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Tessa Leona Hutchinson
The Role of Psychotherapy in
the Age of Medication: A
Theoretical Analysis of
Biological and
Psychodynamic Perspectives
on the Etiology and
Treatment of Schizophrenia

ABSTRACT

This theoretical study explores the role of psychodynamic psychotherapy in the treatment of schizophrenia in an era where biologically oriented psychopharmacological interventions are the dominant treatment model. The study was undertaken as an effort towards clarification, as a graduate student from a psychodynamic training program, of how my training might possess or lack efficacy or application in my future work with those living with schizophrenia.

Psychodynamic and biological perspectives on schizophrenia etiology and treatment have, throughout history, remained predominantly in opposition to one another. Currently neither perspective is able to fully explain the origins of schizophrenia, nor is the biologically-based treatment system adequately supporting the recovery of a large percentage of those with this diagnosis. A review of the literature from biological and psychodynamic perspectives shows that the rationale for the current unfavorable position of psychodynamic psychotherapy as a supplemental treatment for schizophrenia comes primarily from a lack of research, confusion about technique, and fear surrounding psychogenic theories of schizophrenia etiology.

Although far more research into psychodynamic psychotherapy with schizophrenia needs to occur, these interventions are difficult to study and do not provide

the evidence-based criteria that is essential within the current managed care mental health model. However, the success of alternative treatment programs which utilize dynamic interventions, as well as continued evidence of the role of stress and trauma in exacerbating genetic vulnerabilities to schizophrenia, suggest that there may be an increased role for psychodynamic perspectives and interventions in the future of schizophrenia treatment.

THE ROLE OF PSYCHOTHERAPY IN THE AGE OF MEDICATION:
A THEORETICAL ANALYSIS OF BIOLOGICAL AND PSYCHODYNAMIC
PERSPECTIVES ON THE ETIOLOGY AND TREATMENT OF SCHIZOPHRENIA

A project based upon an independent investigation,
submitted in partial fulfillment of the requirements
for the degree of Master of Social Work.

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2008

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CHAPTER I

INTRODUCTION

There are over 2 million people living with schizophrenia in the United States, and each of these people has a unique story of their struggle with the illness. While the narratives and individual experiences of schizophrenia are myriad in variation, there is one story that for me set in motion my current interest in schizophrenia and schizophrenia treatment. Larry was one of my first clients during my first year of clinical internship at Smith College School for Social Work. I was placed on the psychiatric assessment team at a small New England hospital, and I heard Larry singing before I ever saw him. He was singing Elvis's "Devil in Disguise" at the top of his lungs, and as he came through the double doors, I saw he was clutching a food processor in his arms.

As we sat down to talk, Larry began telling me about his wife. She had died at the age of 27, only three years after they were married. As he spoke of missing her, he became agitated and began to sing again, this time singing "Hound Dog" and pacing the room, staring at me as if the lyrics had been written for me. When he slowed to a halt and was able to sit again he began to tell me how he had seen the inside of more than 80 psychiatric hospitals around the United States. From here, he told me about his love for piano, and how he would stay up all night to play concertos until the neighbors pounded on the ceiling. His mood quickly soured again though as he told how he hated life because he felt like a rubber ball, a rubber ball without his medications (breaking into song again) bouncing from wall to wall.

Larry is a 57 –year-old Caucasian man, diagnosed with paranoid schizophrenia for more than thirty years. He had recently gone off all of his medications, because they made him feel “numb” but soon after arrived at the hospital of his own accord, plagued by hallucinations and delusions. Larry had been in state hospitals, crisis units, residential programs, community programs, and outpatient treatment on and off for the past thirty years. Larry had no therapist, did not have a relationship with his case manager, and his outpatient treatment consisted of seeing a doctor a few times a year for medications. Larry currently lived alone, with disability income, and was rarely seen in the community unless he stopped taking his medications.

I introduce this study with a glimpse into Larry’s life, because it was through my work with him, as he came in and out of the hospital throughout that first year of my clinical internship, that my curiosity and confusion about the state of schizophrenia treatment in this country began. First, I often times felt at a loss about how to approach my work, albeit brief, with Larry. While I knew supportive and cognitive behavioral therapy were the only recommended therapeutic interventions with schizophrenia, I was very unsure of where the line lay between supportive and psychodynamic therapies and was also unclear about why this line existed. Although I understood that the experience of psychosis is a horrific and overwhelming experience requiring support, at times there was such a depth to our conversations, to our transference relationship, and to his insight (despite being labeled as chronically and severely mentally ill) that I felt there might have been a possibility for more psychodynamically oriented work with Larry, or at least utility in a more psychodynamic perspective in his treatment.

At the same time, it became apparent to me over that year that Larry wasn't getting better. While Larry's medications relieved a lot of his symptoms if he stayed on them, he had never achieved stability or full recovery. Why wasn't Larry getting better? Why weren't his medications providing for his recovery, why wasn't he in therapy as well, and how was I supposed to work with him coming from the background of a primarily psychodynamic training program? While I experienced such a richness, complexity, and meaning in my work with Larry, I felt like the tools I had for working with him had no place or accepted role in the current schizophrenia treatment model - and yet I didn't understand, why not? Would more insight-oriented work be harmful to Larry? Was there something I didn't perceive or grasp that made him incapable of engaging in this form of treatment? Was the current treatment system really providing him with as much recovery as he could ever hope for?

It was around these treatment questions that the focus for this current study arose. I realized, as I began to look for answers as to why Larry's treatment was occurring the way it was, and why psychodynamic therapy with schizophrenia is no longer recommended, that there is very little information available in the literature around these questions. While the current treatment system for schizophrenia is based on a biological model which explains schizophrenia as a biological disease and emphasizes psychopharmacological and supportive psychosocial interventions, (Lehman, Steinwachs et al. 1998; Whitaker, 2002; Whitaker, 2005; Valenstein, 1998; Austrian, 2000; Kraly, 2006; American Psychiatric Association (APA) (2004), psychodynamic treatment has at times in the past half century been the recommended treatment for schizophrenia (Arieti, 1980, 1955; Hornstein, 2000; Searles, 1965; Bullard, 1959; Fromm-Reichmann, 1950;

Karon & Vandenbos, 1981). The interplay between these etiological and treatment perspectives over the years has grown into a contentious debate, which at present exists as a sharp dichotomy, with a dearth of literature providing any sort of exploration of this dichotomy or movement towards a possible synthesis between these theories (Gottdiener & Haslam, 2003; Ver Eecke, 2003; Silver, 2003; Whitaker, 2002; Luhrmann, 2000). This dichotomy and the confusion surrounding it, due to limited research, continues to promote an atmosphere in which managed care companies are allowed to dictate treatment according to cost reduction rather than long-term benefit or quality of life (Luhrmann, 2000).

It is this void in the research and literature, specifically as it relates to those from psychodynamic training programs entering the clinical field in the treatment of major mental illnesses, that this study attempts to fill. To this end, this thesis is focused on answering the question: What is the role of psychodynamic psychotherapy in the treatment of schizophrenia in an era where biologically oriented psychopharmacological interventions are the dominant treatment model?

The next chapter, Chapter II, provides further introduction to the conceptualization and methodology of this study, and defines the key terms including schizophrenia, and “biological” and “psychodynamic” perspectives. Chapter III provides an introduction to the epidemiology of schizophrenia, the history of schizophrenia treatment, and a brief overview of the current treatment system in the United States today. Chapters IV and V provide an in-depth review of the literature on how schizophrenia is conceptualized and treated from psychodynamic and biological perspectives. Chapter VI summarizes the study, discussing and analyzing the findings,

seeking areas of synthesis and possible integration, providing suggestions for clinicians, and exploring directions for future research on this topic.

CHAPTER II

METHODOLOGY

Within this chapter I provide a conceptual framework for the theoretical study to follow. I provide a definition of terms as I have used them in this study, and conclude by identifying potential methodological biases and considering both the strengths and limitations of the study.

Conceptualization

The purpose of this theoretical study, as discussed in the introduction, is to answer the question: What is the role of psychodynamic psychotherapy in the treatment of schizophrenia in an era where biologically oriented psychopharmacological interventions are the dominant treatment model? In an effort to answer this question, I will review the relevant literature from both biological and psychodynamic perspectives.

This study is conceptualized as an exploration, based on the premise that a great amount of confusion remains for me and for many psychodynamically trained clinicians, and graduate students alike, about how, if, and in what ways to integrate psychodynamic theory or techniques into our work with people with schizophrenia.

Definition of Terms

Schizophrenia

Schizophrenia is defined by The American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition, text revision (DSM-IV TR) along a set of six criteria. These include criteria for the characteristic symptoms of delusions, hallucinations, disorganized speech and behavior, as well as social and occupational dysfunction and duration of symptoms. While for many, (Boyle, 2002; Bentall, 2003, 1990; Kirk & Kutchins, 1992; Conrad & Schneider, 1980; De Waelhens & Ver Eecke, 2001), this diagnosis and description of schizophrenia lacks greatly in objectivity, accuracy, validity, and reliability, it remains the most commonly used and widely agreed upon definition available today.

My subject matter focuses specifically on schizophrenia and its subtypes (paranoid, disorganized, catatonic, undifferentiated, and residual), and therefore when I refer to psychosis, I am referring to the psychosis experienced within this disorder. When referring to schizoaffective disorder, schizophreniform disorder or other diagnoses within the schizophrenia family I will specify these individual diagnoses. While it is necessary, for the purposes of this study, to limit my exploration of the applications of psychodynamic psychotherapy to the treatment of schizophrenia, many of the questions addressed may also be very applicable to the treatment of the other psychotic illnesses as well.

While I try to refer to schizophrenia as schizophrenia the majority of the time, I also at times utilize the terms illness or disease. This does not imply an allegiance with any theory of schizophrenia etiology, and is not meant in a stigmatizing manner. While I

would prefer to not use terms that inherently imply brokenness or difference while speaking of mental health issues, there are no terms that to my knowledge that would not do that to some extent.

People with Schizophrenia

Within this study I use the words client, patient, consumer, and person with schizophrenia to refer to those with schizophrenia. I do not use the term schizophrenic, as I believe this term is harmful and stigmatizing, and identifies a person as their illness rather than as a person with an illness. While I do feel the terms patient, client, and consumer are all problematic as well, implying power differentials, they are less cumbersome than always utilizing person living with schizophrenia to describe those with this diagnosis.

Psychodynamic Psychotherapy

The psychodynamic perspective or psychodynamic psychotherapy are the terms I use to refer to the many theories (i.e. interpersonal, ego psychology, self psychology, object relations) that are concerned with “inner energies that motivate, dominate, and control people’s behavior... energies...based in past experiences and present reality” (Berzoff, Flanagan, & Hertz, 1996, pp. 11-12). I use the term psychodynamic psychotherapy rather than psychoanalytic psychotherapy as the former includes the possibility of incorporating psychoanalytically based interventions while considering the broader dynamics of the social and cultural world (Berzoff, Flanagan, & Hertz, 1996).

When referring to psychodynamic psychotherapy I am referring to those therapies considered to be “ego modifying, “expressive,” “insight oriented” or “exploratory” not solely those known as ego supportive (Pinsker, 1997). While psychodynamic psychotherapy does utilize supportive techniques, it is necessary to define these two as separate here because supportive therapy is currently recommended for the treatment of schizophrenia while the efficacy of the more psychodynamic and ego-modifying techniques remain the question at the heart of this study. Therefore if I am referring to purely supportive psychotherapy I will do so specifically, otherwise it can be assumed that I am speaking of more psychodynamic interventions.

The Biological Perspective

The biological perspective is currently the most widely accepted understanding of etiology and treatment of schizophrenia. I have chosen the term biological perspective as the most descriptive and non-biased term to represent the orientation most commonly known as the medical model, biomedical, or psychiatric model. Biological perspective will be used as the umbrella term to describe genetic, neurochemical, and neurobiological research on schizophrenia as well as medically based interventions such as antipsychotic medication and cognitive and behavioral therapy methods.

Mind and Brain

Mind is the term I use to represent what some like Frattaroli (2001) define as the psyche or soul, where mind is defined as “the seat of consciousness, thought, and volition” (Oxford Dictionary, 1998). This is in contrast to the term brain, which is

defined as “an organ of soft nervous tissue contained in the skull of vertebrates, functioning as the coordinating center of sensation and of intellectual and nervous activity” (Oxford Dictionary, 1998).

I utilize the terms mind and brain, or the phrase theories of mind versus theories of brain, to discuss psychodynamic versus biological orientations on the etiology and treatment of schizophrenia. I use these terms because they have come to represent, throughout the literature (Andreasen, 2001, 1997; Miresco & Kirmayer, 2006; Ghaemi, 2003; Luhrmann, 2000, Gabbard, 2005), the dichotomy, albeit false to many in a post-Cartesian era, between these two central theories (Gabbard, 2000). These terms historically came to represent this division because the psychodynamic perspective located the source of etiology and healing for mental illness in making meaning from the symptoms of a person through understanding workings of a humans conscious and subconscious built from environmental and social history (Berzoff, Flanagan, Hertz, 1996), while the biological perspective located the etiology and treatment of mental illness in the balance of chemicals within the brain. (Andreasen, 2001; Luhrmann, 2000).

Methodology

This theoretical thesis contains six chapters. In the first chapter, the introduction, I provide an opening explanation of the research topic, the reasons for the research, and the biological and psychodynamic theories I have chosen to guide to my literature review. In this, the second chapter, I have described how the study is conceptualized and the methodology to be used as well as introduced and defined some of the central terminology.

The third chapter contains a brief history and introduction to the phenomenon being studied, namely the role of psychodynamic psychotherapy in the age of predominantly biomedical interventions. Because the point of this research is essentially to clarify this phenomenon, this chapter does not provide such clarification. However, in an effort to provide a more multi-dimensional picture to begin the clarification process, I have divided the chapter into three sections. First, I address the symptoms, prevalence, and diagnosis of schizophrenia, the population diagnosed, and the course of the illness and recovery. Secondly I provide an overview of how schizophrenia has been understood and treated historically. And finally, I provide a brief overview of the treatment system today.

In the fourth chapter I review the literature from the biological perspectives on schizophrenia, and in the fifth chapter I review the literature pertaining to psychodynamic treatment and understandings of schizophrenia. I will address the questions: how are the causes of schizophrenia understood from each of these perspectives, what interventions are suggested today, and what are the outcomes of treatment within these two models?

The literature reviews for these two chapters were conducted through reading early writing from central figures in both perspectives as well as through reviews of articles in academic search databases including primarily Social Work Abstracts. I made the decision to work primarily from the social work abstracts as I am not only a clinical social work student, but this study's intended audience is clinical social workers, psychologists, and students.

Finally, the sixth chapter of this study provides discussion and summary of these two theories, illuminating ways in which the dichotomy between the approaches is in

some sense false and may actually serve to undermine the best possible approach to treating and understanding schizophrenia. I conclude this chapter with recommendations for greater synthesis between these theories in clinical practice and recommend areas for further research.

Study Biases and Limitations

The central limitation of this study is that, due to the time-limits of the project, the focus of my research had to be on only two dimensions of the complex system of schizophrenia treatment. As a social work student training in a psychodynamic perspective, I am most interested in understanding to a greater degree what my role as a psychotherapist will be in a treatment system dominated by psychopharmacological interventions and understandings. Therefore, within this study, I will not be addressing the literature on community programs, peer-led services, occupational training, or the various other interventions that are used beyond or in conjunction with biological and psychodynamic interventions, some to great success, in the treatment of schizophrenia.

Another central limitation of this study is that it does not consider consumer perspectives to the degree that, in the contexts of a larger study, I would consider appropriate. In some ways however, I would like to believe that this research is inspired by my reading of consumer perspectives, and my relationships with people with schizophrenia. I embarked on this study because of the desire to improve the treatment system for those with schizophrenia; I believe that the dichotomy that exists between psychodynamic and biological perspectives actually serves to only perpetuate and reinforce stigma. If we understand schizophrenia solely as a biological entity, we are

ignoring the complex and unique human being beneath this diagnosis working towards recovery. If we work solely from a psychodynamic perspective, people with schizophrenia might be seen by some as to blame for their illness and would not be provided with the amazingly powerful medications that have freed so many from the agony of neurochemically based hallucinations and delusions.

I was also aware, embarking on this study, that to many consumers the history of mental health treatment both psychodynamic and biological is, in truth, a genealogy of power (Foucault, 1965; 1977), and violence (Sedgwick, 1982), and that to some, the academic exercise of debating treatment styles as undertaken here is merely a perpetuation of this painful history.

The biases in this study are, most significantly, my own experience working within mental health, and navigating the mental health system with a family member recovering from a psychotic illness. Both of these experiences no doubt influence the initial motivation, assumptions, and direction of this study. From both of these perspectives I have experienced the current public mental health system as a huge and dehumanizing catastrophe, yet I have also witnessed the profound healing offered by both psychopharmacology and therapeutic relationships. I should also acknowledge my own relative inexperience as a clinician and my unceasing optimism for the potential for human resilience and recovery - especially in the relational process of individual therapy.

Conclusion

Within this chapter I have provided further explication of this theoretical study. This has included addressing how the study is conceptualized, a definition of key terms, a

structural framework for the chapters to follow, and a discussion of methodological biases and study limitations. In the following chapter, Chapter III, I address the phenomenon of schizophrenia.

CHAPTER III

SCHIZOPHRENIA AND SCHIZOPHRENIA TREATMENT

Introduction

This chapter provides an introduction to the phenomenon being studied in this theoretical research project. As explained in the previous chapters, this study is undertaken in an effort to clarify the current role of psychodynamic psychotherapy in the treatment of schizophrenia. The purpose of this chapter is to give the reader a greater understanding of schizophrenia, the controversial history of how it has been understood and treated, as well as how the treatment system looks today.

This chapter is divided into three parts. First I address the scope of schizophrenia, the population diagnosed, and the course of illness and recovery. Secondly I provide a historical background on the diagnosis and treatment of schizophrenia focusing on the development of the debate, which continues today, between psychodynamic and biological perspectives. While the following chapters, Chapters IV and V, provide literature reviews of current research and treatment suggestions from biological and psychotherapeutic perspectives, this section of this chapter aims to provide a history of how the current treatment system evolved. Finally I present a brief illustration of the current treatment system for schizophrenia today.

Schizophrenia

Scope, Population, and Epidemiology

It is currently estimated from combined available data, that 2.2 million people in the United States are living with schizophrenia during any given year (Kemp, 2007). To put that number into perspective, 2.2 million people is the same number of people as constitute the populations of Rhode Island, Alaska, and North Dakota combined (Torrey, 2001). This figure means, according to Shean (2004), that approximately 7 to 8 people per every 1,000 have schizophrenia, which again means that in every town of 3,000, 21 to 24 people are diagnosed with schizophrenia, and a city of 3 million would have approximately 21,000 to 24,000 people with schizophrenia. Worldwide it is thought that up to 51 million people are suffering with schizophrenia (Torrey, 2001).

Schizophrenia symptoms commonly begin around late adolescence and early adulthood, and the illness is thought to affect men and women equally although the mean age of onset is slightly higher (5 years) for women (Kemp, 2007). There is some geographic variation in the prevalence of schizophrenia as the United States, Ireland, and Finland report prevalence of 8 cases per 1,000 people while countries like Ghana, Botswana, and Taiwan report prevalence rates closer to 2 people per 1,000 and one area in Northern Sweden reports rates as high as 17 per 1,000 people (Torrey, 2001). There is also a strongly proven urban risk factor for schizophrenia in the United States. People in low socioeconomic strata in urban areas have been shown to be at twice the risk for the development of schizophrenia than people born and raised in rural areas (Shean, 2004; Harrison, Gunnell, & Glazebrook, 2001). There are various theories proposed to explain

this variation including the possibility that schizophrenia causes a decline in socioeconomic status, or that people with schizophrenia migrate to cities in search of services, or that people in urban areas are exposed to more stress in the form of hostility and violence than people rurally (Berzoff, Flanagan, & Hertz, 1996).

There have also been studies done comparing prevalence of schizophrenia between ethnic and racial groups (Berzoff, Flanagan, & Hertz, 1996; Torrey, 2001; Whitaker, 2002). While Mexican Americans, Hutterites and the Amish are shown to have a much lower risk for schizophrenia, some studies have suggested that African Americans are at one and a half times greater risk for schizophrenia than Caucasian Americans (Torrey, 2001). These studies appear to have little validity however, for once the numbers are corrected for the large number of African Americans living in urban areas, there is no merit for claims of difference due to race (Shean, 2004). There is also strong evidence that a long history of racism and bias in mental health, perpetuated by psychiatrists who were predominantly white, has actually led to the over-diagnosis of schizophrenia among people of color (Berzoff, Flanagan, & Hertz, 1996; Torrey, 2001; Whitaker, 2002; Loring, 1988).

Course and Recovery

Initially Emil Kraepelin, one of the leading psychiatrists of the 19th century, thought that 75 percent of people with schizophrenia could be expected to deteriorate into a state resembling that of the end-stage of dementia (Bentall, 2003; Whitaker, 2002, Kraepelin & Roberson, 1919). For many years this theory was believed valid as schizophrenia was viewed as a degenerative illness, one that, as suggested by Emil

Kraepelin could be defined by having a uniformly poor outcome (Desisto, Harding, McCormick, Ashikaga, & Brooks, 1995). While it has been argued by the historian Mary Boyle (2002), that a large majority of the population with whom Kraepelin conducted his research may have been actually suffering from organic degenerative brain diseases and not schizophrenia (Whitaker, 2002), even today, a diagnosis of schizophrenia is often compared to a diagnosis of cancer, meaning that too often it is considered a “sentence as well as a diagnosis” (Torrey, 2001, p. 1; Berzoff, Flanagan, Hertz, 1996).

Recent research of prognostic outcomes conducted within the United States point to a much higher degree of variation in the course of the schizophrenia than originally thought (DeSisto, et al., 1995). A study by Courtenay Harding (2003) compared prognostic outcomes for schizophrenia in 10 long-term research studies of schizophrenia with the historical prognosis for the illness initially given by Bleuler and Kraepelin. Harding found that within the 10 studies reviewed in the article there was actually a wide heterogeneity of outcomes. “Each finds that approximately one-half to two-thirds of people with schizophrenia can achieve a state of significant improvement or even recovery” (Harding, 2003, p. 2).

Harding’s study findings are similar to research by Stephens (as cited in Torrey, 2001) which states that after 10 years, 25 percent of individuals with schizophrenia recover completely while 25 percent are much improved, 25 percent are improved, but require extensive support network, 15 percent are hospitalized and unimproved, and 10 percent are dead mostly due to suicide. After 30 years, the research shows these numbers shift slightly as 25 percent still recover completely while 35 percent are much improved and relatively independent, 15 percent are improved, but require an extensive support

network, 10 percent are hospitalized, unimproved, 15 percent are deceased, again mostly from suicide.

While it is beyond the scope of this study to debate the causes for discrepancy amongst recovery rates in schizophrenia, it is also relevant to note that some studies also suggest a large difference amongst schizophrenia outcomes on an international scale. One such study, conducted by The World Health Organization over an eight year period starting in 1969, and followed-up by a repeat of the study in the early 1990's, showed that people who suffered first schizophrenic psychotic breaks in India, Nigeria, or Colombia, with all people in each country diagnosed using the same criteria, were far more likely to recover than people suffering a similar break in the United States (Jablensky, 1992). People in less developed countries had a two-thirds rate of recovery, while in direct reversal of these numbers, two-thirds of people in the United States, and other developed countries, became chronically mentally ill (Whitaker, 2002). The causes for these outcome variations remain a mystery, and are widely critiqued, but do suggest that there is still a large amount about the epidemiology of schizophrenia that remains to be understood.

History of Conceptualization and Treatment

Theories of Causality and Diagnosis

There is debate about when exactly schizophrenia or schizophrenia-like illnesses first appeared. In part this is due to the ways in which criteria, understandings, and definitions of the illness have continually changed throughout history (Bentall, 2003). Some say schizophrenia is an ancient illness, citing examples of figures from Sanskrit

and biblical texts (Torrey, 2001; Shean, 2004). Other scholars point to figures from the late middle ages such as Henry VI or Shakespeare's *Hamlet* (Shean, 2004) as the first known examples of the illness, while still others like Gottesman (as cited in Shean, 2004), argue that "no descriptions of a separate mental disorder that resembles modern definitions of schizophrenia are available prior to the nineteenth century" (p.5).

In late 1800's the diagnostic system for understanding schizophrenia was first created which evolved into the diagnostic system used today (Bentall, 2003).

Schizophrenia was initially discussed in the work of neuropsychiatrist Emil Kraepelin as a disease he identified as *dementia praecox* (Bentall, 2003). Kraepelin's classification grew from the idea of the psychoses being divided into two apparent types, those accompanied by emotional disturbances (manic-depression), and those accompanied by a lack of affect, beginning in adolescence, and marked by a degenerative course (thus *dementia praecox*) (Whitaker, 2002).

At the turn of the eighteenth century, as Freud's theories of psychoanalysis were just beginning to gain prominence, Eugene Bleuler, a Swiss psychiatrist, took over the classification work begun by Kraepelin. It was during this time period that the debate was born, which continues today, between biological and psychological theories of etiology (Shean, 2004).

Initially Bleuler and Freud were enthusiastic about the work of the other, with Bleuler especially influenced in this enthusiasm by his assistant Carl Jung, however they soon had a falling out (Shean, 2004). While Bleuler was willing to integrate Freudian ideas into his work, such as the idea that delusions and hallucinations served psychological functions, he was not to be swayed from his belief of the predominantly

biological origins of mental illness (Shean, 2004). Freud however steadfastly adhered to his theories of the unconscious, repression, and conflict as the roots of mental illness and would not consider biological causes (Bentall, 2003). By 1908, Bleuler officially changed the name of *dementia praecox* to schizophrenia (Whitaker, 2002).

In the early 1900s, Adolph Meyer, an anatomical pathologist by training, continued Bleuler's work of building upon the classification system created by Kraepelin, and pursuing a unity between biological and psychological explanations of causality of schizophrenia. For Meyer, originator of the idea of psychobiology, a precursor of the biopsychosocial model still utilized today, schizophrenia was "not a disease in the traditional sense" (Shean, 2004, p. 13), but rather a "loose grouping of behavioral and psychological reactions to the interaction of biological vulnerabilities and environmental stressors" (p. 13). The first Diagnostic and Statistical Manual (DSM-I) of the American Psychiatric Association (APA), published in 1952, reflected the influence of this more psychobiological explanation.

The influence of Meyer's psychobiological explanation of the causes and symptoms of schizophrenia remained present in the initial two publications of the DSM (Ghaemi, 2003). The DSM II described the symptoms of schizophrenia as "characterized chiefly by a slow and insidious reduction of external attachments in interest and by apathy and indifference leading to impoverishment of interpersonal relations, mental deterioration, and adjustment on a lower level of functioning" (Luhmann, 2000, p. 229).

By the 1950's onward, when neuroleptic medications, specifically chlorpromazine, were discovered to have calming effects on patients with schizophrenia, biological explanations of schizophrenia once more came to the forefront. This change

was spurred on by a Neo-Kraepelinian movement amongst psychiatrists, who were disenchanted with psychoanalysis, and resolved to keep psychiatry a branch of medicine (Bentall, 2003). These scientific or “remedicalized” psychiatrists “argued, in effect, that psychiatry had made a wrong turn by following Freud instead of Kraepelin” (Luhmann, 2000, p. 226). In part this renewed division is attributed to the fact that as demand for therapy increased in the mid 1900’s, psychologists and social workers also came to be recognized as legitimate therapists. This in part contributed to a movement by psychiatrists to return to a more scientifically based practice because they “no longer looked as if they were doing something special, something that no one else could do” (Luhmann, 2000, p. 225).

During the 1970’s, there was extensive debate back and forth between biological and psychodynamic perspectives. Eventually the biopsychosocial model, which incorporated both biological and psychological factors, was agreed upon as a compromise for how treatment should be structured (Ghaemi, 2003). Despite this compromise, the following edition of the DSM, the DSM III, showed a significant change in language and format to reflect a more scientific approach (Shean, 2004). As described by Luhmann, (2000), the DSM III marked the turning point in the study of mental illness in which,

“the patients personal history- his or her ambivalence, potty training, basic trust, resolution of the Oedipus complex, dependency, whatever- was irrelevant... what mattered was that he met a the necessary number of criteria. All of a sudden, there was a sharp clean dividing line between mental health and illness” (p. 228).

Biological perspectives on the etiology of schizophrenia remain the dominant model today as schizophrenia is widely regarded as a brain disease and scientists continue to pursue (as I review in Chapter IV) research into genetic, neurochemical, and

neurobiological explanations of its epidemiology (Shean, 2004; Torrey, 2001). In part the disease model may have achieved widespread acceptance also due to restrictions around insurance coverage.

“Faced with the fear that psychiatric care would not be reimbursed, many psychiatrists, psychiatric lobbies, and patient lobbies (the most effective probably being the National Alliance for the Mentally Ill) have argued that psychiatric illness is a medical disease like any other and deserves equal coverage, or ‘parity’” (Luhmann, 2000, p. 250).

The dominance of the biological perspective is reflected in the DSM IV-TR’s continued pursuit of scientifically accurate criteria and diagnosis (Ghaemi, 2003). There remains great controversy however as to the descriptiveness and accuracy of the current diagnosis of schizophrenia as found in the DSM-IV-TR. Some suggest that schizophrenia is actually a spectrum of disorders (Shean, 2004), while others suggest that a diagnosis should only serve as a “shorthand description of a patients complaints” (Bentall, 2003, p. 68). As discussed by Jablensky (as cited in Ghaemi, 2003), the DSM-IV does not

“represent classifications in the usual sense in which the term is applied in biology. Essentially they are augmented nomenclatures (i.e., lists of names for conditions or behaviors, supplied with explicit rules as to how these names should be assigned). As such they are useful tools of communication” (p. 172).

For now it seems, as suggested by Shean (2004), categories like schizophrenia as it appears in the DSM-IV should be “recognized as the provisional consensus agreements that they are” (p. 38) but that “reification of the DSM-IV diagnosis can foster the impression that we know what schizophrenia is, what causes it, and how best to treat it, when in fact we do not” (pg. 37).

History of Treatment

The history of schizophrenia treatment has always mirrored the history of the theories of schizophrenia etiology. As the theories of cause in schizophrenia reflect an ongoing debate between theories of mind and brain, so also does the history of treatment (Karon & Vandenbos, 1981; Whitaker, 2002; Luhrmann, 2000).

In ancient times treatment suggestions for psychosis ranged from cheerful chat, good counsel, and the admonitions of philosophers, to skull drilling, bleeding with leeches, shocks with electric eels, baths with vinegar, and even death – thought to be the only and final remedy to rid the body of demons. (Sedgwick, 1982; Karon & Vandenbos, 1981).

In the 1600's, one of the first treatment guides for “madness” was published, written by a physician named Thomas Willis. Willis suggested that the only cure for the mad, whom he suggested to be “animal-like in kind” (Whitaker, 2002, p. 6) was torture and intimidation. Similar practices were still carried out by doctors in the late 1700's, as it was believed that anything that physically weakened the mad, and thus calmed the mind, was helpful. Practices of this time period moved beyond physical violence alone to bleeding, near-drowning, and purging practices, seen to quiet hallucinations and “ranting” (p. 7).

The 1800's brought the dawn of “moral treatment” in America and Europe. Inspired by the work of Phillippe Pinel in France and the Quakers in England, this treatment system came about from Pinel's observations of the positive effects of better treatment of patients by staff at a men's asylum at which he worked. The Quakers in Philadelphia opened the first moral treatment asylum in the United States in 1817 and by

1841 there were 16 asylums in the United States offering moral treatment methods (Whitaker, 2002). Many hospitals were reported to have quite remarkable results with this treatment method. During a period between 1833 and 1837, hospitals like Worcester State Hospital in Massachusetts, Hartford Retreat in Connecticut, and McLean hospital in Boston, discharged between 60 and 70 percent of their clients as “recovered” (Whitaker, 2002, p. 27; Karon & Vandenbos, 1981).

While moral treatment arose in opposition to medical interventions of the time, physicians, especially in the United States, began lobbying by the mid 1800’s for increased medical interventions in asylums (Whitaker, 2002). In part, this was informed by the case histories kept by the moral treatment communities that provided extensive material for medical theories of disease (Karon & Vandenbos, 1981). At the same time, reforms brought about by Dorothea Dix led to the release of a huge number of the mentally ill from prisons into hospitals (Torrey, 2001). Not only did this require the opening of a large number of new hospitals, but also these recently released prisoners often did not have the means to pay for private treatment in the more expensive moral treatment facilities. As new asylums opened, laws were passed at the urging of physicians, which stated that physicians must supervise all new facilities. These new supervisory physicians initially integrated some moral treatment methods into the new facilities, stating that although insanity was a medical disease, moral treatment was a medical intervention as it served to “soothe the nerves” (Whitaker, 2002, p. 29).

At the same time that medical interventions were once more gaining prominence, some of the main thinkers in the field of psychoanalysis and psychology took more pessimistic views of schizophrenia treatment (Karon & Vandenbos, 1981). Freud

believed little work could be done with schizophrenia due the inability to form a transference relationship, and Jung, while reporting some therapeutic successes with schizophrenia, at times in his work suggested that he believed that there was solely an organic basis for schizophrenia (Karon & Vandenbos, 1981). There were, however, a select few people who built on the successes of the moral treatment movement to pursue theories of psychogenic etiology and attempt psychologically based interventions with schizophrenia. These people included D.W. Winnicott, Melanie Klein, Harry Stack Sullivan, Frieda Fromm-Reichmann, and Harold Searles.

During the 1930's, Sullivan, Fromm-Reichmann, and others continued to practice psychotherapy with people with schizophrenia (Karon & Vandenbos, 1981) and by the end of World War II psychodynamic interventions were the dominant model of treatment of schizophrenia and mental illness in general (Luhmann, 2000). In part this was due Meyer's work on classifying mental illness through psychobiology, which was "rather hostile, in practice, toward biological approaches to psychiatry" (Ghaemi, 2003, p. 6).

The central role of psychotherapy (specifically psychoanalytically oriented psychotherapy) during this time period was also due to the stress and anxiety that war and industrialization were causing. These factors served to create a broader public interest and need for mental health services (Luhmann, 2000). People wanted "help" (p. 212) and "psychoanalysis introduced a theory of mind that in its complexity and explanatory power was clearly superior to its predecessors and clearly better equipped to handle mental distress" (p. 212).

Throughout the 1930's and 1940's, in addition to the strong support for psychodynamic and psychoanalytic interventions, the search continued for biological

treatments of schizophrenia as well (Whitaker, 2002). The 1940's brought about the first trials of electro-convulsive therapy (ECT), and soon after lobotomy surgeries were first performed (Whitaker, 2002). In 1951, a surgeon named Henri Laborit first tested chlorpromazine on surgical patients, and soon after, chlorpromazine, later to be marketed as Thorazine, was first tried on people with psychosis. Over the next ten years, Thorazine came to be marketed as a "safe, antischizophrenic treatment" (Whitaker, 2002, p. 159).

During the 1950s and 1960s, multiple factors combined with the birth of neuroleptic medication to begin the movement towards deinstitutionalization. There were fiscal concerns within states about the burden of hospitalization as well as multiple exposes in the press about the horrible conditions within state hospitals. Initially during the period from the mid 1950's to mid 1960's the population within state hospital facilities decreased from 559,00 to 515,000 (Whitaker, 2002). However, during the course of the 1960's through 1970's, as federal subsidies disappeared for state hospitals and legislation passed for social security payments for the mentally ill, the hospital population began to decrease more drastically. By 1980, according to Whitaker (2002), the "census in the public mental hospitals in the United States had declined to 132,164" (p. 227).

While deinstitutionalization was a "humane and reasonable idea" (Torrey, 2001, p. 21), it was also a failure for a number of reasons. One reason is that although there was, as described, a huge shift of the mentally ill population from the hospitals back into the community, "personnel and fiscal resources did not follow them" (Torrey, 2001, p.

21). According to psychiatrist Alexander Gralnick (1986), in his article entitled “Future of the Chronic Schizophrenic Patient: Predictions and Recommendations”,

“community resources for even minimally good standards of treatment for such number of patients are grossly inadequate. It may be fairly said that today totally insufficient care is being rendered in the community to the vast number of chronic schizophrenics” (p. 8).

The central cause for the poor care provided to those with mental illness after deinstitutionalization, was the failure of the 3 billion dollar Community Mental Health Care (CMHC) program funded by the federal government (Torrey, 2001). The CMHC programs were intended to serve as support systems for people as they were discharged from the hospital. Along with the CMHC program came legislation to discourage hospitalization, like the Institutions for Mental Disease exclusions, which states that federal Medicaid would not reimburse states for patient hospitalization in state hospitals. As noted in Torrey (2001), due to the fact that the majority of people diagnosed with schizophrenia are on Medicaid, there is large fiscal incentive for states to deny hospitalization, and “no fiscal incentive...to provide aftercare” (pp. 23-24).

Throughout these changes, and an increase in medication trials, psychotherapy with schizophrenia continued. As described in Luhrmann (2000),

“the new psychiatric science did not in itself pose a life-threatening danger to psychodynamic psychiatry, because for all the foolishness of psychoanalysis in the era of its great arrogance, psychodynamic psychotherapy made a significant difference to the lives of patients and most psychiatrists knew it” (p. 237).

For this reason throughout the 1980's, while not to the same degree as before, psychiatrists and hospitals continued to utilize and promote both psychodynamic and biomedical models of treatment (Luhrmann, 2000).

While debate continued between those who felt psychotherapy had significant results treating schizophrenia and those who felt it did little good (Luhrmann, 2000; Karon & Vandenbos, 1981) it was ultimately “economic and social problems under which the old psychoanalytic paradigm gave way” (Luhrmann, 2000, p. 220). In the 1980’s and early 1990’s as states continued to look for ways to cut mental health costs, close hospitals, manage insurance care, and reintegrate clients in to the community, antipsychotic medication increasingly became the favored treatment (Whitaker, 2002).

Psychotherapy became literally impossible on an inpatient basis due to time constraints. Insurance companies continued to cut coverage for outpatient treatment due to the lack of empirical data for psychotherapy treatment (Luhrmann, 2000). This was because with mental health treatment as opposed to general medical treatment “there is less clarity, uniformity of terminology concerning mental health diagnosis, treatment modalities and types of facilities providing care... One dimension of this arises from the latent or private nature of many services” (Luhrmann, 2000, p. 225).

The decline of psychotherapy occurred for numerous reasons beyond insurance changes alone. These reasons included (as discussed) antipsychotic medications, the neo-Kraepelinian movement in psychiatry, deinstitutionalization, and the failure of community mental health (Luhrmann, 2000; Torrey, 2001). While the “history of ideological warfare [between biomedical and psychodynamic perspectives] invites us to infer from the use of biomedical treatment the failure of the psychodynamic approach” (Luhrmann, 2000, p. 262) the merits of psychotherapeutic interventions with schizophrenia continue to be debated in the current literature.

While the role of psychodynamic treatments remains unclear, it is psychopharmacology, insurance restrictions, evidence based treatment, and brief treatment which dominate the current mental health treatment system today (Luhrmann, 2000; Whitaker, 2002).

Current Treatment System

The current treatment system for schizophrenia is vast and complex. While this study does not focus on the delivery system of schizophrenia treatment, it is apparent from the history of the etiology and treatment that the way in which services are delivered has always been inherently connected to how schizophrenia is conceptualized. Currently, possibilities for private treatment include everything from intensive psychoanalytically oriented treatment at The Austen Riggs hospital in Western Massachusetts, to Buddhist based community treatment in Colorado, to intensive family based interventions in Europe. For the majority of consumers with schizophrenia, however, these treatments options are not available. And while these programs provide fascinating topics for further research, for the purposes of this study I am defining the current treatment system as the model of service delivery reflective of the dominant biological model, the model most frequently encountered by consumers with schizophrenia.

The current suggested model of treatment, promoted by the National Institute for Mental Health, is primarily adherence to a regimen of antipsychotic medication, with adjunct psychosocial treatments including case management or supportive therapy, social and vocational rehabilitation, and possible cognitive behavioral therapy (NIMH, 2006).

Similarly, treatment recommendations made by the Schizophrenia Patient Outcomes Research Team (PORT) (Lehman, et. al., 1998), a research project undertaken by the Agency for Health Care Policy and Research and the National Institute for Mental Health, included strong emphasis on medication interventions and adherence. Of the 30 recommendations in this report, the first twenty-one focused on medical interventions with schizophrenia. Of these twenty-one, the first nineteen discussed psychopharmacology and three focused on electroconvulsive therapy. Other treatment recommendations included vocational training, psychosocial interventions with families, assertive case management (ACM) and assertive community treatment (ACT). The report also recommends against the use of psychodynamic psychotherapy. While these recommendations for treatment are how the current treatment system is supposed to function and does for some consumers, for many the picture is quite different (Whitaker, 2002).

Of the 2.2 million people currently diagnosed with schizophrenia in the United States, it is estimated that 100,000 of these persons are homeless and 135,000 are in prison on any given day (Torrey, 2001). Other studies suggest that half of the 400,000 individuals who make up the homeless population of the United States, a doubling of the other estimate, have schizophrenia (Carpenter & Buchanan, 1994). Similarly stark is a report from a National Institute for Mental Health (NIMH), which found that only 60 percent of people with schizophrenia received any type of medical or psychiatric care during a given year (Regier, Narrow, Rae, Manderscheid, Locke, & Goodwin, 1993). Another study detailed in Torrey (2001), found that only 3 percent of all mental health consumers seen by psychiatrists in private practice carried the diagnosis of schizophrenia.

Under new restrictions from insurance companies, psychiatric inpatient hospital stays are often limited to 3 days (Luhmann, 2000). As described by one psychiatrist in Luhmann (2000), referring to cuts for coverage in hospital stays, “many of the cuts are really better for patients. Now hospitalization will focus on moving people to healthier levels of functioning immediately rather than doing deep intrapsychic work” (p. 242). Despite this optimism, a study published in *Hospital and Community Psychiatry* in 1992, as described in Torrey (2001,) found that many hospitals admit and discharge clients as many as 121 times during their lifetime. Similar studies were conducted in Illinois (Torrey, 2001) and New York (Muller and Canton, 1983). In Illinois it was found that 30 percent of patients discharged were readmitted in 30 days, and in New York it was found that 60 percent of discharged patients were readmitted within one calendar year.

Because hospitals are only reimbursed for brief inpatient hospitalizations, and there are minimal resources for community care, many people with schizophrenia currently continually revolve between shelters, hospitals, and jails (Torrey, 2001). And, as described in Luhmann (2000), despite recommendations by the NIMH and others, psychosocial interventions get “shortchanged because at present you cannot document its effectiveness the same way you can document the effectiveness of medication trials” (p. 264).

While it appears that the current treatment system is not meeting the needs of the population in the United States with schizophrenia, this system is also significantly costly. According to a study by Wu, Birnbaum, Shi, et. al. (2005) published in the *Journal of Clinical Psychiatry*, the overall cost of schizophrenia in the United States in 2002 was \$62.7 billion. Of this amount, \$22.7 billion was direct health care cost of

which \$7.0 billion went to outpatient, \$5.0 billion to drug costs, \$2.8 billion inpatient costs, \$8.0 billion long-term care. The total direct non-health care costs, including living cost offsets, were estimated to be \$7.6 billion. The total indirect costs were estimated to be \$32.4 billion. Torrey (2001) estimates that the costs of federal disability payments for schizophrenia alone were to equal \$10 billion in 1999.

There is great variability between private and public treatment facilities as well as facilities and programs state to state. A brief summary of federal programs and nationally recommended treatment methods however, shows a model, based primarily on medication, which is very costly and which is not reaching or successfully treating a large percentage of people diagnosed with schizophrenia in the United States.

Conclusion

Within this chapter I began by detailing the scope, population, demographics and course of illness and recovery in schizophrenia. Next, I provided a historical synopsis of the diagnosis of schizophrenia, theories of etiology, and treatments. I concluded with a description of the current mental health treatment system, both in terms of how treatment is recommended to be delivered, and how treatment is actually delivered since deinstitutionalization. The next chapter, Chapter IV, is a literature review of current research on the etiology and treatment of schizophrenia from the biological perspective.

CHAPTER IV

BIOLOGICAL PERSPECTIVES

The previous chapter, Chapter III, provided an introduction to schizophrenia, the history of division between biological and psychodynamic perspectives on schizophrenia etiology and treatment, and an overview of the current treatment system. As detailed in Chapter III, the current treatment system is based on a biological perspective that has become the increasingly dominant model since the time of the discovery of neuroleptic medications (Whitaker, 2002; Whitaker, 2005; Valenstein, 1998; Kraly, 2006).

As schizophrenia, within the medical model, has come to be considered a biological disease, research on schizophrenia is now primarily based around biological etiology models, and treatment focuses on psychopharmacology (Austrian, 2000, Kraly, 2006). This chapter, Chapter IV, reviews the literature from the biological perspective on schizophrenia. The purpose of this chapter is to understand current theories of schizophrenia etiology and treatment within this model in order to further illuminate the role of psychodynamic psychotherapy in the treatment of schizophrenia. In order to achieve this purpose, this chapter addresses questions such as: is schizophrenia solely a biologically based illness; should it only be treated with psychopharmacology and supportive therapies; what do these treatments address, and how effective are these treatments? In order to answer these questions, within this chapter I will review research on genetic, neurochemical, neuroimaging, neurobiological, viral and combined theories

of the etiology of schizophrenia, recommended treatments from a biological perspective as well as the efficacy of these treatments.

Significantly, while researching for this literature review, there were very few resources available on the topic of biological perspectives on schizophrenia in social work sources. The most recent resource reviewing research on the biological basis of schizophrenia in the social work databases comes from Taylor (1987, 1989) in *Social Work*. More recent research available on the etiology of schizophrenia is published in medical journals such as *Schizophrenia Bulletin*, *American Journal of Psychiatry*, and *Biological Psychiatry* with language that is often prohibitively technical to those without a medical degree. The lack of biological or integrated biopsychosocial explanations of schizophrenia in social work journals is also reflected in graduate social work classrooms, according to a study by Lacasse and Gomory (2003). Lacasse and Gomory (2003) found that although a biomedical model is taught in a majority of social work courses on psychopathology, “a handful of secondary (textbooks) rather than primary (research articles) sources provide the majority of the mental health content in these courses” (p. 383). If there is very little information about biological advances in schizophrenia etiology and treatment taught to or available to social workers, how then do social workers practice effectively within this model?

Etiology

Neurochemical

Neuroleptic medications were first found to have anti-psychotic properties in the late 1950's and early 1960's. Researchers soon understood that these medications

blocked dopamine receptors in the brain (Whitaker, 2002). Since that time, research on the neurochemical etiology of schizophrenia has focused mainly on understanding the connection between dopamine and schizophrenia (Valenstein, 1998; Andreasen, 2001).

Until very recently, it was assumed by researchers that because neuroleptic medications worked by blocking dopamine receptors in the brain, that schizophrenia must be caused by excess dopamine in the brain (Seeman, 1995; Snyder, 1972; Taylor, 1987). It was initially deduced, as suggested by Snyder (1989), that if blocking transmission of dopamine “relieves the schizophrenic symptoms, then one could speculate that schizophrenia abnormalities are related to excess dopamine release or perhaps hypersensitive dopamine receptors” (pp. 174-175). Despite extensive research into the role of dopamine in the etiology of schizophrenia, and promotion by pharmaceutical companies of the dopamine hypothesis (Valenstein, 1998), there is little agreement “on how neurochemistry is actually altered” (Torrey, 2001, p. 144).

Torrey (2001) suggests that some impediments to any concrete evidence or conclusions on the role of dopamine may stem from the fact that most people with schizophrenia are on medications and medications also affect neurotransmitters. The progress of research on the role of dopamine is also impeded by the fact that neurotransmitter levels vary from brain area to brain area so it is difficult to create consistency within studies (Torrey, 2001). A comprehensive review of all current findings on dopamine by Carlsson and Lecrubier (2004), suggests that there can be no one hypothesis of dopamine in schizophrenia as some areas of the brain in people diagnosed with schizophrenia show too little dopamine while other areas show an excess of the neurochemical. Other research into neurochemical theories has found that the role

of dopamine in schizophrenia may actually be dependent on variations in glutamate or gamma-aminobutyric acid (GABA) other essential neurotransmitters in the brain (Shean, 2004; NIMH, 2006; Grace, 1991; Huang, Matevosian, Whittle, Kim, Schumacher, Baker, & Akbarian, 2007; Garbutt & Van Kammen, 1983; Brenner, Boker, & Genner, 2001; Wassef, Baker, & Kochan, 2003).

It is concluded in an article by Garza-Trevino, Volkrow, Cancro, and Contreras (1990) that “a dopamine hypothesis by itself does not appear to be sufficient explanation for the schizophrenic syndrome, and dopamine is most likely to play only a partial role in a complex disturbance” (p. 978). This theory is supported by Torrey (2001) who states that dopamine and other neurochemical theories of schizophrenia “are not really theories of causation but rather theories of pathophysiology” (p. 161), meaning that whether or not altered dopamine or glutamate or GABA are eventually proven to cause schizophrenia, the question remains as to how these systems became altered (Torrey, 2001). It is thought that these alterations may occur genetically, and so a great amount of current research is also focused on the role genetics in schizophrenia.

Genetic

It has been shown in multiple family and adoption studies that genetics may play a role in the development of schizophrenia (Dworkin & Lenzenweger, 1984; Gottesman, 1991; Andreasen, 2001). While there have been harsh critiques of these studies by researchers like Litz, Blatt, and Cook (1981) (as cited in Shean 2004), it is generally accepted that genetics do play some causal role in schizophrenia (Torrey, 2001; Shean, 2004; Valenstein, 1998). One example of the likelihood of a genetic basis for

schizophrenia comes from the statistic that children with one parent with schizophrenia have been shown to have a 10 percent chance of having the illness, while if both parents have schizophrenia the risk increases to 40-50 % (Andreasen, 2001). Research by Grinspoon and Bakalar (as cited in Austrian, 2000) suggests that for monozygotic twins, if one twin has schizophrenia there is a 50-60 percent chance that the other will have schizophrenia, while in dizygotic twins, “the probability is only 10 to 15 percent” (p. 101).

While there have been strong findings over the past 20 year suggesting a genetic basis for increased risk of schizophrenia within families (Dworkin & Lenzenweger, 1984), little is still known about exactly how or which genes play a role in the illness. Initially it was thought that the effected genes must be genes related to dopamine, however a study by Sabate, Campion, d’Amato, Martres, Sokoloff, Giros, et al. (1994), proved that this was not the case.

A summary of research by the editors of the American Journal of Psychiatry in (1994) reported that,

“surprisingly, despite decades of epidemiologic research pointing to genes as etiologic mechanism for schizophrenia, application of the sophisticated methods of molecular and statistical genetics that are now available has not revealed which genes are involved and how gene products lead to the disorder” (Editorial, 1994, p. 3).

Another review by the same editors (2000) suggested there had still been little progress, (Editorial, 2000), as they once again called for a renewed focus on “identifying the elements of the phenotype that are heritable” (p. 1039).

Other research reviewed by Torrey (2001) suggests that some genes have been identified on chromosomes, 1,5, 6, 8, 13, 15, and 22, which were thought to have a role in

schizophrenia, however researchers have had trouble replicating results. Some of the most current research on the genetics of schizophrenia by Bray and Owen (2001), suggests that the risk of schizophrenia is caused by multiple genes working together, however very little else is known about the actual number or activity. Bentall (2003), seconds these findings by Bray and Owen (2001) stating that, if “one or two genes played a major role, they would have almost certainly been identified by now” (p. 451). Bentall also suggests that the “genetic contribution to psychosis is caused by many genes of minor effect” (p. 451), which may play a role in schizophrenia in some people and none in others as. According to Bentall, psychotic disorders appear to be “heterogeneous on a molecular level” (Bentall, 2003, p. 451).

One argument for why there has been so much difficulty in finding a stronger genetic link, or specific genes responsible for schizophrenia comes from research suggesting that schizophrenia is actually a spectrum of illnesses (Shean, 2004). The author cites a study by Gurling, Read, and Potter (1991), that suggests that schizophrenia may be an illness, like diabetes, that has different genetic factors for different sub-types. The idea of diagnosis actually serving to confuse genetic research is supported by a study by Dworkin and Lenzenweger (1984). These researchers found that in at least 3 family studies the genetic component to the findings was entirely dependent on what criteria was used to diagnose schizophrenia (Dworkin & Lenzenweger, 1984).

According to some researchers, genetic research itself contains evidence showing that genes may only play a partial role in causing schizophrenia. As suggested by Tsuang (2000) and supported by Dworkin and Lenzenweger (1984) and Torrey (2001), even in the scientifically sound genetic research studies, “identical twins show average

concordance rates of only 50%” while “rates of 100% would be expected on the basis of genetic equivalence alone” (p. 210). Further critique is brought by Torrey (2001) who questions why, if from “the early nineteenth century to the mid-twentieth century, most individuals with schizophrenia in Europe and the United States were institutionalized, and their rate of reproduction was extremely low” (p. 160) did incidence rates of schizophrenia not only not decrease but actually increase?

Torrey (2001) concludes his chapter on genetics in his book *Surviving Schizophrenia* with the proposal that genetic research in schizophrenia may turn towards suggesting that genetics only predispose a person to schizophrenia while other factors in the environment including obstetric complications, season of birth, and possible viral exposure may serve to trigger this predisposition. This view is supported by Williamson (2006), and Tsuang (2001), who suggest that “genetic factors may cause errors in brain development and synaptic connections” while “a broad range of environmental components may further damage the brain” (p. 210).

Environmental, Neurobiological, and Viral Theories

In a critical review of brain imaging and neurobiology literature from 1980 until 1999, Harrison (1999) concluded that there are significant structural differences in the brains of people with schizophrenia.

“Despite the many controversies and contradictions, there are now established facts about the neuropathology of schizophrenia. The disorder is associated with ventricular enlargement and decreased cortical volume. The pathology is neither focal nor uniform, being most convincingly demonstrated in the hippocampus, prefrontal cortex and dorsal thalamus. The pattern of abnormalities is suggestive of a disturbance of connectivity within and between these regions, most likely originating during brain development.” (p.p. 611-612).

These findings are supported by Johnstone and Owens in their chapter in Lawrie, Johnstone, and Weinberger's (2004), *Schizophrenia: From Neuroimaging to Neuroscience*. Johnstone and Owens found that despite the fact that some studies had small samples and flawed methodology, "on macroscopic study, strong and consistent support can be found for ventricular enlargement, decreased cortical volume, and disproportionate volume loss from the temporal lobe" (p. 14). Harrison (1999), concludes his review however with the statement that despite some significant findings, the "neuropathology of schizophrenia remains obscure" (p. 593).

In his book *Mind, Brain, and Schizophrenia*, Peter Williamson (2006) explains how with the birth of neuroimaging techniques like the PET scan, scientists envisioned that the understanding of the neurobiological basis of schizophrenia was not far away. According to Williamson (2006), this has not been entirely the case, although he points to some interesting findings from PET and MRI scans which point back to neurochemical theories in that they show "dopaminergic hyperresponsivity" (p. 10).

While studies do show that there are definite structural changes in the brain anatomy and neurochemical functioning of people with schizophrenia, as reviewed, genetic theories do not fully explain how these changes occur. For this reason, researchers have also looked to developmental and viral possibilities to explain these changes that occur physically for those with schizophrenia.

Evidence for structural differences being developmentally related to schizophrenia, as opposed to genetically, is made evident in the research by McNeil, Cantor-Graae, and Weinberger (2000) published in the *American Journal of Psychiatry*. McNeil et al. (2000) found in a study of twins, which compared twins with and without

schizophrenia, that twins with the illness “had smaller left and right hippocampi as well as larger left lateral ventricles and third ventricles” (p. 203). The authors concluded that these differences were explained by obstetric complications, specifically trauma during labor or prolonged labor (McNeil, Cantor-Graae, Weinberger, 2000). Andreasen (2001) argues against such theories of early developmental interruption however, stating that they do not explain why brain abnormalities do not show up in people with schizophrenia until the late teens and early twenty’s (as opposed to other developmental disorders).

Breggin (1994), and Valenstein (1998), also both discredit the majority of research into structural abnormalities in brains of people with schizophrenia. These authors argue the observed brain abnormalities in the studies could easily be a result of the fact that a large percentage of people in the research samples had been on neuroleptic medication or had electroconvulsive therapy. The authors also cite evidence showing that life experiences (stress, social interactions etc.) have been shown to create structural changes in the brain.

Viral theories are supported by statistics in Williamson (2006) and Torrey (2001). These figures show an increased schizophrenia among people born in spring months (their mothers may have been exposed to influenza), as well as among people living around domesticated cats, animals that are known to carry a parasite whose antibody has been found in increased levels in people with schizophrenia. Research, documented by Garza-Trevino et al. (1990), has shown that unknown viral agents have been isolated in the cerebrospinal fluid of people with schizophrenia, although when injected into primates, these fluids failed to cause the animals to acquire schizophrenia or

any other symptoms. Other theories of alternative causes for schizophrenia include ideas of drug use, exposure to toxins, and hormonal changes (Andreasen, 2001).

While the combined efforts of genetic, neurochemical, neuro-imaging, development, and viral research have begun to more clearly delineate the physiological changes that occur in schizophrenia, they still do not fully explain what causes the illness. Marley (1998) suggests that this may be in part due to the flawed idea, as previously discussed, of schizophrenia being one illness. “One possible reason for the lack of clarity arising from the biological research is that schizophrenia may be more than one illness” (p. 438). Marley goes on to suggest that the other reason for the lack of clarity might be due to the “limited role played by nonphysical factors that influence the individual and the illness” (Marley, 1998, p. 438). Another reason why it may be publicly emphasized that schizophrenia is a purely biological disease, despite continued confusion as to the exact nature of how it is caused, is due to the mental health parity movement led by organizations like the National Alliance for the Mentally Ill (NAMI). These organizations promote the biological disease model, not only because it is essential in reducing stigma, but if schizophrenia is considered a disease by insurance companies, mental health consumers have increased hope of receiving full medical benefits akin to those provided for chronic physical illnesses (Luhmann, 2000).

Theories of Combined Etiology

Due to the findings that no one area of scientific research can fully explain the etiology of schizophrenia (Siebert, 1999) many theories exist which explain schizophrenia as some combination of all of the possible etiological factors discussed

above. Such models include the “diathesis stress model” proposed by Gottesman and Shields (1982) the “interactive developmental systems model” of Strauss and Carpenter (1981) and the vulnerability-stress model proposed by Zubin and Spring (1977). These models are reviewed in Yank, Bentley, and Hargrove (1993). All of the models are similar in that they all propose some interplay of genetic, biological, developmental, environmental and stress-related factors. As they are described in Yak, Bentley, and Hargrove (2003), they are all paradigms which integrate factors including “heredity, abnormal brain structure and functioning, physiological and psychological development, and early learning” (p. 56) all of which are vulnerability factors which then “[interact] with stress to create a threshold for symptomatic illness” (p. 56). These theories are supported by research done by Tienari Wynne, Sorri, Lahti, Laksy, Moring, et al. (2004), that studied the adoption of children with a high genetic risk for schizophrenia. The research showed that adoptees at high genetic risk for schizophrenia are “significantly more sensitive to adverse” versus “‘healthy’ rearing patterns in adoptive families than are adoptees at low genetic risk” (p.216). Meaning that those children adopted into more “stressful” families were actually more likely to manifest their genetic predisposition to schizophrenia (Tienari, et al. 2004).

The National Institute for Mental Health announced in 2000, that in the last 50 years,

“much has been learned about mental disorders and their effects on the brain. Revolutionary scientific advances in neuroscience, molecular biology, genetics and brain imaging have provided some of the greatest insights in the complex organ that is the seat of thought, memory, and emotion. Thanks to these new tools, the scientific evidence that mental illnesses are brain disorders now exists” (as cited in Lacasse & Gomory, 2003, pp. 392-393).

However it is clear from this brief review of the literature, that while schizophrenia may indeed be an illness of the brain, the explanations for this illness are highly complex and multilayered and may also include some elements of environmental influence.

Treatment

While the etiology of schizophrenia is clearly a complicated and multidimensional picture, biological perspectives on treatment of schizophrenia have, since the 1950's, been strongly focused on anti-psychotic medications. As described in Bola (2006), clinical treatment guidelines “uniformly recommend treatment with anti-psychotic medication” (p. 264). An international survey of treatment guidelines by Gaebel, Weinmann, Sartorius, Rutz, and McIntyre (2005) showed that 24 of 24 reviewed treatment guidelines recommended anti-psychotic drugs as the first line of treatment. The three most widely cited and utilized references for treatment of schizophrenia in the United States are the “Schizophrenia Patient Outcomes Research Team (PORT)” treatment recommendations, Lehman et al. (1998, 1998a), The American Psychiatric Association's (APA) “Practice Guideline for the Treatment of Patients With Schizophrenia” (APA, 2004), and the “Clinical Antipsychotic Trials of Intervention Effectiveness” (CATIE) study conducted by investigators Lieberman, Stroup, McEvoy, Swartz, Rosenheck, Perkins, et al. from the National Institute of Mental Health (NIMH) and published in the *New England Journal of Medicine* (2005). All of these studies recommend anti-psychotic drugs as the “cornerstone of treatment for schizophrenia” (Lieberman, et. al. 2005, p. 1210), and all three are authored or edited by either or both Jeffrey Lieberman or Anthony Lehman.

According to the research by Lieberman et al. (2005) while new atypical antipsychotics (as opposed to first generation “conventional” antipsychotic drugs), “are similar to conventional drugs” (p. 1210) in regards to their reduction of psychotic symptoms, they produce far fewer neurological side effects like tardive dyskinesia. While Lieberman et al. (2005) recommend the new atypical antipsychotics, due to fewer side-effects and therefore better medication compliance amongst participants in the clinical trials, the researchers still question some of the safety risks of these newer drugs. The authors state that these new drugs have been shown to “induce weight gain, and alter glucose and lipid metabolism” (p. 1210), and state that ultimately “how clinicians, patients, families, and policymakers evaluate the trade-offs between efficacy and side effects, as well as drug prices, will determine future patterns of use” (p. 1222). There is no mention anywhere within this study, even as it raises questions about compliance, about psychotherapy or psychosocial treatments.

The APA treatment guidelines (APA, 2004) also recommend that during acute psychosis in schizophrenia that “pharmacological treatment be initiated promptly” (p. 4). The APA guidelines do however also stress the importance of other elements of treatment including developing a therapeutic alliance to promote treatment adherence, psychoeducation and therapy with the family, coordinating treatment with case managers, and attending to the patients social circumstances and functioning (p. 11). There is no mention in the guidelines of psychodynamic psychotherapy as a recommended intervention, however supportive therapy on an outpatient basis or in the form of an assertive community treatment team (ACT) is recommended (p. 10).

The recommendations by the PORT research team, are also focused on psychopharmacological interventions. Of the 30 recommendations for schizophrenia treatment put forth by the authors of the PORT report (Lehman, et al., 1998a) the first 18 recommendations involve psychopharmacology, and recommendations 19 through 21 are focused on electroconvulsive therapies for treatment resistant patients (p.7).

Recommendation 23, the first and only instance in which psychodynamic psychotherapy is mentioned in any of the treatment guidelines, recommends explicitly against it. The recommendation states that, “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” (Lehman, et al., 1998a, p. 7). The rationale given by the study authors for this recommendation is that,

The scientific data on this issue are quite limited. However, there is no evidence in support of the superiority of psychoanalytic therapy to other forms of therapy, and there is a consensus that psychotherapy that promotes regression and psychotic transference can be harmful to persons with schizophrenia. This risk, combined with the high cost and lack of evidence of any benefit, argues strongly against the use of psychoanalytic therapy, even in combination with effective pharmacotherapy” (p.p. 7-8).

Lehman, et al. (1998), note that this recommendation is a “recommendation based primarily on expert opinion, with minimal research-based evidence” (p. 2).

Other recommendations within the PORT report include individual and group therapy “employing well-specified combinations of support, education, and behavioral and cognitive skills training” (Lehman, et al. 1998a, p. 8), psychosocial family interventions, and vocational rehabilitation. Two recommendations are aimed specifically at enforcing the fact that no family interventions should be based on the premise that

family dysfunction is the etiology of schizophrenia or that only families with high levels of expressed emotion should be included in such interventions (Lehman, et al., 1998).

The final two recommendations promote assertive case management (ACM) and assertive community treatment (ACT) programs.

Treatment Outcomes and Recovery

From a review of the literature on etiology and treatment, it is clear that the biological model of schizophrenia is centered on the theory of schizophrenia as a brain disease that should be treated by psychopharmacology. The question remains however as to how effective the biological model is at treating and improving quality of life for those with schizophrenia.

Currently, the most commonly quoted statistics within the biological community on anti-psychotic treatment are that 70 percent of people with schizophrenia show significant symptom improvement on anti-psychotics, while 25 percent show minimal improvement and 8 percent show no improvement or actually get worse (Torrey, 2001; Shean, 2004; Whitaker, 2005). While research shows that 70 percent of people with schizophrenia show improvement on anti-psychotic medications, this should not necessarily be taken to imply that 70 percent of people recover while taking anti-psychotics. According to Fonagy and Roth (2005), “there is evidence that despite appropriate levels of medication, many patients continue to experience residual symptoms...full remission of symptoms occurs in less than two-thirds of patients” (p. 266). These statistics are particularly significant when compared to recovery rates in alternative treatment programs (DeSisto, et. al. 1995; Harding, 2002, 2003) or on an

international scale, where full recovery has been documented to be as high as two-thirds of those diagnosed with schizophrenia (Jablensky, 1992).

A comprehensive review of biological research on pharmacological treatments by Cohen (2002) found that many such studies were of “uniformly poor” (p. 228) quality or were “conducted in such a way as to make results of drug trials and other studies appear in the best light possible for tested drugs” (p. 228). It has also been found that authors of research studies often have ties to pharmaceutical companies or that pharmaceutical companies may actually author most of the major research studies, merely paying doctors for the use of their names (Cohen, 2002; Whitaker, 2002; Valenstein, 1998). Also damaging to the biological model’s pursuit of scientific credibility is a study by Kuno and Rothbard (2002), that found racial disparities in antipsychotic prescription patterns among publicly insured clients.

While the efficacy of antipsychotic medications continues to be promoted within the biological model, and has inarguably brought great relief and healing to large numbers of people plagued with psychosis (Torrey, 2001, APA, 2004; Lehman, et al., 1998, 1998a; Lieberman, et. al. 2005) many outside this model continue to question these conclusions (Cohen, 2002; Whitaker, 2002; Breggin, 1994; Valenstein, 1998). Some studies suggest that not only are people with schizophrenia treated within this model not recovering with greater frequency, they may actually be getting worse and in greater numbers (Cohen, 2002, Whitaker, 2005). According to an article by Heggarty, Baldessarini, Tohen, Waternaux, and Oepen, (1994), published in the *American Journal of Psychiatry*, outcome for schizophrenia “is now approaching levels reported prior to the 1950’s, despite the availability of modern treatment” (p. 1415). While this trend towards

poorer outcomes may in part be due to increasingly strict diagnostic criteria in newer publications of the DSM, research documented by Whitaker (2005) suggests that antipsychotics themselves may be in part to blame for these statistics. According to Whitaker (2005) the research,

“suggested that with minimal or no exposure to neuroleptics, at least 40% of people who suffered a psychotic break and were diagnosed with schizophrenia would not relapse after leaving the hospital, and perhaps as many as 65% would function fairly well over the long term. However, once first-episode patients were treated with neuroleptics, a different fate awaited them. Their brains would undergo drug-induced changes that would increase their biological vulnerability to psychosis, and this would increase the likelihood that they would become chronically ill (and thus permanently disabled)” (p. 29).

Current research on and treatment of schizophrenia with psychopharmacology is primarily focused on the effectiveness of medications as defined by tolerability and efficacy (reduction of symptoms) (APA, 2004; Freudenreich, 2008). Effectiveness remains an issue with neuroleptic medications, even the newer atypical antipsychotics, due to high number of side effects and extremely varied results (Fonagy & Roth, 2005). One example of the central role of tolerability, adherence, and efficacy in medication treatment is the CATIE study (Lieberman, et al. 2005). While goal of the study was to measure effectiveness of antipsychotics, ultimately effectiveness was measured by which medications people stayed on due the fact that 74 percent of the total 1493 people in the sample discontinued the medication due to “inefficacy or intolerable side effects or for other reasons” before the study was completed (p. 1209). Despite claims that the new atypical antipsychotics actually have fewer of the extra-pyramidal symptoms (EPS) (including dry mouth, tachycardia, constipation, weight gain, and tardive dyskinesia) than the conventional antipsychotics (Torrey, 2001), to some “these claims are largely drug

company hype” (Read, Mosher, & Bentall, 2004, p. 109). As suggested within a guide to psychiatric treatment of psychosis written for psychiatrists (Freudenreich, 2008), reducing side effects is often as large a goal of treatment as reducing symptoms,

“Do not promise a cure, but suggest that improvement in overall well-being without freedom from symptoms is probably a more realistic goal. In many patients, switching [medication] to reduce cardiovascular risk factors (as opposed to psychiatric efficacy) has become an important risk-benefit consideration” (p. 114).

Side effects with medications are not just limited to weight gain or involuntary movements, many people with schizophrenia report that whether or not their psychotic symptoms have been reduced, they still feel emotional side effects. As described in Barron and Sands (1996) while antipsychotics reduce what are known as the positive symptoms of schizophrenia (i.e. hallucinations) “in the area of negative symptoms (particularly apathy and difficulties in interpersonal and social role functioning) neuroleptic medications appear to have a limited effect, and may indeed at times, exacerbate such conditions” (p. 110). A study by Wallace (1994) documenting thousands of calls regarding medications to SANELINE, a telephone support line for people coping with severe mental illness, found that

“almost all of our callers report sensations of being separated from the outside world by a glass screen, that their sense are numbed, their willpower drained and their lives meaningless. It is these insidious effects that appear to trouble our callers much more than the dramatic physical ones, such as muscular spasms” (as cited in Cohen, 2002, p. 227).

The failure of biological research and treatment to consider the actual lived experience of those on anti-psychotic medications is becomingly increasingly well documented (Cohen, 2002). With the biological models focus on psychopharmacology, and the restrictions on hospital stays and outpatient treatment placed by managed care,

treatment of schizophrenia has become increasingly focused on reducing psychotic symptoms (Whitaker, 2002; Cohen, 2002; Breggin, 1994; Luhrmann, 2000). Because of this focus, David Cohen, a social work professor at Florida International University suggests that research on medications does not actually “provide meaningful information about the real-life ‘effectiveness’ of neuroleptics” (Cohen, 2002, p. 223) as little attention is paid to how people with schizophrenia are recovering in regards to their emotional or occupational functioning. Interestingly as this focus on symptom reduction increases, and “medications increasingly [take] the place of relationships with patients” (Luhrmann, 2000, p. 258), people may be actually less apt to take medications, as it has been shown repeatedly that it is clinical relationships that promote medication adherence (Harvard Mental Health Letter, 2007).

Conclusion

The literature on the etiology of schizophrenia from a biological perspective shows that the causes of schizophrenia are still being sought amongst interrelated biochemical, genetic, viral and neurobiological factors. None of these avenues of research have yet to fully explain how schizophrenia originates. There remains some possibility that environmental factors such as stress may play an important role in contributing to the development of schizophrenia as well as how and when it manifests. While biological model literature on the treatment of schizophrenia strongly recommends treatment with atypical antipsychotic medications and recommends against psychodynamic interventions, there remains unanswered questions about how effective these biological treatments are, and how people with schizophrenia are feeling and

recovering in areas beyond symptom reduction. In the following chapter, Chapter V, I will review the literature from psychodynamic perspectives on the etiology and treatment of schizophrenia.

CHAPTER V

PSYCHODYNAMIC PERSPECTIVES

The previous chapter, Chapter IV, contained a review of the literature from a biological perspective. While there is some strong evidence in the literature for genetic and neurobiological factors in the etiology of schizophrenia, there continues to be no comprehensive biological theory on why and how schizophrenia develops. Similarly, while biological treatments have done a great deal towards improving the lives and reducing the painful psychotic symptoms of those with schizophrenia, critical research suggests these treatments have not yet provided the possibility for full recovery for the majority of people with schizophrenia. In this chapter, Chapter V, I review the literature on psychodynamic therapies for schizophrenia. I will examine the evidence for psychogenic explanations of schizophrenia and evaluate current recommended interventions from a psychodynamic perspective to evaluate how and if this modality may play a role in schizophrenia treatment.

Brief Historical Overview

Since the time of Freud and Jung, there have been countless theories on the psychogenic origins and treatment of schizophrenia (Stone, 1999). While Freud throughout much of his life did not believe in the utility of psychoanalytically oriented treatments for schizophrenia, due to an inability for the formation of transference, he did

believe in the possibility for psychogenic origins of schizophrenia (Freud, 1911). Freud's theory of schizophrenia appears based in the idea of an original trauma, or narcissistic injury, leading to a withdrawal of internal and external objects creating a decathexis of objects and a hypercathexis of ego. This process leads to the symptoms of psychosis, which he saw as defensive attempts at self-healing or at rearranging reality to decrease conflict with the id. According to Freud "a fair number of analyses have taught us that delusion is found applied like a patch over the place where originally a rent had appeared in the ego's relation to the external world" (Freud, 1924, p. 150).

The work of Carl Jung also explored ideas for the psychogenic etiology of schizophrenia. Jung's early theories proposed schizophrenia to be caused by vulnerabilities of the ego due to an inability to assimilate the archetypes, with the purpose of therapy aimed at providing a corrective experience of these "emotional predispositions derived from the collective unconscious" (Bachmann, Resch & Mundt, 2003, p. 156; Jung, 1939). Later Jung came to believe that trauma also played a significant role in the origins of schizophrenia (Jung, 1914, 1939; Koehler, 2004), concluding ultimately that psychotherapy of schizophrenia could be successful, but that

"For psychotherapy to be effective, a close rapport is needed, so close that the doctor cannot shut his eyes to the heights and depths of human suffering. This rapport consists, after all, in a constant comparison and mutual comprehension, in the dialectical confrontation of two opposing psychic realities" (Jung, 1995, p. 166).

Evolving out of the work of Freud and Jung, and gaining prominence during the period surrounding World War II (Luhmann, 2000), many famous therapists and analysts devoted themselves to theories of the psychogenic origins and psychological treatments of schizophrenia (Read, Mosher, & Bentall, 2004). These influential theorists

included Klein (Koehler, 1994), Searles, (1960, 1965), Fromm-Reichmann (1950), Winnicott (1975) Semrad (1969), Federn (1952) Arieti (1955), Spohnitz (1969) and Sullivan (1956, 1962, 1964)

While it is not possible within this chapter to fully examine the numerous theories laid forth by these early clinicians, I found it necessary to introduce them here as their names appear continually in modern literature on this topic and their influence remains the foundation of the majority of the work of all psychodynamic clinicians attempting to understand and treat schizophrenia today (Stone, 1999).

Etiology

Although biological research is now becoming more accepting of the possibility for a diathesis or vulnerability stress model of schizophrenia (Yak, Bentley, & Hargrove, 2003), strong controversy remains surrounding psychogenic models of schizophrenia development (Willick, 1990). These avenues of research are important to pursue though because as Siebert (1999) pointed out, if schizophrenia was a purely biological disease, why then would recovery outcomes be so significantly positively influenced by various non-pharmacological treatments (Harding, 2003, 2002)?

The divisive nature of psychogenic theories of schizophrenia etiology can mostly be traced to early hypotheses regarding the family's role in schizophrenia. The most controversial of these theories were those which contained painful and stigmatizing ideas like Fromm-Reichmann's "schizophrenogenic mother" (Robbins, 1993) or the idea that the parents "double bind" communication style caused confusion to the degree to which psychosis was the only escape for a child (Bateson, 1972). Interestingly, despite the

current emphasis on a predominantly biological etiology of schizophrenia, 91 percent of U.S. citizens polled in a study by Link et al. (as cited in Read & Haslam, 2004) still believe that “stressful conditions” cause mental illness.

While some researchers still adopt a strict classically psychoanalytic view of schizophrenia (Frattaroli, 2001, 2002), the majority of clinicians writing on psychogenic origins of schizophrenia today are focused instead on how family, stress, or trauma might be contributing factors in the development of schizophrenia. It is generally accepted by these researchers that there is a genetic component to the illness (Berger, 2001; Robbins, 2002) and so their work is centered on how environmental situations may exacerbate or trigger the illness, or to say it differently, their work is focused on defining the “stress” in the vulnerability stress model (Gabbard, 1994; Stone, 1999). In the next section I will review two of these theories, those of family environment and trauma.

Family Theories

One of the major arguments against the use of psychotherapy in the treatment of schizophrenia is the idea that implicit within a psychodynamic psychotherapeutic approach is the suggestion that the person with schizophrenia, or more specifically their parents, may be blamed for causing the illness (Lehman et al., 1998). With the increased emphasis on genetics in mental health, exploring the role of family in the manifestation of schizophrenia has become “taboo” (Read, Seymour, & Mosher, 2004, p. 253). Some critics argue that the silence or denial around these issues by the biological perspective is quite hypocritical because psychosocial interventions (recommended within the biological model) do suggest behavior and communication changes that families can

make to help their family member's recovery, and prevent relapse (Read, Seymour, & Mosher, 2004). While it is in no way universally true that clinicians promoting a psychodynamic approach think family of origin plays a causal role in schizophrenia, and many point to the significant contribution (up to 86 percent) of genetics (Cannon, Kaprio, Lonnqvist, Huttenen, & Koskenvuo, 1998; Robbins, 1992) some researchers do still suggest that family environment and communication styles affect schizophrenia etiology (Horowitz, 2004).

One of the most commonly cited sources of evidence for the role of family environment in the etiology of schizophrenia is the fact that identical (MZ) twins only have a 50% chance of both acquiring schizophrenia (Tienari et al., 2004). Based off these initial findings, a 40-year study was conducted in Finland by Tienari et al. (1985, 2004). The study followed 91 children of mothers with schizophrenia who had been adopted versus a control group of children with no genetic predisposition for the illness. Of the 7 children who later developed psychosis, 6 were children of mothers with schizophrenia. Significantly however, all of the children who later developed psychosis were found to have been adopted into families that were assessed by the researchers to be severely disturbed by an emotional health rating scale (OPAS), while no children, even with genetic predispositions, raised in families deemed healthy later developed schizophrenia. The authors concluded from this research that the health of a family environment (assessed by criteria such as hostility, rigidity, conflict, and productivity of communication) can reduce the risk for a genetically susceptible child by 86 percent. (Tienari et al., 2004). This research is of particular interest because it was adoption studies conducted by Kety, Rosenthal, Wender, and Schulsinger (1968), that initially

gave great weight to the role of genetics in schizophrenia etiology, instigating the beginning of the end of psychotherapy as a recommended intervention.

Family communication, specifically communication deviance, expressed emotion (EE), and affective style are some of the oldest psychogenic theories and widely researched causal factors in the study of schizophrenia (Lidz, 1969; Wynne & Singer, 1963). Fort (1990) conducted a study in which parents of children with schizophrenia and parents with normal children, as well as children with schizophrenia and normal children, were asked to explain proverbs to one another on tape recordings and listen blindly. Remarkably, Fort found that although the effects of communication deviance were present for both parents of children with schizophrenia and children with schizophrenia on the task, when children with schizophrenia listened to the parents of normal children they performed on par with normal children but were impaired in the task when listening to the parents of other children with schizophrenia.

Another test of the role of EE (defined by the categories of hostility, criticism, and emotional over-involvement) and affective style was conducted at UCLA with 64 parents of teenagers with some behavioral difficulties but no history of psychosis. Fifteen years later, this study found that of the families where both parents had rated very high in expressed emotion, 76 percent of the children had a broad-spectrum form of schizophrenia (Goldstein, 1987). Also of note in EE studies are findings that clients returning to homes with families with high EE have a 66 percent chance of relapse over a two-year period as compared to 27 percent rate of relapse for those in low EE families (Kavanagh 1992, as cited in Read, Seymour, & Mosher, 2004).

Horowitz (2004) presents a detailed account of how EE and communication deviance may contribute to schizophrenia. He asks,

“what would a vulnerable child experience if, day after day, an adult were vague, unclear, or tangential...affected by his or her own fluid associations, challenged, distorted, and ‘corrected’ the child’s feelings thoughts and wishes, as though the child kept misreading internal states of self and others?” (p. 237).

According to Horowitz, the effects of these styles of relationships in children include poor concentration, a social withdrawal, and isolation. These changes lead to less opportunity for interaction, reassurance, and reality testing, which in turn leads to a less clearly defined self (Horowitz, 2004). In some cases the effects of this are an extreme anxiety of self, what Laing (1965) termed ontological insecurity, which according to Horowitz, (2004) is a state of being which underlies a lot of behaviors within schizophrenia.

Another important line of research into the role of family in the development of schizophrenia is a study done by Karon and Widener (1994), on pathogenesis in parents of those with schizophrenia. Within this study a pathogenesis score was created through the analysis of Thematic Apperception Tests (TAT) responses by parents. The researchers scored a response as pathogenic if the respondent considered their own needs over those of a child or dependent person if those needs conflicted with their own. On this test, mothers of normal children scored a pathogenesis score of around 35 percent, while mothers of children with schizophrenia scored approximately 75 percent. Other research by Myhrman and colleagues (1996), (as cited in Ver Eecke, 2003) found that of all the children born in Finland in 1966, the incidence of schizophrenia was double for those children whose mothers had expressed not wanting to have them as compared to

children whose mothers had planned and wanted their pregnancies. Karon and Widener (1994) also cite research by Searles (1965) that suggests that families of those with schizophrenia have limited contact with other families or the community, suggesting that increased contact with other families leads to more opportunity for a family to correct their parenting and communication styles.

One of the newest, and less blaming and controversial, theories of the role of the family in schizophrenia is a reframed model focused not around whether parents provide flawed parenting, but whether the parenting provided is capable of dealing with the specific needs of a child with the genetic predisposition for schizophrenia (Gabbard, 1994). As described by Gabbard (1994) clinicians today are,

“less inclined to blame mothers for causing schizophrenia. Rather, a more balanced perspective is that a disturbed relationship results from an interaction between an infant with neurobiologically based impairments and a mother who may not be emotionally equipped to handle an unusual child...the notion of a mother-infant ‘fit’” (p. 191).

There is significant other research to suggest that children who will grow up to develop schizophrenia often show early signs of the illness (Walker & Lewine, 1990). These characteristics include hypersensitivity to stimulation, difficulty with attentions and concentration and possibly some difficulties with object relations (Gabbard, 1994).

Because of this, Robbins (1993) suggests that parents should not be seen as “schizophrenogenic” but rather in a lot of cases as “reparative” or as “intuitively gifted caregivers” (Robbins, 1993, p. 168). This is because, as described by Robbins (1993), “it is an extraordinary challenge and stress to have the responsibility, with little respite, for the rearing of a child who cannot tolerate stimulation, is averse to emotional involvement and to sustaining a relationships” (p.168). In essence, raising such a child may “stimulate

the mother's own vulnerabilities, psychological and constitutional" (p. 168).

While research into the characteristics of a child with vulnerabilities for schizophrenia does seem to focus blame on the child for bringing about whatever deficits may have occurred in parenting, this is an interesting perspective, especially as it relates to understanding why, in cases of monozygotic twins or in cases of genetic vulnerability, the "phenotypic vulnerability" is not activated (Robbins, 1993, p. 168). This theory, it should be noted, is remarkably similar to Winnicott's (1975) notion of the "good enough mother" and the infants "unspeakable anxiety", which he too thought might play a causal or protective role in the later development of psychosis (p. 127). Another study which supports further questioning of the mutual difficulty inherent in parenting and growing up with pre-schizophrenic vulnerabilities is a study by Cannon, Walsh, Hollis, Maresc, Taylor, Murray, & Jones, (2001). This research found that children later diagnosed with schizophrenia were 2.7 times more likely to have been institutionalized or put in children's homes during their early childhoods.

While a lot of the theories covered in this section could be perceived as very blaming and condemning of families of people with schizophrenia, and are difficult to determine as necessarily causative, there is some significant evidence to suggest that families could benefit, and relapse rates could improve, if therapy was provided that could work on communication patterns, affect regulation, as well as empathy and boundaries within families of those with schizophrenia. While family psychosocial interventions are suggested within treatment models like the PORT Report (Lehman, et al., 1998) it is unclear how effective such interventions may be in addressing the deeper communications patterns that exist. There is also a sense that if communication and

relapse rates are to improve with therapy, that the therapist themselves must have low EE (Karon & Widener, 1994), a significant finding in selecting what type of therapist is appropriate for the psychotherapy of schizophrenia.

Trauma and Stress

Many of the early theories of schizophrenia were related to trauma, stress, and anxiety mostly as they existed within early primary relationships (Freud, 1924; Jung, 1939; Sullivan, 1962; Fromm-Reichmann, 1950; Searles, 1965). Current research, (Read, 2005, 2005a, Read, Perry, Moskowitz, & Connolly, 2001; Read & Ross, 2003; Ford, 1998; Siebert, 1999), is less focused on these more abstract issues of early object relationships and instead aims to more specifically understand abuse and neglect as well as economic and social stressors as causative factors in schizophrenia.

While child abuse is seen as playing a significant causal role in many mental health disorders including anxiety, depression, post-traumatic stress disorder, anorexia, and substance abuse disorders, it is rarely discussed as having a causal role in schizophrenia (Read, Goodman, Morrison, Ross, & Aderhold, 2004). Despite this, even early research like the 30 year study done by Robins (1966) (as cited in Read, Goodman, Morrison, Ross, & Aderhold, 2004) found that of 500 children followed, 35 percent of those who later developed schizophrenia had been removed from their homes as children for neglect.

An extensive review of studies of abuse on inpatients units, where more than half of the population was diagnosed with psychosis (Read, Goodman, Morrison, Ross, & Aderhold, 2004), found that 55 percent of women and 24 percent of men had been “either

sexually or physically abused at some point in their lives” (p. 225). It is concluded within this study that, “both men and women in psychiatric hospitals are at least twice as likely as men and women in general to have suffered child abuse” (p. 226). This study also found very strong correlations between hallucinations and abuse, with those suffering both physical and sexual abuse as children far more likely (almost 71 percent) to have hallucinations. These findings correlate interestingly with research summarized by Fletcher (1996) who found that up to 25 percent of hostages exposed to torture, physical abuse, threat of death, and isolation or confinement have trauma related hallucinatory experiences. Incest has also been shown to dramatically increase the risk of later life hallucinations, (Ensink, 1992, as cited in Read, Perry, Moskowitz & Connolly, 2001).

As discussed, trauma theories of schizophrenia are mainly understood as fitting the stress category within the diathesis-stress or vulnerability-stress models of schizophrenia (Walker & Diforio, 1997). Read and Ross (2003) argue however that abuse should also be seen as a diathesis or increased vulnerability and thought of as “acquired vulnerability” (p. 250), meaning that for some people with schizophrenia,

“Adverse life events or significant losses and deprivations can not only ‘trigger’ schizophrenia symptoms but may also, if they occur early enough or are sufficiently severe, actually mold the neurodevelopmental abnormalities that underlie the heightened sensitivity to stressors so consistently found in adults diagnosed schizophrenic (Read & Ross, 2001, pp. 319-320).

According to Read, Perry, Moskowitz, and Connolly (2001), abuse actually can be proven to create the structural abnormalities found in the brains of people with schizophrenia leading the authors to suggest a traumagenic neurodevelopmental model for schizophrenia.

There has also been research done, beyond child abuse, into culturally and economically based stressful and traumatic factors that could influence the development of schizophrenia. These topics include racism, immigration, poverty, urbanicity, gender and homophobia (Read, Mosher, & Bentall, 2004). For example a British study by Harrison et al. (2001) found that children born into situations of deprivation were up to 8 times more likely to later develop schizophrenia than children without deprivation. A study of the high incidence of schizophrenia among British Afro-Caribbeans, ruled out genetic factors and suggested instead that either the diagnostic system itself was racist, or that social isolation, racist discrimination, or financial disadvantage created by economic racism influence the increased risk (Read, Mosher, & Bentall, 2004). Another study documented by the same authors, found that those who had experienced “severe racist verbal abuse in the past year were 2.9 times more likely than others to be experiencing psychotic symptoms” (p. 175).

Trauma and stress theories have strong implications for the role of psychotherapy in the treatment of schizophrenia. If it is indeed true that such a large percentage of those with schizophrenia have suffered abuse and trauma, then is it fair to conclude, as Read and Ross (2003) did, that the majority of such individuals “could benefit from being offered the full range of psychological and psycho-social treatment modalities described in the literature on psychological trauma”(p. 247)? This is also a pertinent question in light of the research by Lundy (1992) that the experience of having schizophrenia itself may be enough to create the symptoms of post-traumatic stress disorder.

The significant effects of trauma and family issues in schizophrenia, whether believed to be factors in relapse, causation, or a result of the illness, do have important

implications for the role of psychodynamic psychotherapy of schizophrenia. Primarily, these implications are that whether the biological model can adequately explain the etiology of schizophrenia, there are in many cases, it appears, significant other obstacles encountered by individuals with schizophrenia in their lives which can not be addressed by medications (Read & Ross, 2003). Whether these issues should be most effectively addressed by psychodynamic therapy or by psycho-educational, psychosocial and supportive treatments remains to be examined. In the next section I will review the current literature on the psychodynamic treatment of schizophrenia.

Treatment

Before the biological model rose to dominance in the latter half of the twentieth century, psychodynamic treatments, generally more strictly psychoanalytic in nature than current treatments, were the recommended treatment model for schizophrenia (Arieti, 1980, Hornstein, 2000; Searles, 1965; Arieti, 1955; Bullard, 1959; Fromm-Reichmann, 1950; Sullivan, 1962; Spotnitz, 1969; Karon & Vandenbos, 1981). Today there are a number of clinicians and researchers who still believe strongly that various psychodynamic methods have proven efficacy in the treatment of schizophrenia (Gottdiener & Haslam, 2002; Silver 1989, 2001, 2003; Silver & Larsen, 2003; Horowitz, 2004; Robbins, 1993; Berger, 2001). As summarized by Bachmann, Resch, and Mundt (2003), although “the classical psychoanalytic setting is contraindicated” it remains true that “psychotherapy is possible” (p. 159).

Early studies of the efficacy of psychodynamic psychotherapy for the treatment of schizophrenia like that done by May (1968) and Karon and Vandenbos (1981) achieved

conflicting results. May's study found that improvement rates for solely psychodynamic interventions were far inferior to treatment with medications and psychodynamic therapy, while Karon and Vandebos achieved directly opposite results, with those treated solely with medication showing the least improvement. Karon and Vandebos's found that after 20 months, patients in their psychotherapy study group had 31-51 percent fewer hospitalization days. Also of significance in Karon and Vandebos's research is that the patients shown to improve the most, as measured by thought disorder, were working with the two most experienced therapists in the study, a factor similarly emphasized as related to good clinical outcomes by Gottdiener (2006).

While Fenton (2000) summarizes all the early research on psychodynamic psychotherapy including studies done by McGlashan (1984, 1988) at Chestnut Lodge (the early home of Fromm-Reichmann, and H.S. Sullivan) by stating that, at least in these early results, "the majority of patients...remained seriously disabled by chronic schizophrenia" (p. 51), in more recent years studies have shown some more positive results.

One of the most extensive studies of psychodynamic psychotherapy for schizophrenia was conducted by Gottdiener and Haslam (2002). The study showed drastically different results than those found by Fenton. Their meta-analytic review of 37 studies, conducted with over 2642 patients, found that psychodynamic therapies, as well as supportive and cognitive behavioral interventions were "all associated with improvement in functioning" (p.163). However, according to the researchers, "the largest improvement rates were associated with psychodynamic and cognitive-behavioral therapies (p. 181). Previous studies have also found qualitative differences in the

recovery experiences of those provided with supportive versus psychodynamic treatments. A 2-year follow-up study conducted in the early 1980's (Gunderson, Frank, Katz, Vannicelli, Frosch, & Knapp, 1984) found that people with schizophrenia receiving supportive therapy at the two-year period showed "less recidivism and better role performance" while those receiving psychodynamic therapy "had greater improvements in "cognition and ego functioning" (Gabbard, 1994, p. 194).

Within the review by Gottdiener and Haslam (2002), psychodynamic treatments were shown to almost double improvement rates from 34 to 67 percent. While the quality of therapy was not addressed (i.e. the experience of the therapist, or the specific techniques utilized) there was evidence that people with schizophrenia did improve slightly more on an outpatient basis (a result which may be entirely dependent on the fact that those still hospitalized generally are in more acute periods of psychosis). Of note regarding the methodology of some of the studies reviewed by Gottdiener and Haslam (2002) is the fact that about half of the studies did not assign patients randomly to treatments, suggesting that there still could be some selection bias (specifically that those more capable of benefiting from, or more interested in, psychodynamic psychotherapy self-selected to be engaged in this form of treatment). Interestingly, a study by Malmberg and Fenton (2001) reviewing three of the studies included in Gottdiener and Haslam (2002) found that psychodynamic psychotherapy provided no additional recovery benefit for those on anti-psychotic medication.

In their extensive study, Gottdiener and Haslam (2002) also found that that the proportion of patients likely to improve from conjoint medication and therapy was equivalent to the number showing improvement from therapy alone. This suggested to

the researchers that “individual therapy alone might be a viable treatment option for some patients who do not improve from treatment with anti-psychotic medications, for some patients who refuse to take medications, or for patients who are treated by therapists that choose to use little or no adjunctive medication” (p. 181). Studies of those with schizophrenia not on medication is highly prohibited within the current treatment system, however research by Bola (2006a), Lehtinen, Aaltonen, Koffert, Rakkolainen, and Syvalahti (2000), and Irwin (2004), suggests that within the right therapeutic environment medications may be tapered, or for some might not be the appropriate first line of treatment (Alenen, 1997; Mosher, 1999; Podvoll, 2003). The promotion of such ideas and programs should be approached with great caution and education however, as the suffering of those given intensive psychotherapy and denied pharmacotherapy has been well documented in cases like Osheroff versus Chestnut Lodge (Klerman, 1990) and Reed’s (1977) *Anna*.

Research reviewed by Fonagy and Roth (2005), found that studies on the efficacy of psychodynamic psychotherapy tend to be largely negative. However, these researchers stress that very few rigorous studies of these interventions have been undertaken. This dearth of research may be in part due to the fact that many such studies, including those of supportive psychotherapy, have drop-out rates as high as 50 percent by the 6 month period. These drop-out rates, a figure supported by Gabbard (1994), are significant in suggesting that at very few points has real long-term psychodynamic psychotherapy ever been studied as a treatment for schizophrenia. Roth and Fonagy conclude that whether or not formal psychotherapy is “judged appropriate” (p. 292), there is evidence that the

development of a good therapeutic alliance “promotes better compliance with medications and may be a helpful adjunct to treatment” (p.292).

Long term follow-up studies by McGlashan (1984, 1988) of 163 people with schizophrenia previously hospitalized at Chestnut Lodge, found that one-third of these people had moderately to good recoveries. Of those who had recovered, McGlashan interestingly found two groups. Those who had integrated their illness into their identity, and those who had in some sense sealed over that period of their lives. According to a review by Gabbard (1994), these findings are significant in that they suggest that “patients who can integrate a psychotic experience into their life may benefit from exploratory work in the context of psychotherapy” (p. 196) while those who recover through sealing over the psychotic episode would probably not benefit from such therapies (Gabbard, 1994). These findings could also simply suggest that those more initially capable of insight and relationship, such as was required within therapy at Chestnut Lodge, were more apt to find recovery within this context.

While there is some evidence from very comprehensive studies like Gottdiener and Haslam (2002), and Karon and Vandenbos (1981) that psychodynamic psychotherapies can be effective for some, it is clear that far more research needs to be done on the topic. One difficult aspect of conducting this research may be the challenge of retaining participants in a study, as discussed, or defining techniques and interventions, (i.e. the fine differences between supportive, cognitive-behavioral and psychodynamic interventions). One example of this difficulty with definition is apparent in the PORT Report (Lehman et al 1998, 1998a), which recommends against psychodynamic psychotherapy because it utilizes regression. This is interesting because regression is a

far more strictly psychoanalytic concept and is not necessarily a central intervention of psychodynamic treatment. Regression is actually recommended against within most psychodynamically informed treatments (Ver Eecke, 2003). Another example of the conflict around definition in psychodynamic research and support for psychodynamic interventions is the evidence that cognitive behavioral therapies, currently a recommended intervention within the biological model (Turkington, Kingdon, & Weiden, 2006; Pfammatter, Junghan, & Brenner, 2006), actually share a number of techniques with psychodynamic interventions. The interplay of all of these techniques is described by Gottdiener and Haslam (2002),

“Cognitive behavioral and non-psychodynamic supportive therapies primarily consist of supportive interventions, but not exclusively. Some cognitive-behavioral treatments extensively use insight oriented interventions. Although psychodynamic therapies are generally thought to consist of insight-oriented interventions, they traditionally consist of supportive interventions in the early stages of treatment with severely disturbed patients” (p. 186).

The fact that there is so little research, and thus a minimal level of evidence either for or against psychodynamic treatments (Wasylenki, 1992), and that the definition of what psychodynamic treatments are remains unclear to many researchers, is significant because why then are psychodynamic interventions so categorically denounced both within the biological model and by some within the field of psychodynamic therapy (Torrey, 2001; Lehman et al, 1998; Willick, 2001)? A second important question is why there are not more studies which bridge the dichotomy between psychodynamic perspectives and biological perspective, researching how these treatment methods might work together? Alenen (1997) suggests that one of the greatest hurdles to research into psychodynamic treatments is that researchers are still clinging to the idea of

schizophrenia as one illness. According to Alenen, because schizophrenia will manifest uniquely in relationship to a person's psychological make-up, some people will be very appropriate for psychodynamic therapy and others not – leading research results to often be inconclusive or contradictory.

While quantitative research in support of psychodynamic treatment remains scarce, another source of evidence for the efficacy of psychodynamic interventions comes from the writing of those, not only early clinicians with some strong success like Sullivan (1956, 1953), Searles (1960, 1965) and Fromm-Reichmann (1950), but from current therapists (Karon, 2003; Robbins, 1992, 1993, Silver, 1989, 2001, 2003; Horowitz, 2002; Yip, 2002; Benedetti & Peciccia, 1998) and even from those who have received these services (A Recovering Patient, 1986; Greenberg, 1964).

Another reason, beyond the current limited research, for why psychodynamic therapies are not promoted for the treatment of schizophrenia is the huge power of the pharmaceutical industry. Some critics writing about the current state of the biomedical model in mental health believe that the emphasis on pharmacology, and the denouncement of psychodynamic interventions, is based on the profit interests of pharmaceutical companies (Moncrieff, 2003; Thomas, Bracken, Cutler, Hayward, May, & Yasmeeen, 2005; Bola, 2006). These critics suggest that research may be oriented towards proving biological causes and the success of medications because,

“It was also shown recently that 87% of authors of clinical practice guidelines had some interaction with the pharmaceutical industry, and 38% had served as consultants or employees of companies. ... This is a cause of concern since guidelines usually command professional respect and have a strong impact on practice” (Moncrieff, 2003, p. 3).

Because of these tendencies, Moncrieff concludes that,

“The hegemony of biological psychiatry that now exists stifles other approaches to understanding the complex behaviors that constitute psychiatric conditions. It elevates quantitative positivist research methods, borrowed from the natural sciences. This approach depends on the notion that psychiatric conditions can be conceptualized as discrete entities occurring in individuals, which can be defined independently of their social context. Other philosophical and sociological approaches that seek to understand the meaning of psychiatric disorders at both an individual and social level are relegated to the fringes of psychiatric academia” (p.5).

While it may be true that the large financial assets of pharmaceutical companies influence research, and over-emphasize the efficacy or role of psychopharmacology, the problem remains that there have been very limited studies of psychodynamic treatments (Wasylenki, 1992). Gunderson and Gabbard (1999), suggest some concrete steps to not only creating more research, but also making psychodynamic interventions more researchable. Their suggestions include:

“1) define the distinguishing features; 2) identify clear indications and contraindications; 3) systematically collect case histories of successfully treated mentally ill (diagnosable) patients; 4) increase vigilance (together with the patient) toward assessing progress in treatment” (p. 679)

Another important factor to be considered in discussion of psychodynamic outcome research is the fact that although psychopharmacology and other programs have brought great changes to schizophrenia treatment, a huge percentage of people are not recovering within this treatment model. This model is not adequately treating a large number of those with schizophrenia. As discussed by Silver and Larsen (2003),

“There is today no longer any doubt that most patients suffering from psychosis have great benefit from antipsychotic medication, especially of the ‘new’ or ‘atypical’ types with less unwanted side effects. There is however, also no doubt that many people who suffer from severe psychosis never recover; 15-20% do not respond well to medication and many suffer from long-lasting psychological and social handicaps caused by the disorder. These aspects of the persons illness

cannot be ignored and must be treated, even if evidence-based randomized studies do not prove that the outcome gets better” (p. 6).

While it is true that a large percentage of people with schizophrenia are not recovering within the current treatment system, and there is some evidence both from research and personal accounts as to the efficacy of psychodynamic interventions, it is still unclear exactly what psychodynamic interventions in schizophrenia would look like, how they would be structured and how such services would be delivered.

Current work within the field of psychodynamic therapy of schizophrenia is widely varied in style. As discussed by Gabbard (1994), “There is no such thing as *the* treatment of schizophrenia. All therapeutic interventions must be tailored to the unique needs of the individual patient. Schizophrenia is a heterogeneous illness with protean clinical manifestations” (p. 184). According to Robbins (2002) most therapists, “have already adopted some version of the interactive, multisystemic model of illness and treatment...and as a consequence view mental illness as a complex process with organic, psychological, interpersonal, and sociocultural aspects” (p. 311).

Arieti (as cited in Berger, 2001) feels that the work of psychodynamic therapy begins once medication has provided symptomatic treatment, a view seconded by Gabbard (1994). According to Gabbard, even if biological models of etiology and treatment are effective, they do not address the greater needs of the complex person facing the illness.

“None of the findings of biological research attenuates the impact of one irreducible fact – schizophrenia is an illness that happens to a person with a unique psychological makeup. Even if genetic factors accounted for 100% of the etiology of schizophrenia, clinicians would still be faced with a dynamically complex individual reacting to a profoundly disturbing illness. Sophisticated

psychodynamic approaches to the management of the schizophrenic patient will always be vital components of the clinician's treatment armamentarium" (p. 184).

According to Karon and Teixeira (1995) the goals of psychodynamic treatment in schizophrenia are those of therapy with anyone "namely, to live a more fully human life" (p.98), however more specific goals should include the following: the ability to take care of one's self; ability to work; social adjustment; sexual adjustment; absence of hallucinations and delusion; becoming freer from anxiety and depression; amount of affect; variety and spontaneity of affect; satisfaction with life and self; achievement of capabilities; and benign versus malignant effect on others.

Work with those diagnosed with schizophrenia, as discussed by Karon and Teixeira (1995) should be focused around the therapeutic alliance, an alliance that can be formed with the "healthy part of the patient." This idea of a healthy core, an underlying health within a person with schizophrenia, was also written about by Bleuler (as cited in Harding, 2003), who learned from his clinical research that,

"The inner life of the schizophrenic is never 'burnt out' it always continues on its way. When ceaseless attempts are made to establish contact with him as a normal person, and he is not left to stand aside like an outsider, a communal relationship is established that means a great deal to both the patient and the doctor" (p. 10).

Bleuler continues on to say that people with schizophrenia, despite appearances at times, "have not lost touch with healthy psychic life...in their case, healthy perception, memory, recall, judgment, and feeling are merely concealed behind their pathological behavior" (p. 10). Because of these layers of concealment, symptom, and defense, according to the Karon and Teixeira (1995), the initial work of creating a therapeutic alliance, of joining with this healthy part of the client, "may be the center of the work for a long time" (p.101). Of significance due to the seeming emphasis on relationship proposed within

psychodynamic theory is research conducted by Marley (1998), that found that those diagnosed with schizophrenia do feel that their symptom fluctuations are greatly related to interpersonal interaction and stress in interpersonal relationships, even with staff in mental health facilities.

According to Benedetti and Peciccia (1998), the patient therapist relationship in treating schizophrenia naturally achieves the purpose of creating a self-object for the client through the transference relationship. This goal is achieved by the therapist's empathy that creates an experience of duality for the client, a duality in which the patient's overwhelming feelings of "symbiosis with the world" (p.169) become a healing "therapeutic symbiosis" (p. 170). As described by Benedetti and Peciccia (1998), the difference between pathological symbiosis, which is the basis of the psychosis, and therapeutic symbiosis, "is that the 'self object', or the 'therapeutic mirror image' confronts the patient with a therapeutic self, which is similar to him, in order to make it more positive, enrich it and make it independent" (p. 170). This treatment method is similar the intersubjective and phenomenological model suggested by Stanghellini and Lysaker (2007) in which, "the therapeutic process consists of assisting persons to move towards recovery by providing an intersubjective space where they can evolve the first-person perspective of themselves and the second-person perspective when encountering others" (p. 163).

Arieti (in Berger, 2001) like Karon and Teixeira (1995), emphasizes that the process of relationship building, a most central tenet of treatment, may also be some of the most difficult work due to the common schizophrenia symptoms of either withdrawal or paranoia.

“When it comes to other parts of treatment, let’s say, to the attack, psychologically, of the individual symptoms of the patient or his psychological structure or an understanding of his psychodynamic history – for that we already have some scientific ground. But when it comes to establishing relatedness, the therapist must be more of an artist than a scientist” (p. 7).

By artist Arieti means that the clinician’s role is not to ask questions to probe or interpret, but to intuitively give the client, “friendship, our interest, our desire to share in his anxiety” (Berger, 2001, p. 9). Another fundamental aspect of the work of relationship-building identified by Arieti (1955) and Berger (2001), is the understanding that the client is not avoiding relationship out of hostility but rather out of profound feelings of projection and devaluation of himself.

Arieti (in Berger, 2001), suggests that the difficulty encountered by the therapist in creating a relationship with a client with schizophrenia may be similar to what was experienced by the person’s parents. He says that it is this difficulty that is in part what gave birth to the idea of the schizophrenogenic mother. Those with schizophrenia, in extreme sensitivity as children, “make a whole out of the negative parts of mother” and therapists have mistakenly taken this to mean that the mother was destructive rather than helping the client integrate good and bad parts of the mother and thus begin to heal in interpersonal relationships and “re-establish object relations” (Berger, 2001, p. 14).

The fact that psychodynamic treatments do not promote the idea of schizophrenogenic parents is one of a number of seeming misconceptions about this approach. Another misconception is that, despite psychodynamic psychotherapy’s image as promoting regression and insight, Karon, credited with writing one of the most definitive books on the psychotherapy of schizophrenia (See Karon & Vandenbos, 1981; Karon, 2003), does not promote immediate insight or interpretation. Karon suggests

instead that “insight is only tolerable and attainable within the security of a warm, strong, dependable, and safe relationship with the therapist” (Karon & Teixeira, 1995, p. 101). Within this relationship, according to Karon and Teixeira (1995), transference and eventually insight will occur. And, most importantly, the profound anxiety experienced within the psychosis will begin to abate. According to the authors, “every therapeutic interaction is aimed at the reduction of the terror to manageable proportions. Behind the inaccurate thinking (thought disorder), distorted perceptions, hallucinations, withdrawal, and inappropriate reactions to other people always lies terror” (p. 103).

It is clear that many any of the interventions suggested by the psychodynamically oriented literature promote a style that incorporates a large number of interventions that are technically more supportive and or cognitive behavioral in nature (Shean, 2004). The interconnected relationship between psychodynamic and supportive interventions is one reason why psychodynamic clinicians responded strongly (Gottdiener & Haslam, 2003; Silver & Larsen, 2003; Ver Eecke, 2003) to the promotion of supportive therapy and condemnation of psychodynamic interventions within the Port Report (Lehman et al, 1998). The report, in a sense, implied that these two were entirely distinct. According to Silver (2001)

The Port Study’s recommendations were based on a literature review by Scott and Dixon (1995) which states that “individual psychotherapy historically connotes either dynamically oriented psychotherapy, which typically seeks to increase insight, or supportive psychotherapy, which typically seeks to build ego strength” (as cited in Silver, 2001, p. 26).

But as argued by Silver (2001) “How can ones ego become stronger without an accompanying increase in awareness of one’s patterns of interaction and of reaction, of

one's strengths and weakness, and one's ways of coping with anxiety?" (Silver, 2001,p. 26).

Contemporary psychodynamic theories reviewed by Shean (2004) also showed a de-emphasis on interpretation and a pragmatic and supportive approach to symptom reduction. According to Shean, initial interventions within this modality are focused on "limiting regression, reducing symptoms, strengthening defenses, sealing over psychotic experiences, and encouraging the development of stable, trusting object relationships between patient and staff" (p. 229). In outpatient work, psychodynamic interventions should be focused more on "exploration of previous conflicts and the complications of the patient's life brought about by the psychosis. Feelings of guilt and inner badness, experiences of inner emptiness and despair, and problems with good and bad object representations" (Shean, 2004, p. 229). Shean does report that there is some evidence that more insight oriented or interpretative work may be more effective with certain populations of people diagnosed with schizophrenia. Primarily those with whom psychodynamic interventions are effective are "young, intelligent" and had "good premorbid functioning, with a history of achievement at work, as well as some degree of success in interpersonal relationships" (p. 230). Barron and Sands (1996) found slightly different results, suggesting that treatment resistant patients with primarily negative signs of schizophrenia tend to engage better and improve more within psychodynamic treatment.

Gabbard, (1994) a psychiatrist and director at the Menninger Clinic, also supports psychodynamic interventions similar to the treatment strategies discussed by Shean (2004) and Karon and Teixeira (1995). According to Gabbard, the division between

expressive and supportive therapies should become more fluid in the treatment of schizophrenia, and treatment should be organized along 9 main principles. These include:

“1) The main focus should be on building relationship; 2) The therapist must maintain a flexible stance regarding the mode and content of therapy; 3) For psychotherapy to proceed, therapists and patients must find and maintain an optimal distance; 4) The therapist must create a holding environment; 5) The therapist must serve as a ‘container’ for the patient; 6) The therapist must serve as an auxiliary ego for the patient; 7) The therapist must be genuine and open with the patient; 8) The therapist should postpone interpretation until the therapeutic alliance is solid” (p.197).

The ninth criterion included by Gabbard (1994) is that “the therapist must maintain respect for the patient’s need to be ill” (p. 197). The most psychoanalytic of his techniques, this appears to refer to the idea that the symptoms of the illness of schizophrenia are a solution to intrapsychic conflict (see also Karon & Teixeira, 1995; Berger, 2001). While this idea seems, in its essence, to deny the large established role of genetic factors (Berger, 2001) in schizophrenia and imply a more strictly psychogenic etiology, this is dependent upon interpretation. This principle might also be utilized clinically as a means of understanding how much difficulty the process of recovering poses at times, for example the deafening silence experienced by some when hallucinations stop after many years (Steele & Berman, 2001)

A psychodynamic treatment style outlined in a case study by Yip (2002) also stresses the essential importance of interpersonal relationships in the psychodynamic approach to treating schizophrenia, an approach highly influenced by the theories of Sullivan (1953). Sullivan’s model promoted very normalizing view of schizophrenia that explained that people with schizophrenia

“manifest more or less the same kinds of dynamisms as other persons. In other words, the psychotic experience of persons with schizophrenia may be similar to inner experiences of normal persons. They may represent the suppressed feelings, needs, and desires of an individual facing external threats and hostilities” (Yip, 2002).

From her work using Sullivan’s model, Yip concludes that there are elements of Sullivan’s approach that are critically useful to a strengths-based psychodynamic treatment of schizophrenia. These include mainly his understanding of the role of hallucinations and delusions which he thought should be de-pathologized, be understood to have meaning within the life of the person, and should be understood to be a reaction or adjustment to external threats or stress – making the clinicians job in part to help reduce stress, improve coping, and remove any external threats.

The idea of the inherent meaning of hallucinations and delusion is an important aspect of the psychodynamic treatment of schizophrenia and is a place, eventually, where interpretation may have value. According to Karon and Teixeira (1995), all of the symptoms of schizophrenia do actually have meaning and are embedded in the history of the client and are the “best coping mechanisms available to him or her at the pretreatment stage” (p. 105). In regards to interpretation, the authors suggest that the only rule that should be followed is that “the therapist interprets what the patient can make use of at the time. Whether to interpret early or late, deep or shallow, defense or impulse, is always a clinical judgment that may be in error and may require constant revision” (p. 108).

The value of meaning making in hallucinations and delusions, according to Selzer and Carsky (1990), is that it is through this that the therapist and client can find a common ground, similar to the self-object described by Benedetti and Peciccia (1998). It is this common ground on which the client can gain insight about their illness as well as

move beyond concrete thinking to more symbolic understanding of themselves within their illness. According to Selzer and Carsky (1990), finding this common ground, locating the patients “fundamental orientation”, requires “that the therapist pay particular attention to the content of the patient’s belief systems and, without necessarily agreeing with them, employ this understanding to develop a shared language” (p. 507). This joining process allows the therapist to set out on the journey with the person with schizophrenia without the “classic imperative to talk ‘normal’” (p. 507). The importance of listening to and trying to comprehend the content of psychosis, especially the meaning constructed by clients, is also emphasized by Werbart and Levander (2005) who report a “clinically founded conviction that the subjective meaning constructions of psychotic patients constitute an important source of knowledge and a potential contribution to the process of recovery” (p.103).

There are a number of guiding principals for psychodynamic therapy with schizophrenia which can be gleaned from a review of the literature. Major themes include relationship building, belief in the humanity and healthy core of the client, respect for subjective experience, and transference as a means of regaining interpersonal skills and exploring and mirroring identity. While there is some evidence to support the efficacy of such interventions (Gottdiener & Haslam, 2001), the question remains as to what therapists should engage in this work, how such services might be delivered, and why there is currently very little (at least identified as such) psychodynamic psychotherapy being practiced with schizophrenia.

There is some conflict in the research suggesting what characteristics are most important in therapists treating schizophrenia. Karon and Teixeira (1995) emphasize that

most importantly, therapists must be consciously interested in being helpful to those with schizophrenia. This criterion is supported by research that suggests that often in cases where psychotherapy is ineffective as a treatment for schizophrenia, the therapists did not volunteer willingly for the work. Karon and Teixeira (1995) also stress the importance of a therapist's familiarity with the work of those who pioneered psychodynamic interventions with schizophrenia as well as a strong supervisory relationship. Strong training and a "sound psychodynamic understanding" are also cited as important therapist characteristics by Barron and Sands (1996, p. 113), who say that it is "neophytes" ambivalent practitioners" and overworked "case managers" who have "given therapy a bad name" (p. 113). Gottdiener (2006) and Karon and Vandenbos (1981) also both found that effectiveness in psychodynamic interventions was correlative with level of the therapist's experience.

Breggin (1994), on the other hand, does not feel that there is much experience necessary to provide a strong therapeutic relationship for those with schizophrenia. According to Breggin, from his many years of experience on inpatient units, clients have repeatedly expressed that simply talking, to anyone, and being believed in and given hope is the most important part of treatment. Because of this feedback Breggin pioneered a program that brought college students in psychiatric units to work as aides. This program, according to Breggin, resulted in helping "nearly all their patients leave the hospital" (p. 380). The significance of hope on the part of the therapist is also supported by Itzhaky and Chopra (2005) and Desisto et al (1995), who found in their extensive research on recovery that many who have achieved significant recovery reported that the

most essential aspect of their treatment was the fact that someone had told them that they could recover (Desisto, et al, 1995).

After reviewing the literature on etiology, treatments, and therapist characteristics the fact remains that psychodynamic interventions are currently not widely offered for those with schizophrenia. While intensive, private treatment which includes psychodynamic, biomedical, and psychosocial interventions is available at some programs including the Austen Riggs hospital, and the Windhorse communities, as well as internationally in Finland with the Open Dialogue model (Alenen, 1997; Podvoll, 2003) these options are not widely available within the current treatment system, as discussed in Chapter III. Beyond the currently limited research proving the efficacy of these treatments, the lack of access to psychodynamic treatment might also be due to what is perceived as the prohibitive cost of providing such therapies (Barron & Sands, 1996; Lazar, 1997). As described by Luhrmann (2000),

“I saw unmistakably, in my time at the hospitals and in my discussions with staff and patients, that psychotherapy had been muted under the impact of managed care policies. This was happening to meet the concerns of insurers. It was not because the new developments in psychopharmacology and biological psychiatry had led psychiatrists to that the talk-oriented approach is not important but because psychotherapy just didn’t accommodate as well to the short-term approach insurance companies understandably favor” (Luhrmann, 2000, p. 249)

There is some evidence however that the treatment alliance developed within therapy can have a profound effect on medication compliance (Fonagy & Roth, 2005), a factor which, according to Abramson (2001), can actually serve to drastically reduce the cost of treatment through decreasing hospitalization. This view is supported by evidence from Barber (2008) and Barron and Sands (1996), who ask if the costs of psychodynamic therapies could really ever equal the billions of dollars spent in the United States on

emergency mental health services as a result of non-compliance with medication, not to even mention such factors as work lost by those with mental illness?

Conclusion

Within this chapter, I reviewed literature on the psychogenic origins of schizophrenia as well as the efficacy of psychodynamic treatments and the models proposed for such interventions. There is strong evidence that a large majority of those with schizophrenia have suffered extensive trauma and abuse, including, for some, living within family systems in which there has been a lot of conflict. It remains to be fully documented however, as to how much this trauma may play a causal role in the illness or be a side-effect of living with the illness or vulnerability for the illness. While the research into the efficacy of psychodynamic interventions remains minimal, this is in part due to the difficulty with categorizing interventions and documenting change within such treatments. What evidence that does exist for psychodynamic treatments suggests that the therapeutic alliance, support, reality testing, and valuing of subjective experience within psychodynamic interventions can provide strong positive changes within the illness. There is also evidence, from within the descriptions of psychodynamic interventions, that such interventions remain fluid in nature utilizing numerous approaches including cognitive behavioral and supportive techniques as needed. Also contained within the literature is the understanding that it is not necessarily true that those who practice psychodynamic interventions promote a psychogenic model of schizophrenia etiology, in fact a majority accept the genetic basis for the illness. This might be an important factor in decreasing the controversy of these treatments and allow

them once more gain some wide-spread utility. In the following concluding chapter, Chapter VI, I will summarize the literature that has been reviewed throughout this study, discuss possibilities for increased synthesis and dialogue between the biological and psychodynamic perspectives, as well as suggest topics for further research.

CHAPTER VI

DISCUSSION

The purpose of this study has been to explore the question: What is the role of psychodynamic psychotherapy in the treatment of schizophrenia in an era where biologically oriented psychopharmacological interventions are the dominant treatment model? In this final chapter I provide an analysis of my findings, seeking elements of synthesis between the perspectives reviewed, as well as exploring the limitations of the study. I conclude with recommendations for practice and areas for continued research.

Summary

The previous five chapters have addressed the research question on the role of psychodynamic psychotherapy through introducing the topic, delineating methodology and terminology, summarizing the epidemiology of schizophrenia, outlining the current treatment system, and reviewing the literature from both biological and psychodynamic perspectives on the origins and treatment of schizophrenia.

In Chapter III I introduced the phenomenon being studied, namely schizophrenia and schizophrenia treatment. Research reviewed within this chapter showed that schizophrenia appears to be present at different rates both between countries as well as between urban and rural areas (Shean, 2004). There is also evidence that there is variation in recovery from schizophrenia, with significant differences on an international scale

(Jablensky, 1992; Whitaker, 2002) as well as a greater possibility for recovery from the illness than previously thought by researchers (Harding, 2003)

Chapter III also gave a brief history of how schizophrenia has been understood and treated over the past few centuries. This history showed a longstanding division between biological and psychodynamic approaches to the illness (Whitaker, 2002; Luhrmann, 2000) and illustrated that it is only in the past 50 years that psychodynamic treatments have fallen out of favor and biological interventions have become the dominant model for treating schizophrenia (Luhrmann, 2000). While the change to reliance on antipsychotic medications as the primary form of treatment for schizophrenia was undoubtedly beneficial to many, the section of Chapter III documenting the current treatment system shows that despite this increased reliance, and continued research, the mental health system continues to fail to provide for or promote recovery in a huge number of those with schizophrenia in this country. Few people with schizophrenia have access to psychiatrists, and an alarming number remain homeless or in prison, spending their lives revolving between hospitals, shelters, and jails (Torrey, 2001).

Chapter IV reviewed the literature from the biological perspective on the etiology and treatment of schizophrenia. The literature showed that despite continued research into understanding the origins of schizophrenia from genetic, biochemical, neurobiological, and viral theories, none of this research has yet to fully explain the cause or causes of schizophrenia (Siebert, 1999). The literature also showed that there is still a distinct possibility for a role of environmental factors, and that most biological researchers accept a schizophrenia etiology model like the diathesis-stress or vulnerability-stress models (Yak, Bentley, & Hargrove, 2003).

Research on the biological model of schizophrenia treatment showed that antipsychotic medications are the essential treatment tool within this model, treatment is focused on symptom reduction, and psychodynamic interventions are widely discouraged. While some feel that the biological model is a highly effective treatment model (Lieberman, et al., 2005; Lehman, 1998; Torrey, 2001), others continue to question its efficacy, raising concerns about side-effects, poor recovery rates, and the loss of human connection within this treatment model (Cohen, 2002). Others stress that there needs to be an idea of recovery that goes beyond the symptom reduction provided by medications (Whitaker, 2002; Cohen, 2002; Luhrmann, 2000).

Chapter V reviewed the literature from the psychodynamic perspective on schizophrenia etiology and treatment. While psychogenic theories of schizophrenia remain highly controversial, research shows that a large percentage of those with schizophrenia have experienced trauma in their lives and 40 percent of those with schizophrenia have endured childhood sexual abuse alone (Read, 2004; Silver, 2003). Research into the effectiveness of psychodynamic interventions remains scarce, and results appear mixed, however some studies suggest that psychodynamic interventions might very well contribute to positive recovery from schizophrenia (Karon & Vandenbos, 1981). The literature reviewed in Chapter V on technique in psychodynamic treatment shows that the most commonly utilized interventions are a focus on the therapeutic alliance or relationship, support, reality testing, the subjective experience of the client, stabilization of ego-boundaries and integration of psychotic experience (Bachmann, Resch, & Mundt, 2003; Silver, 2003; Karon & Vandenbos, 1981).

Analysis

A summary of the research covered within the various chapters of this study shows that while the biological perspective, and biomedical treatment model, currently dominates how schizophrenia is understood and treated in the United States, this model is not meeting the recovery needs of too many people with the illness. Also apparent from the research is that a huge amount remains unknown about the epidemiology, etiology, and treatment of schizophrenia. While the biological literature suggests that there is no role for psychodynamic theory or therapy in the schizophrenia treatment system, the psychodynamic literature continues to suggest ways in which understandings and interventions from this perspective might be useful. While it is clear from the research there are central elements of the biologically-based treatment model which are not working, and there is some research to suggest that psychodynamic interventions may prove useful, the question of the exact role of psychodynamic therapy in the treatment of schizophrenia remains unclear. In this section I will analyze my findings in further detail in an effort to solidify the answer to this question, addressing first my findings on etiology and then on treatment.

Etiology

First, while there is some significant evidence suggesting a genetic role in schizophrenia etiology, I expected to find much stronger evidence as to what causes schizophrenia. Despite the wide-spread popular emphasis on schizophrenia as a biological disease, or a chemical imbalance, there is actually no comprehensive scientific theory of schizophrenia origin. The biological perspective, within most of the literature,

supports a vulnerability or diathesis-stress model, a model which sees schizophrenia as caused by some combination of environmental and genetic factors. Surprisingly, these conclusions from the biological perspective coincide with my findings on current psychogenic theories of schizophrenia from the psychodynamic perspective. No longer blaming a “schizophrenogenic mother” psychogenic theories accept genetic factors but continue to try to define how stress, environment, and trauma serve to activate or exacerbate the illness (Read, Mosher, & Bentall, 2004). The discovery that both biological and psychodynamic researchers accept a relatively similar model of schizophrenia etiology is significant in that it shows that these perspectives might not be as dissimilar as they are often presented as being (Luhmann, 2000).

The Influence of Theories of Etiology on Treatment

While the research shows that the most accurate model of schizophrenia etiology actually combines environmental and genetic factors, a more strictly biological perspective on schizophrenia etiology continues to be the more widely accepted and publicized model. One reason for the continued emphasis on the biological factors of schizophrenia etiology both in research and consumer publications is that it is perceived to be significantly less stigmatizing than psychogenic models. I am concerned however, after reviewing the research, that biological theories have become so heavily promoted that they are actually very stigmatizing as well. Chronicity and psychopharmacology are emphasized within the biological perspective to the extent that it feels as if people are no longer seen as capable of recovery or worthy of interaction or dialogue. The focus within

the biological model is on symptom reduction rather than the promotion of recovery on a social, occupational, and psychic level. As described by Robbins, (2002)

“The more schizophrenics come to be viewed as organically impaired individuals whose mental life is a kind of brainstorm of meaningless fragments, and who are to be symptomatically regulated with medications, the less likely it is that anyone will take the trouble to get to know them in depth” (p. 312).

This fear is echoed by Silver (2000) who discusses the social worker being reprimanded by the psychiatrist who asks, “‘Why are you talking with this man? I’m giving him medications.’ This doctor seemed to say that the patient is not worth talking to, is somehow pre-verbal or perhaps sub-human” (p. 25).

While much remains unknown about the effects of psychodynamic psychotherapeutic work with schizophrenia, this should not mean that human interaction and clinical relationships with those with schizophrenia should be totally neglected. After reviewing the literature, I feel the risk for this has greatly increased within the biological model of treatment however – as if all of the elements of psychodynamic therapy, even the less dynamic techniques, were proverbially thrown out with the bathwater as schizophrenia came to be seen as a biological illness.

One way the psychodynamic perspective may regain a more significant role, as well as credibility within schizophrenia treatment, comes from the findings that a huge percentage of those with schizophrenia who have also suffered trauma. These findings suggests that a psychodynamic perspective might be an essential tool in treatment, as this perspectives can more fully comprehend how stress and trauma and the environment work within a person’s life and interact with their illness.

Ultimately it seems, that no matter what etiological discoveries are made, and what they tell us about schizophrenia, they will never explain who a person is, what their unique history is, what traumas and stresses they have experienced, and how all of these factors interact in their individual experience of the illness. This is described by Gabbard (1994),

“None of the findings of biological research attenuates the impact of one irreducible fact – schizophrenia is an illness that happens to a person with a unique psychological makeup. Even if genetic factors accounted for 100% of the etiology of schizophrenia, clinicians would still be faced with a dynamically complex individual reacting to a profoundly disturbing illness” (p. 184).

Treatment

It is clear that a lot remains to be understood about the etiology of schizophrenia. Similarly, extensive further research needs to be undertaken around schizophrenia treatment. While antipsychotic medications do appear from the literature to be effective for many in reducing symptoms, studies on recovery suggest that these medications are only a part of the puzzle. The literature showed that side-effects and poor medication compliance greatly reduce what efficacy can be achieved with these medications, and the lowest estimates show that 15-20% of those with psychosis do not respond at all or respond very poorly to neuroleptics (Silver & Larsen, 2003). Vocational training and supportive therapy, as recommended within the biological literature, may help greatly to supplement this treatment, but the fact remains that people with schizophrenia have a greater possibility of becoming chronically ill in this country than in many other countries internationally.

There is little to no mention, within the biological literature, on a role for psychodynamic therapy. Most of what I read flatly dismissed such interventions but gave little evidence as to why they should not be utilized. My research suggests that the dismissal of psychodynamic interventions as a supplementary treatment for schizophrenia actually seems to be more rooted in linguistics and confusion around technique than it is in empirical research on the ineffectiveness or harmfulness of these interventions (Wasylenki, 1992). As described by Silver (2003) psychodynamic interventions were first “attacked for being powerless...now we have graduated to being powerfully destructive, this without any supporting data” (p. 331). There is a dearth of research on psychodynamic interventions for a number of reasons, as described in Chapter V, but these are summarized by Silver (2003), as being based in the fact that you cannot conduct “double blind studies on the human relationship” (p. 331).

The PORT research by Lehman et al (1998) is one example of how, with little supporting data and a lot of confusion, there is strong opposition to the integration of psychodynamic therapy as a supplementary treatment for schizophrenia. The report recommends that “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 those with schizophrenia” (p.7). The reports continues on to say that “psychotherapy aimed at understanding unconscious drives or getting at the psychological roots of schizophrenia is never appropriate” (p. 7) and also states that family therapy “based on the premise that family dysfunction is the etiology of the patient’s schizophrenic disorder” (p.8) should never be used.

These statements within the PORT research are illustrative of a lot of the confusion and misinformation that exists about psychodynamic treatment. Such statements, within one of the most recent and comprehensive publications from the biological perspective, suggest that there continues to be extensive misinformation about psychodynamic approaches both in the distinction between psychodynamic approaches to neurosis and psychosis, as well as differences between psychodynamic approaches and more classically psychoanalytic approaches. First, as we have seen there is little basis for assuming that psychodynamic approaches inherently blame families for schizophrenia. Secondly, none of the psychodynamic literature on schizophrenia promotes regression, and what discussion of transference and interpretation that might arise is suggested used with great caution and care. Thirdly, the fact that psychodynamic psychotherapy is only mentioned within Lehman et al (1998) as something that is a source of danger or shame is significant in and of itself. To write the psychodynamic perspective off so entirely, without mention of elements of this model that might have utility, displays a stronger attachment to dichotomies and simplification than it does to true scientific evidence or the promotion of healing (Ver Eecke, 2003).

As discussed, the psychodynamic literature promotes a far more supportive, yet dynamically informed approach to treatment than seemingly interpreted by the biological community. Psychodynamic clinicians working with schizophrenia are cautioned to “maintain a flexible stance regarding the mode and content of therapy” (Gabbard, 1994, p. 199). Because of this flexibility, the psychodynamic literature also showed that cognitive behavioral and supportive techniques are often used by psychodynamic clinicians in their work with those with schizophrenia, a notable finding since these

interventions are more widely accepted treatments within publications like Lehman et al. (1998) while psychodynamic interventions are not.

While there may be evidence that I have not found in my research that shows more definitively that psychodynamic interventions should not be utilized, within this study it appears possible that the dismissal of this perspective is more a matter of misunderstanding of the theoretical position, or techniques as discussed above, and even may be influenced by economics. The literature shows that the fall in popularity of psychodynamic interventions with schizophrenia was not necessarily a change brought about due to any proven ineffectiveness of these interventions. While continued research had found antipsychotic medications to be very effective in quieting psychotic symptoms, they also simply provide the fastest and cheapest treatment (Luhmann, 2000). This is important because as pharmaceutical companies have grown in power, their influence on research and treatments choices has grown concurrently. As discussed by Bola (2006),

“Close financial relations between academic psychiatry and the pharmaceutical industry have created growing concerns for the quality of scientific knowledge. The recent estimate that industry-funded studies are 3.6 times more likely to reach pro-industry conclusions than are independent studies addressing the same questions should be of great concern to social work and other professions practicing in the mental health field” (p. 263).

The Role of Psychodynamic Psychotherapy

I have found that a lot of the basis for recommending against psychodynamic psychotherapy comes from a lack of research, confusion, and misinformation about the practice and fear surrounding psychogenic theories of schizophrenia etiology. While this may be the case, this does not mean that a clear-cut role or place for psychodynamic

treatment exists within mental health treatment, that any or all psychodynamic techniques may eventually prove efficacious in providing supplementary treatment to schizophrenia, or that the support or money exists within a biologically dominated managed care system for psychodynamic work to be done. While the recommended psychodynamic interventions within the literature from the psychodynamic perspective are far more varied, supportive, and supplementary than seemingly perceived by the biological perspective, they are in their very nature difficult to research and do not fit into the evidence-based criteria that increasingly exists at the core of therapies promoted within the current mental health model.

One way of defining the role of psychodynamic psychotherapy in treating schizophrenia comes from trying to imagine a treatment system without these interventions. This question is explored by Neugeboren (2001) who describes asking a prominent psychopharmacologist what he believed would make the greatest difference in the lives of people with major mental illness in the next half-century. The psychopharmacologist responded, “Clozaril without side effects” (p. 304). However, Neugeboren responds in turn by asking,

“But if there are no systems in the world that are organized so as to allow an individual taking Clozaril to make use of its beneficial effects in a sustained way, what then? If, like my brother, individuals with long-term histories of madness and institutionalization – along with homelessness, poverty, drug abuse, prostitution, incarcerations, and violent acts perpetrated and/or endured – suddenly, through the agency of medication, come back literally and clearly to their senses, what do they do then? If medications suddenly and miraculously alleviate symptoms – and even their *causes* – what do these individuals do in the next hour, day, month, or year of their lives? What do they do, for starters, with regard to the basics of life – food, clothing, shelter, education, finances, and employment? What do they do with their desires for friendship and love? What do they do with their fears and their sorrows?” (p. 304).

This quote in itself summarizes one place that psychodynamic treatment holds. While psychopharmacology can reduce symptoms, psychosocial interventions may help with housing, finances, and occupational training, and the amazing power of consumer-led treatment will continue to play an essential role in social recovery and the decline of stigma (Neugeboren, 2001), psychodynamic interventions continue to hold a specific place, and play a distinctive role in providing a place for fear, sorrow, hope, spirituality, love and acceptance during the recovery process (Gabbard, 1994).

Beyond its utility as a place for relationship-building, meaning-making, and valuing of subjective experience, the psychodynamic perspective should continue to play a role in schizophrenia treatment because it is only this perspective that can comprehend the way in which schizophrenia interacts with and affects the individual history and emotional, spiritual, physical make-up of the individual human being. As described by Silver and Larsen (2003),

“Psychosis and schizophrenia specifically, attacks both the functioning of thinking, feeling, and reasoning, but also the person’s conception of self. Psychosis is not something you have in addition to being you, it changes your experience of being all together. I do not mean to say that people are their psychotic experiences, but their sense of being is very often dramatically changed due to psychotic experience. These are profound psychological effects of the disorders and need to be addressed in therapy” (p. 6).

It is the psychodynamic perspective that can comprehend the place between mind and brain, how the illness plays out upon the unique landscape of the individual person’s psychological make-up. This is explained by Gaetano Benedetti and Maurizio Peciccia (as cited in Silver, 2003) who say that, “Biological research into ...schizophrenia has, over the last twenty years, become more and more fascinating; but psychodynamic reflection on psychopathology remains indispensable because only this forms the

obligatory connection between the brain disturbance and the human condition of the patient” (p. 30). Silver (2003) continues this thought by saying,

“If we lose a humanistic striving to understand each human being who comes to us for psychological assistance, we forego the essence of professional availability. If we objectify those who are most alienated from society, we relinquish our responsibility to build an I-Thou relationship, and encourage in its place an I-it, dehumanizing one” (p. 30).

To Luhrmann (2000), the question of surrendering the psychodynamic perspective, and giving up this unique possibility for human contact and relationship in schizophrenia treatment, becomes a moral question. She describes a conversation with a psychiatric administrator who told her that, “you can handle schizophrenics by putting fifty of them in a room with beds, a few nurses, and lots of Thorazine” (p. 292).

According to Luhrmann however,

“We need...to make a moral decision, which is whether to understand such people only as the detritus of a broken brain or also as people whose suffering implication us, whose struggles are resonant with our struggles, who are located in a particular culture, and whose complexity and depth demand that we see their suffering” (p. 293).

Psychodynamic clinicians and researchers are not the only people concerned with retaining the human connection and relationship within schizophrenia treatment. Mental health consumers also consistently rank interpersonal relationships with providers as the most essential part of their treatment and recovery. A comprehensive research project by Tunner and Salzer (2006), showed consumer’s ranking “the interpersonal process of service delivery” (p. 678) over medications, case management, and psychosocial treatments in importance.

Seeking Synthesis

While there are clearly moral implications in how schizophrenia is understood and treated, and the psychodynamic perspective carries the great strength of meeting people where they are, making meaning, and comprehending them biopsychosocially, what would a model of integration look like in schizophrenia treatment? Some researchers like Karon (2006) and Sparks, Duncan, and Miller (2006) say that such integration will not be easy. To these researchers, “medical dominance precludes a level-playing field, and...integration further disguises the power dynamics that privilege a medical perspective and diminish the role of therapy” (Sparks et al. 2006, p. 85).

There are some current alternatives programs being built however, integrating both psychodynamic and psychopharmacological perspectives, which could inform the mental health treatment model utilized in the United States. One such program is the Windhorse model (Podvoll, 2003), which utilizes medications at the onset of psychosis but also focuses on community living and psychodynamic therapy. Windhorse adheres to a “basic premise requiring all clients to do two hours a week of intensive psychotherapy with a trained therapist” (Neubegoren, 2001, p. 193) that derives from “the belief that human intimacy can be a significant catalyst for recovery from psychosis” (p. 193).

Another promising treatment model comes from Scandinavia and is based out of the work of Alenen (1997). The research in Scandinavia has culminated with treatments known as the “need –adapted” and “open dialogue” models, which focus on immediate intervention, family and client communication and contribution, and treatment based in individual’s unique needs and experiences. Research on these treatment models has shown remarkable results in regards to decreasing both hospitalization as well as the

potential for the illness to become chronic (Alenen, 1997; Seikkula, Alakare, & Aaltonen, 2000).

While both Windhorse and the Scandinavian models offer promising alternatives to the current treatment system, and both show significantly improved recovery rates, the issue remains not only whether mainstream psychiatry would ever adopt these models, but whether they could ever be affordable. Surprisingly, it seems that the decreased hospitalization and decreased medication required within both of these programs could actually serve to decrease mental health costs overall. As described by Alenen (1997), “the more expensive treatment may sometimes be cheaper for society in the long run” (p. 240).

Even if alternative programs, or implementing aspects of such programs into the mental health treatment system, were cost-effective, this change will probably not occur without significantly more research and funding for research. Whitaker (2002), suggests that a first step towards this would be a National Institute for Mental Health funded research project based on the results found at the University of Turku by Alenen (1997) and others.

Another means by which the biological and psychodynamic perspective might ultimately integrate, and the psychodynamic perspective regain acceptance within the schizophrenia treatment model, actually comes from within the biological perspective itself. Continued research in to the neurobiology of psychotherapy is increasingly showing the profound effects that these interventions have on brain structure, chemistry, and even genetics (Gabbard, 2000). While this in a sense will be the psychodynamic perspective gaining validity through becoming a biological science, it may prove

beneficial in helping these two perspectives ultimately bridge their age-old dichotomy. This does not mean that psychotherapy will replace medications, but rather that these two might increasingly work hand-in-hand to target specific aspects of illnesses.

Further evidence for the importance of the integration of psychodynamic perspectives within the biological model comes from Mintz (2005). Mintz describes how, despite the idea of psychopharmacology as a strictly scientific endeavor, psychological factors play a huge role in how medication is prescribed, taken, and even how effective it is.

“In fact we know that medication response is not straightforward. An emerging evidence base shows how psychological factors play a significant role in the outcome of psychopharmacological treatments. The placebo effect may account for more than 75% of the efficacy of anti-depressants. Patient’s interpersonal styles and attitudes about medication and their treaters can profoundly effect treatment compliance. Psychological variables, such as perception of alliance and readiness for change appear to be powerful determinants of medication response, even more powerful than treatment with active drugs” (Mintz, 2005, p. 187).

An analysis and synthesis of the research conducted within this study shows two critical points regarding the role of psychodynamic therapy within the current treatment system. First, a huge amount remains unknown about schizophrenia and schizophrenia treatment. Despite this, medications should clearly play a central role as they provide the best symptom relief currently available. Secondly, it is still important however that the psychodynamic perspective continue to exist within schizophrenia treatment. This is due to evidence that whether or not the actual interventions utilized by clinicians working with schizophrenia are psychodynamic in nature, the awareness, focus, and perspective provided by a psychodynamic understanding of the dynamic individual living with mental illness is invaluable. As Neill (1990), emphasizes, “those who advocate the

‘twisted molecule’ as the cause of schizophrenia may err just as surely as those who championed the ‘twisted mother’ as necessary and sufficient causes...This illness clearly asks that we take a pluralistic approach, both etiologically and therapeutically” (504).

Implications for Practice and Future Research

There are a number of implications for practice highlighted within this study. The first and most prominent is that the current treatment system, dominated by biological perspectives, remains inconclusive in its findings on the etiology of schizophrenia and approaches to treatment are currently not adequately supporting recovery for a disturbingly high number of people. For this reason, those in the social work and psychology professions must continue to actively engage in advocating for and supporting increased research, understanding, and improved treatment for schizophrenia from all perspectives that may prove beneficial.

Secondly social work and psychiatry training programs must improve their integration of multiple perspectives in treating schizophrenia. Psychodynamic programs must strive to more fully integrate biological theories and provide scientific backgrounds for their students, education which is essential not only as clinicians but as advocates for clients navigating the treatment system. Also, until more complete evidence for or against such interventions can be provided, social work programs must also more fairly include the treatment of major mental illness in their curriculum, doing away with the idea that psychodynamic theory has no bearing with this population. As is evident in the research by Karon and Vandenbos (1981) clinicians with limited training may not be able to provide the tools for adequate treatment, thus creating a self-perpetuating cycle in which

psychodynamic psychotherapy is continually deemed inadequate. Similarly, psychiatry residents could benefit greatly from increased understanding of the psychodynamics of psychopharmacology as described by Mintz (2005).

It is also clear from my research that the role of those with an interest in psychodynamic theory and treatment should be to continue to educate one another, those in the biological community, and the broader public about the nature of psychodynamic interventions. It seems psychodynamic interventions have been largely disavowed due to misinformation and confusion surrounding their connection to psychoanalytic theory and ideas such as regression and interpretation. While supportive interventions and relationship-building may ultimately be the core focus of psychodynamic interventions, it is clear (simply from the extensive data on early trauma in those with schizophrenia) that the psychodynamic perspective will be at least an essential tool for the clinician to more fully understand the client's struggles, whether or not the therapy itself takes on a form that would be considered more ego-modifying, dynamic, or interpretive.

Finally, what is also clear from the research is that all theories, labels, and dichotomies aside, the human relationship plays an essential role in the process of recovery from illness (Tunner & Salzer, 2006; Barber, 2008). It is horrifying to imagine that due to poor funding, or lack of scientific evidence, the possibility for relationship-building may be disappearing from mental health treatment. Not only do relationships strongly improve medication compliance and insight, but it is only through human contact that the subjective experience of those with schizophrenia will ever be understood. There are countless reflections from consumers on the role of hope and

connection in their recovery process (Fisher, 1999; Everett, 2000). This role is described by Fisher (1999),

"When someone is labeled with mental illness, it is as if all that has been learned to be helpful in therapy is thrown out. Medical students are taught to medicate not to converse with mental patients. They are told that people labeled with mental illness have a brain disease and you cannot talk to a disease. Our lived experiences speak otherwise. Our lives show that people labeled with mental illness need a therapist and other people who believe in them. We who have been labeled with mental illness, remain just as human if not more so than others who are temporarily not labeled. Our needs are human needs of which the most basic is to enter into trusting, loving, and caring relationships. These relationships need to be nurtured and cultivated for us to find the compass of our true self to guide our recovery. Any system of care which disturbs or interferes with these relationships is preventing not promoting recovery" (p. 2).

For this reason, those from all therapy backgrounds as clinicians and as humans undoubtedly touched in some way by mental illness personally, must continue to join forces, through compilations of individual case studies, lobbying for funding both for research and insurance coverage, and research coordination between perspectives to help allow these essentially healing relationships to exist.

Conclusion

This chapter has summarized the findings of this study on the role of psychodynamic psychotherapy in the mental health treatments today. While a biologically-oriented and pharmacologically focused model is the prevailing treatment for schizophrenia today, this study highlighted ways in which psychodynamic psychotherapy may serve a purpose both in application and as a useful perspective for clinicians on treatment. The chapter has analyzed key points and discussed possible

avenues for decreased dichotomization and increased synthesis between opposing biological and psychodynamic theories.

Although an integrated model of schizophrenia treatment utilizing the strengths of the biological perspective, the psychodynamic perspective, as well as psychosocial, occupational and consumer-led treatments may be many decades away from realization, the move towards integration must begin now so that we may to more fully and fairly support and witness the recovery of those with schizophrenia.

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