for the Treatment of Schizophrenia

	Generic Name	Brand Name
Typical Neuroleptics	chlorpromazine	Thorazine
	fluphenazine	Prolixin
	haloperidol	Haldol
	thiothixene	Navane
	trifluoperazine	Stelazine
	perphenazine	Trilafon
	thioridazine	Mellaril
Atypical Neuroleptics	aripiprazole	Abilify
	risperidone	Risperdal
	clozapine	Clozaril
	olanzapine	Seroquel
	ziprasidone	Geodon

Table 2. Example of Non-Equivalent Dosing

	Olanzapine (atypical neuroleptic)	Haloperidol (typical neuroleptic)
Low Dose	5mg +/- 2.5 mg	10-20 mg
Medium Dose	10 mg +/- 2.5 mg	10-20 mg
High Dose	15 mg +/- 2.5 mg	10-20 mg

Table 3. Interrater Reliability of the SCORS Variables

SCORS Variable	ICC (*1*,*1*)[aj]
COM[b]	.71
AFF[c]	.71
EIR[d]	.75
EIM[e]	.71
USC[f]	.74
AGG[g]	.83
She	.76
ICS[i]	.77

Note. $n = 20$. [a]ICC = intraclass correlation coefficients (*1*,*1*); (Shrout & Fleiss, 1979) one-way random effects model; [b]COM = Complexity of Representations; [c]AFF = Affect Tone of Representations; [d]EIR = Emotional Investment in Relationships; [e]EIM = Emotional Investment in Values and Morality; [f]USC = Understanding of Social Causality; [g]AGG = Experience and Management of Aggressive Impulses; [h]SE = Self-Esteem; [i]ICS = Identity and Coherence of the Self. [j]ICC < .40 = poor; .40 - .59 = fair; .60 - .74 = good; ≥ .75 = excellent.

Table 4. SCORS Means of Outpatients Experiencing Mild to Moderate Distress as Reported in Peters et al. (2006)

SCORS Variable	*Mean*	*SD*
COM[a]	4.02	.96
AFF[b]	3.29	1.02
EIR[c]	3.56	.94
USC[d]	3.94	.98
SE[e]	3.12	.81
ICS[f]	3.74	.94

Note. $n = 90$. [a]COM = Complexity of Representations; [b]AFF = Affect Tone of Representations; [c]EIR = Emotional Investment in Relationships; [d]USC = Understanding of Social Causality; [e]SE = Self-Esteem; [f]ICS = Identity and Coherence of the Self.

Table 5. Convergent and Divergent Validity of the SCORS with a Schizophrenic Sample

	COM[f]		AFF[g]		EIR[h]		USC[i]		SE[j]		ICS[k]	
	r[l]	*p*	*r*	*p*	*r*	*p*	*r*	*p*	*r*	*p*	*r*	*p*
Hospital[a]	-.57	.009	-.38	.10	-.39	.09	-.66	.002	-.40	.08	-.68	.001
Verbal IQ[b]	.52	.03	-.04	.90	.08	.76	.45	.01	.16	.54	.37	.15
Age[c]	.15	.53	-.15	.51	-.07	.78	.02	.92	-.04	.86	.11	.62
Education[d]	.39	.15	-.01	.97	-.08	.79	.41	.13	.12	.66	.35	.20
CSI[e]	.79	<.0001	.50	.02	.42	.06	.71	<.0001	.48	.03	.59	.005

Note. [a]Hospital = Total days in hospital through 20-months (n = 21); [b]Verbal IQ as measured by WAIS (Weschler, 1955; n = 17); [c]Age (n = 21); [d]Education (n = 15); [e]CSI = Clinical Status Interview assessing global health-sickness and psychiatric symptomatology (n = 21); [f]COM = Complexity of Representations; [g]AFF = Affect Tone of Representations; [h]EIR = Emotional Investment in Relationships; [i]USC = Understanding of Social Causality; [j]SE = Self-Esteem; [k]ICS = Identity and Coherence of the Self; [l]Pearson *r* correlational coefficients are considered to represent a small effect from .1 to .3, a medium effect from .3 to .5, and a large effect if greater than .5 (Cohen, 1988).

Table 6. Means, Standard Deviations, and Paired-Samples T Tests for Psychotherapy Group

	Psychotherapy (n = 9)							
	Pre-Treatment		Post-Treatment					
	M	*SD*	*M*	*SD*	*df*	*t*	*p*	*ES d*[gh]
COM[a]	1.64	.36	3.20	1.06	8	4.46	.002	3.15
AFF[b]	2.82	.54	3.59	.63	8	3.89	.005	2.75
EIR[c]	2.21	.36	2.79	.78	8	2.39	.04	2.39
USC[d]	1.69	.36	3.16	.92	8	6.53	.0001	4.62
SE [e]	2.76	.47	3.72	.53	8	4.53	.002	3.20
ICS[f]	2.86	.57	4.11	.55	8	5.01	.001	3.54

Note. [a]COM = Complexity of Representations; [b]AFF = Affect Tone of Representations; [c]EIR = Emotional Investment in Relationships; [d]USC = Understanding of Social Causality; [e]SE = Self-Esteem; [f]ICS = Identity and Coherence of the Self; [g] *ES* = effect size Cohen's (1977) *d*; [h]Cohen's *d* effect sizes are considered to represent a small effect from .2 to .5, a medium effect from .5 to .8, and a large effect if greater than .8.

Table 7. Means, Standard Deviations, and Paired-Samples T Tests for Medication Group

	Medication (n = 12)							
	Pre-Treatment		Post-Treatment					
	M	*SD*	*M*	*SD*	*df*	*t*	*p*	*ES d*[gh]
COM[a]	2.02	.64	2.65	.79	11	2.67	.02	1.61
AFF[b]	2.85	.72	3.08	.73	11	1.25	.24	0.75
EIR[c]	1.98	.57	2.52	.68	11	3.29	.007	1.98
USC[d]	2.00	.68	2.53	.85	11	2.04	.07	1.23
SE [e]	3.07	.64	3.27	.55	11	0.91	.38	0.55
ICS[f]	3.03	.98	3.58	.70	11	1.74	.11	1.05

Note. [a]COM = Complexity of Representations; [b]AFF = Affect Tone of Representations; [c]EIR = Emotional Investment in Relationships; [d]USC = Understanding of Social Causality; [e]SE = Self-Esteem; [f]ICS = Identity and Coherence of the Self; [g] *ES* = effect size Cohen's (1977) *d*; [h]Cohen's *d* effect sizes are considered to represent a small effect from .2 to .5, a medium effect from .5 to .8, and a large effect if greater than .8.

Table 8. Traditional Analyses of Between-Groups Change Using 'Difference Scores'

	Psychotherapy (n = 9)		Medication (n = 12)					
	M	*SD*	*M*	*SD*	*df*	*t*	*p*	*ES d*[gh]
COM[a]	1.56	1.05	0.63	0.82	19	2.27	.04	1.05
AFF[b]	0.77	0.59	0.23	0.65	19	1.94	.07	0.90
EIR[c]	0.58	0.73	0.53	0.56	19	0.18	.86	0.08
USC[d]	1.47	0.68	0.53	0.90	19	2.61	.02	1.21
SE[e]	0.97	.64	0.20	0.77	19	2.40	.03	1.13
ICS[f]	1.25	0.75	0.55	1.09	19	1.65	.12	0.77

Note. [a]COM = Complexity of Representations; [b]AFF = Affect Tone of Representations; [c]EIR = Emotional Investment in Relationships; [d]USC = Understanding of Social Causality; [e]SE = Self-Esteem; [f]ICS = Identity and Coherence of the Self; [g] *ES* = effect size Cohen's (1977) *d*; [h]Cohen's *d* effect sizes are considered to represent a small effect from .2 to .5, a medium effect from .5 to .8, and a large effect if greater than .8.

Table 9. Number of Individual Psychotherapy and Medication Patients Meeting Clinical Significance Criteria for 'Recovered', 'Positive Response', and Peters et al. (2006) for SCORS Complexity of Representations (COM)

	Psychotherapy (n = 9)		Medication (n = 12)	
Criterion	Improved	Deteriorated	Improved	Deteriorated
RCI > 1.96	7	0	5	0
SD > 2.0	5	0	6	0
'Recovered'[a]	5 (56%)	0 (0%)	5 (42%)	0 (0%)
RCI > 1.28	7	0	6	1
SD > 1.0	7	0	7	0
'Positive'[b]	7 (78%)	0 (0%)	5 (42%)	0 (0%)
RCI > 1.96 and Peters et al. (2006)	2 (22%)	n/a	0 (0%)	n/a

Note. [a]'Recovered': RCI > 1.96, *SD* > 2.0; [b]'Positive Response': RCI > 1.28, *SD* > 1.0.

Table 10. Number of Individual Psychotherapy and Medication Patients Meeting Clinical Significance Criteria for 'Recovered', 'Positive Response', and Peters et al. (2006) for SCORS Affect Tone of Representations (AFF)

	Psychotherapy (n = 9)		Medication (n = 12)	
Criterion	Improved	Deteriorated	Improved	Deteriorated
RCI > 1.96	3	0	1	0
SD > 2.0	1	0	1	0
'Recovered'[a]	1 (11%)	0 (0%)	0 (0%)	0 (0%)
RCI > 1.28	5	0	1	1
SD > 1.0	4	0	2	1
'Positive'[b]	3 (33%)	0 (0%)	1 (8%)	1 (8%)
RCI > 1.96 and Peters et al. (2006)	3 (33%)	n/a	0 (0%)	n/a

Note. [a]'Recovered': RCI > 1.96, *SD* > 2.0; [b]'Positive Response': RCI > 1.28, *SD* > 1.0.

Table 11. Number of Individual Psychotherapy and Medication Patients Meeting Clinical Significance Criteria for 'Recovered', 'Positive Response', and Peters et al. (2006) for SCORS Emotional Investment in Relationships (EIR)

	Psychotherapy (n = 9)		Medication (n = 12)	
Criterion	Improved	Deteriorated	Improved	Deteriorated
RCI > 1.96	4	0	4	0
SD > 2.0	3	0	2	0
'Recovered'[a]	3 (33%)	0 (0%)	2 (17%)	0 (0%)
RCI > 1.28	6	1	6	1
SD > 1.0	5	0	6	1
'Positive'[b]	5 (56%)	0 (0%)	5 (42%)	1 (8%)
RCI > 1.96 and Peters et al. (2006)	1 (11%)	n/a	1 (8%)	n/a

Note. [a]'Recovered': RCI > 1.96, *SD* > 2.0; [b]'Positive Response': RCI > 1.28, *SD* > 1.0.

Table 12. Number of Individual Psychotherapy and Medication Patients Meeting Clinical Significance Criteria for 'Recovered', 'Positive Response', and Peters et al. (2006) for SCORS Understanding of Social Causality (USC)

	Psychotherapy (n = 9)		Medication (n = 12)	
Criterion	Improved	Deteriorated	Improved	Deteriorated
RCI > 1.96	7	0	4	0
SD > 2.0	4	0	4	0
'Recovered'[a]	4 (44%)	0 (0%)	3 (25%)	0 (0%)
RCI > 1.28	8	0	6	0
SD > 1.0	7	0	6	0
'Positive'[b]	7 (78%)	0 (0%)	6 (50%)	0 (0%)
RCI > 1.96 and Peters et al. (2006)	2 (22%)	n/a	0 (0%)	n/a

Note. [a]'Recovered': RCI > 1.96, *SD* > 2.0; [b]'Positive Response': RCI > 1.28, *SD* > 1.0.

Table 13. Number of Individual Psychotherapy and Medication Patients Meeting Clinical Significance Criteria for 'Recovered', 'Positive Response', and Peters et al. (2006) for SCORS Self-Esteem (SE)

	Psychotherapy (n = 9)		Medication (n = 12)	
Criterion	Improved	Deteriorated	Improved	Deteriorated
RCI > 1.96	6	0	2	1
SD > 2.0	2	0	0	0
'Recovered'[a]	2 (22%)	0 (0%)	0 (0%)	0 (0%)
RCI > 1.28	7	0	4	1
SD > 1.0	4	0	5	1
'Positive'[b]	4 (44%)	0 (0%)	3 (25%)	1 (8%)
RCI > 1.96 and Peters et al. (2006)	5 (56%)	n/a	0 (0%)	n/a

Note. [a]'Recovered': RCI > 1.96, *SD* > 2.0; [b]'Positive Response': RCI > 1.28, *SD* > 1.0.

Table 14. Number of Individual Psychotherapy and Medication Patients Meeting Clinical Significance Criteria for 'Recovered', 'Positive Response', and Peters et al. (2006) for SCORS Identity and Coherence of the Self (ICS)

	Psychotherapy (n = 9)		Medication (n = 12)	
Criterion	Improved	Deteriorated	Improved	Deteriorated
RCI > 1.96	6	0	3	1
SD > 2.0	1	0	0	0
'Recovered'[a]	1 (11%)	0 (0%)	0 (0%)	0 (0%)
RCI > 1.28	7	0	6	1
SD > 1.0	6	0	6	1
'Positive'[b]	6 (67%)	0 (0%)	4 (33%)	1 (8%)
RCI > 1.96 and Peters et al. (2006)	6 (67%)	n/a	3 (25%)	n/a

Note. [a]'Recovered': RCI > 1.96, *SD* > 2.0; [b]'Positive Response': RCI > 1.28, *SD* > 1.0.

Table 15. Number of Individual Psychotherapy and Medication Patients Meeting Clinical Significance Criteria for 'Recovered' and 'Positive Response' for SCORS-C

	Psychotherapy (n = 9)		Medication (n = 12)	
Criterion	Improved	Deteriorated	Improved	Deteriorated
RCI > 1.96	6	0	4	0
SD > 2.0	4	0	3	0
'Recovered'[a]	4 (44%)	0 (0%)	1 (8%)	0 (0%)
RCI > 1.28	7	0	5	1
SD > 1.0	7	0	5	1
'Positive'[b]	7 (78%)	0 (0%)	4 (33%)	1 (8%)

Note. [a]'Recovered': RCI > 1.96, *SD* > 2.0; [b]'Positive Response': RCI > 1.28, *SD* > 1.0.

Table 16. Differences in RCI between Psychotherapy and Medication Treatments

	Psychotherapy (n = 9)		Medication (n = 12)					
	M	*SD*	*M*	*SD*	*df*	*t*	*p*	*ES d*[gh]
COM[a]	3.50	2.44	1.59	1.78	19	2.08	.05	0.92
AFF[b]	1.59	1.14	0.55	1.20	19	2.01	.06	0.89
EIR[c]	1.78	2.10	1.47	1.56	19	0.38	.71	0.18
USC[d]	3.47	1.79	1.38	2.05	19	2.44	.03	1.08
SE[e]	2.30	1.45	0.59	1.68	19	2.45	.02	1.08
ICS[f]	2.21	1.20	1.02	1.67	19	1.81	.09	0.80

Note. [a]COM = Complexity of Representations; [b]AFF = Affect Tone of Representations; [c]EIR = Emotional Investment in Relationships; [d]USC = Understanding of Social Causality; [e]SE = Self-Esteem; [f]ICS = Identity and Coherence of the Self; [g] *ES* = effect size Cohen's (1977) *d*; [h]Cohen's *d* effect sizes are considered to represent a small effect from .2 to .5, a medium effect from .5 to .8, and a large effect if greater than .8.

VITA

Eric Jason Peters was born on June 11, 1974 in Valley Stream, NY. He is the second of four children to Lyle and Beth Peters. At the age of six he and his family moved to Oyster Bay, Long Island where he and his siblings Alyssa (34), Adam (31), and Wendi (26) completed high school. Eric matriculated at Emory University in Atlanta for his first year of college before transferring to the University of Massachusetts in Amherst where he received his B.A. in Cultural Anthropology in 1996 and played three years on the varsity tennis team. After living and working in Western Massachusetts for nearly three years, Eric moved to Cambridge, Massachusetts to attend Hebrew College where he worked toward a M.A. in Jewish Literature and Philosophy. As part of his Master's work, Eric lived in Jerusalem for one year. During his stay in Israel Eric became interested in the variable responses people had to the violence and trauma associated with the second Palestinian Intifada that was raging the year he spent in Jerusalem. It was at this point he decided to pursue a career in clinical psychology.

In 2003, Eric began his doctoral studies in clinical psychology at the University of Tennessee, Knoxville. He completed research in the areas of therapeutic assessment, borderline character, projective testing, object relations, and the psychotherapy of schizophrenia. As part of his training, he worked as a psychodynamic therapist and assessor for low-income populations through the university clinic and a local community mental health center. Eric has been recognized and honored in a variety of empirical and clinical areas including: two-time recipient of Clinical Honors for Outstanding Clinical Work at the University of Tennessee, the Paul E. Lerner Award for Excellence in

Psychological Assessment, the Chris Hebb Memorial Award for Most Outstanding Contribution to Object Relations Theory, the University of Tennessee Science Alliance Award for Excellent Research Contributions in Clinical Psychology, and recipient of the University of Tennessee Scholarly Activities Research Incentive Fund.

Eric successfully defended his dissertation in May of 2007 and will be completing his pre-doctoral internship at the Bronx Psychiatric Center in New York City.