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The Method of Madness: a Brief Biography of Schizophrenia

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The Method of Madness:

A Brief Biography of Schizophrenia

Riley Hawkins

HONR 401: Scott Ross, Kevin Moore, and Janet Vaglia

April 12, 2016

“What does the brain matter compared with the heart?”

-Virginia Woolf, Mrs. Dalloway

Acknowledgements

First and foremost, I would like to thank my mother. Without her encouragement, I can avouch that this thesis would not exist. So many times I wanted to give up: “But Mom, my senior year would be so much easier!” She persisted, “You’re doing it. You can do anything.” Thank you, Mom--it is your influence that has allowed me to achieve so much. Next, I would like to thank Kevin Moore. I believe that when most people leave college they look back on one professor and think, “wow, that guy was really influential.” For me that is, without a doubt, Kevin. He interviewed and accepted me into the Honor Scholar program and for that alone I owe him a stupendous amount of knowledge--something I will forever be grateful for. By taking two courses with Kevin, he has instilled curiosity and wonder in me on so much of the world: “I wonder how that evolved, or I wonder why humans do this?” Next, I want to thank my thesis sponsor Scott Ross. I do not think my thesis is a typical piece of science or psychological writing, and that is all thanks to Scott for allowing me to branch out and do my own thing. Scott also deserves my appreciation for being so patient and understanding of my chronic procrastination. Next, I would like to thank Janet Vaglia for being my academic advisor and also agreeing to be on my thesis committee. I would also like to praise Janet for being an inspiring female biologist and professor. Next, I would like to thank Amy Welch for all of her hard work she does for the Honor Scholar Program, DePauw, and the Greencastle community. I would like to thank my Aunt Linda for the inspiration that drove me to write on this topic. This is for you, Aunt Linda. Finally, I would like to dedicate my thesis to anyone who has ever suffered from the stigma of mental illness. It is my sincere hope that my knowledge will educate and change the hearts and minds of others.

Table of Contents

Introduction	6
Establishing the Roots: Origins and Early History	13
<i>The Beginning</i> 14	
<i>Genesis of Terminology</i> 15	
<i>Antiquated Treatments</i> 16	
From the Darkest Night: Diagnosis	22
<i>Elyn</i> 23	
<i>Lori</i> 27	
<i>Kurt</i> 30	
An Abnormal Reality: Symptoms	33
<i>Alterations of Senses</i> 34	
<i>Inability to Interpret and Respond</i> 36	
<i>Delusions and Hallucinations</i> 38	
<i>Changes in Emotions</i> 40	
<i>Anosognosia</i> 40	
The Method Behind the Madness: Theories of Etiology	41
<i>Genetic Theories</i> 42	
<i>Neurotransmitter Theories</i> 46	
<i>Neuropathological Theories</i> 49	
<i>Developmental Theories</i> 50	
<i>Infectious Theories</i> 52	
An Enigmatic Legacy: Evolutionary Theories of the Schizophrenia	55
The Battle Behind the Forehead: Treatment Strategies	63
<i>Antipsychotics</i> 64	
<i>Psychosocial</i> 77	
<i>Early Intervention</i> 85	
Invisible Wounds: Issues faced by Schizophrenics	87
<i>Deinstitutionalization</i> 87	
<i>Substance Abuse</i> 92	
<i>Suicide</i> 95	
<i>Stigma</i> 98	
The Sun will Rise: Conclusion	112
<i>The Ever After</i> 112	
<i>Aunt Linda</i> 114	
References	117

The Method of Madness: A Brief Biography of Schizophrenia

Introduction

I awoke that day much more uneasy than I had suspected. I was excited, but also nervous in a way that I could not quite understand. When daunted by nerves, I tend to handle grueling anticipation in a step by step fashion. Step one: get out of bed. Step two: get in the shower. Step three: hair and makeup. Step four: hurry out the door, because at this point I was inevitably behind schedule. The next step in line, was the familiar drive to my Auntie Carol's house. Auntie Carol is my father's sister who is the kind of aunt that feels much more like a second mother in many respects. I arrived at her home just two minutes from her suggested time of arrival, an impressive feat. I stepped inside to find her searching for our destination's address on her iPad, and I recall finding it somewhat odd that she was not exactly certain as to where her sister lived. Regardless, we quickly pinpointed where we were headed on my phone's GPS, and we were on our way to visit my Auntie Carol's sister, my Aunt Linda.

I grew up hearing lots of stories about Aunt Linda from my older cousins: "remember the time she took it upon herself to cut Alex's hair, and dad unleashed his wrath?" or "she once sent me a birthday card with a 5 dollar bill and 2 dimes." I am the youngest of this generation, meaning that I did not have as much interaction with our infamous Aunt Linda. In fact, I had grown up knowing little to nothing about my Aunt Linda. In all of my 22 years on earth, I knew just one, fairly distinct thing about her--she has schizophrenia.

I have known my Aunt Linda suffers from schizophrenia for quite sometime, long before I had any understanding of mental illness. In fact, I can remember when my mother told me about her illness, and I thought the word "schizophrenia" sounded so

bizarre, so completely strange that however the illness afflicted Aunt Linda had to be exceptionally bad, thus making her particularly weird. This rather simple thought process remained steadfast throughout much of my youth. When I was about 9 year old, Aunt Linda moved about an hour away to a group home, and I soon forgot about her existence until I was reminded each year during the holidays when I received my annual, blank birthday card from her. Her logic is that the blank card is the gift--so thoughtful, yet somehow very psychotic. Once when I was about 16, she sent me a bottle of White Rain shampoo for Christmas and my birthday. I distinctly feeling a little troubled by this, because I could not understand how anyone could possibly think shampoo is an appropriate gift.

The first time I ever learned any of the serious implications of schizophrenia was during a lecture in intro psychology during my first semester at DePauw. I remember schizophrenia was covered with an array of psychological disorders during the week that we covered all of abnormal psychology. True to intro psychology, we barely skimmed the surface of schizophrenia during the lecture: disordered thoughts, hearing voices, significant genetic component, with symptoms arising in young adulthood. "Wait a second...", I thought, "I was in the throes of young adulthood, with a schizophrenic aunt... Was I about to go crazy!?" Thankfully, my head remained clear of voices, aside from my own. It was not until my junior year in a class called Evolution and Human nature, that I would hear the word schizophrenia again. The class was examining how psychological disorders like depression, anxiety, and schizophrenia persist in the population, as they should be selected against by natural selection. I recall being particularly interested in the section on schizophrenia in our assigned reading, as it

alluded that schizophrenia persists because relatives of schizophrenics often have evolutionary advantages like increased intelligence and creativity. Not to toot my own horn, but I wondered if this so called evolutionary advantage might apply to me? I have always had a knack for all things artistic: drawing, scrapbooking, mosaics, ceramics, painting, etc. Art class has always been my favorite. In addition to this, I have also always had a certain knack for academics. In other words, I love to learn. Upon finishing the section on schizophrenia in the reading, I was left dumbfounded: “is my academic success due, at least partly, to my Aunt Linda’s illness?” This question lingered in my mind for quite sometime. Like much of society, I knew only the bare minimum about schizophrenia, basically only what I could recall from intro psych. Over the next few months, I began to feel like I owed it to my Aunt Linda to learn about the illness that has taken so much from her and has seemingly given so much to me.

Fast forward one year and Auntie Carol and I are on our way to visit Aunt Linda, so that I could have a frame of reference for my honor scholar thesis. The weather that day was beautiful, unusual for the end of January, even by Indiana’s standards. On the way, Auntie Carol and I had the standard conversation between relatives and college students, “Yes, school is going fine.”, “I’ve been accepted as a corps member for Teach for America”, and “No, there are no boys of interest as of yet.” After covering all the bases, we began to discuss my senior thesis and just why I had requested to make this visit. As we drove, Auntie Carol shared a lot about her childhood with Linda and how she had always suspected that something was off, but her opinion was never given any consideration, as she was just a child.

When we arrived at the group home, I was extremely nervous. I had not a clue what to expect, even after having read substantial literature on schizophrenics. I now understand that this nervousness was just fear of the unknown. I had feverishly speculated, “will she babble to herself?”, “will her laugh be weird or frightening?”, “will she struggle with conversation?” I had no conceivable idea, but I was about to find out.

I soon found myself returning to the step-by-step method to calm my nerves. Step one: get out of the car. Step two: go inside the building. When we opened the door to the small facility we first had to check in with the staff. Auntie Carol politely stated, “Yes, we’re here to see Linda Hawkins.” The staff was eager to meet us, particularly me. They noted that she talked very frequently about her nieces. I found this heartbreaking, as I had very rarely given her any thought or consideration before I started my thesis. The staff directed us to the main living space. The dining room, living room, and kitchen of the group home were each decorated in a manner that I can only describe as “grandparents who had managed to update their home in the late 1990s”: beige walls, dated couches and chairs, oak furnishings, a non-flat screen TV, and mass produced water colors adorned the walls. The place really seemed to strive to embody a group home for the mentally ill. After I had accessed the aesthetics, my eyes quickly found Aunt Linda eating her lunch at the large dining room table. She immediately recognized Auntie Carol and I. Auntie Carol introduced me, just to ensure she knew who I was, to which she responded, “yes, I knew you were coming, but I was expecting Cathy not Riley.” There was no way she had any conceivable idea that we were coming. The visit was something that had worked out last minute, and who knows when the last time my cousin Cathy had talked to her. Immediately I knew this was going to be a very interesting visit...

Auntie Carol and I sat down to join her at the table. She was eating hominy, and every bite seemed like it was carefully thought out. I soon realized it was because she did not have her dentures in. Auntie Carol was very disheartened that she had dentures, as they were due to the inevitable side effects of antipsychotics, causing her to lose her teeth. Auntie Carol later told me that Linda had always taken very good care of her teeth. As Linda ate her lunch, we began to have, for lack of a better word, *normal* conversation. Auntie Carol lead most of their discussions, and I simply observed, taking it all in. They talked about people they had went to school with, and I was quickly fascinated with the fact that Linda was not missing a beat. She seemed to have a pretty powerful memory and seemed genuinely interested in Auntie Carol's gossip. Notwithstanding, moments later she thanked Auntie Carol for a Danielle Steel novel that Auntie Carol had given her for Christmas the year before, and it was shortly after this that I realized that Linda's psychosis is always at arm's reach. I asked, "Do you enjoy reading?"

She replied, "yes, I've been reading a lot lately."

I asked, "what kinds of books do you read?"

To which she responded, "I really enjoy philosophy."

Not expecting such an academic response, I questioned, "Really?"

She said, "Yes, I really like Plato and Aristotle."

For all I knew, she might have *actually* been reading Plato and Aristotle, but then I asked, "How many books have you read lately?" To which she said, "Oh, about 1 every 5 months or so." It was then that I understood she was delusional. I quickly redirected us from our philosophical conversation, but who knows, maybe Danielle Steele novels are

just as difficult to read for her as Plato and Aristotle might be for an unbroken mind.

Ancient philosophy, romance novels--what's the difference, anyway?

After our inquisitive literature conversation, Auntie Carol began to explain to her that I went to DePauw University, and I had received the Lilly scholarship for Orange County. She explained to Linda that this was a full ride scholarship, and that she should be very proud. Aunt Linda remarked, "Yes, college is very expensive. What is it about \$4,000 a year?" Auntie Carol and I both found this extremely comedic, but Auntie Carol responded gently, "No, try about \$50,000." Aunt Linda was astounded, and I could tell that schizophrenia had damaged her ability to interpret time. I puzzled if she understood that it was 2016, not 1973. We made more small talk, and we soon made our way to Aunt Linda's room. Unsurprisingly, her room was an absolute disaster. Anything and everything one could imagine was stacked on the floor, leaving a small path to her bed, and her closet was full of God knows what. In the masses of what I can only describe as junk, my eyes caught sight of a small, unicorn statue. The statue was painted with soft pastels and had an iridescent glaze. It was the embodiment of a little girl's imagination. I told her I loved it and asked her where she got it. She began explaining that she had made it with her dad when she was a little girl. Even if Auntie Carol had not been shaking her head in disbelief, I could easily interpret that this was a delusion. Although I never met my Grandpa Hawkins, as he died long before I was born, I could reason that he was probably not the type of man to make unicorn statues. Nonetheless, I accepted her delusional explanation and moved on.

Auntie Carol and I left shortly after the unicorn fiasco. We reasoned to Linda that we hoped to visit more often and the next time we might go out for dinner. We

exchanged our goodbyes and left the group home sans hugs, which seemed most unusual to me but probably quite customary for Aunt Linda. Seconds later Auntie Carol and I were out the door and headed home. Upon making it to the car, my head was buzzing. What I had just experienced was similar to what one might feel after seeing a Van Gogh painting for the first time in real life. One might learn that he made violent brushstrokes and used vibrant colors, but prints cannot depict these characteristics of the originals. They can only be appreciated by seeing the real thing. Similarly, I had been researching schizophrenia for months, and having met someone with the disease after the research was both awe-inspiring and fascinating--much like seeing a Van Gogh original for the first time.

I had begun the day I visited Aunt Linda quite anxious about making the visit. On the contrary, I went to sleep that night disheartened that mental illness, particularly schizophrenia, is so misunderstood by society. I had come to understand that my Aunt Linda, was *almost* like a normal person. While her delusions and disordered thoughts made life more challenging for her, her illness does not hinder her day to day life. I now understood, first hand, why experts in the field express such frustration with stigma. Consequently, I want this thesis to take my experience with Aunt Linda and enlighten those who might be fearful or ignorant to schizophrenia's obscurities. Schizophrenia has been at the forefront of scientific and political discovery, challenge, controversy, and progress for centuries. This disease has an incredibly fascinating, interdisciplinary story to tell, and I can only hope I do its story justice to help end societal stigma that has victimized my Aunt Linda and so many others.

This thesis will first explore schizophrenia's early history, when and where does madness first appear, and who were the pioneers that established the disease as we know it today? This section will also analyze antiquated treatment methods, focusing on the horrifying doctrines of the 20th century. Next I will introduce three accounts of schizophrenia from three autobiographical accounts. They are taken from *The Center Cannot Hold* by Elyn Saks, *The Quiet Room* by Lori Schiller, and *Me, Myself, and Them* by Kurt Snyder. In this section I will describe what a schizophrenic diagnosis might look like by examining each author's early life up until their first or most dramatic psychotic break. Next, I will describe the symptoms of schizophrenia while incorporating passages from the autobiographies described above to help illuminate the severity of their effect on day to day life. The next section will detail the etiology of schizophrenia. To some this section might be considered dense; however, I encourage one to not be discouraged or bogged down but retain inquiry, as the etiology of schizophrenia has an enormous implication on its treatment and even its perception by society. As a biology major, this section and the next on schizophrenia's evolution are particularly fascinating, as much of the engineering behind the human brain continues to remain elusive even to researchers in the 21st century. The next section will cover the current treatment strategies of schizophrenia, both medicinal and therapeutic. This section and the next will also include personal insight from the autobiographies described above. Next, I describe only a portion or perhaps the most common issues and injustices that schizophrenics continue to face today despite advanced scientific research and improved treatment models. Finally, I will use the stories of Elyn, Lori, Kurt, and my Aunt Linda to depict that a schizophrenia diagnosis today should elicit hope, rather than fear.

Establishing the Roots: Origins and Early History

Some experts contend that schizophrenia is a modern disease and has only arisen within the last 200 years. While we have no definitive proof of schizophrenia's origin, there are accounts of madness that exhibit symptoms similar to schizophrenia dating all the way back to the ancient Egyptians, Greeks, and accounts are further solidified in the Middle Ages. With that being said, the formal description of schizophrenia has only existed for about 150 years. Unsurprisingly, schizophrenia's history is quite mystic, even scary. However, what many find *truly* scary are the antiquated methods of treatment, not the symptoms of madness. The outdated treatments highlighted with insight from antique research papers are electroshock therapy, pre-frontal lobotomies, and psychoanalysis, each being of common practice in the relatively recent history of the 20th century.

The Beginning

Some of the world's oldest medical texts originate in Ancient Egypt. For example, the Papyrus Ebers, a scroll projected to date back to 1500 BC, contains references to mental conditions with associated diagnoses (Owen, 2014). These ancient academics believed the heart and mind were the same entity. Therefore, the ancient Egyptians often diagnosed most conditions that are now associated with the brain as diseases of the heart. Nevertheless, these writings are some of the first to mention any disease associated with the brain (Owen, 2014). It was not until 1100 years after the Papyrus Ebers were written that Hippocrates described mental conditions as problems within the brain, not the heart.

Most ancient diagnosis of disease were summarized in a medical handbook, written by the celebrated Greek physician Aretaeus around the second century AD. Many of the classic symptoms of schizophrenia were depicted in Aretaeus's book as conditions of "mania" and "melancholy" (Owen, 2014). Aretaeus's prescribed treatment? Humoral

pathology, more often referred to as bloodletting. Humoral pathology is the idea that diseases are caused by an imbalance of blood, phlegm, and black and yellow bile within the body. Humoral pathology was also practiced by the Greek physician Galen who influenced much of the medicinal philosophy and ideas of the Roman Empire. Galen, like Hippocrates, attributed mental disorders as a brain disease--setting perhaps the earliest foundation for the field of psychiatry (Owen, 2014).

Galen's ideas influenced much of Europe throughout the Middle Ages; however, Galen's ideas based (somewhat) in science were at odds with the religious culture of the time. Therefore, religious superstitions began to dominate the ideas and treatment of mental health. Many Europeans believed that hallucinations and delusions were a cause of demonic possession and witchcraft. Researcher and medical student Ruaridh Owen postulates that witches were often forced to tell how they came to be possessed, and their confessions were likely similar projections of the auditory hallucinations that are experienced by paranoid schizophrenics even today, thereby making it possible that the "witches" described in the Middle Ages were actually just individuals who suffered from schizophrenia (2014). Exorcisms were the go to method of treatment in the 15th century for mental illness.

Genesis of Terminology

In 1893, the term *dementia praecox* was made famous by the German psychiatrist Emil Kraepelin. Kraepelin was the first to hypothesize that specific combinations of symptoms in relation to the course of psychiatric illnesses allow one to identify a particular mental disorder (Ebert & Bär, 2010) Meaning that he was the first to report a distinction between manic depression and *dementia praecox*. Kraepelin considered

dementia praecox as a biological illness caused by anatomical or toxic processes (Ebert & Bär, 2010). Kraepelin used the term *dementia* because he believed that it was a progressive and degenerative disease, which resulted in the irreversible loss of cognitive functions. In contrast, he described manic depression as an episodic disorder, which does not lead to permanent cognitive function (Ebert & Bär, 2010).

The word schizophrenia--which translates into “splitting of the mind” from the Greek Roots *schizein* (“to split”) and *phren* (“mind”) was coined by Swiss psychiatrist Eugen Bleuler in 1908 while at a meeting for the German Psychiatric Association in Berlin. Bleuler liked the ideas of Kraepelin, but he wanted to make a novel distinction (Ashok et al., 2012). In 1911, Bleuler wrote “I call *dementia praecox* schizophrenia because, as I hope to show, the splitting of the different psychic functions is one of its most important features. In each case there is a more or less clear splitting of the psychological functions: as the disease becomes distinct, the personality loses its unity.” Bleuler first described the the main schizophrenia symptoms as 4 A’s: flat affect, autism, impaired association, and ambivalence. Bleuler was first to notice that some patients improve rather than deteriorate over time, thus the importance of moving away from degradation language proposed by Kraepelin (Ashok et al., 2012).

Antiquated Treatments

Early treatments of schizophrenia were restricted by a lack of knowledge of the brain and the neurochemical aspects of the disorder. However, Bleuler and Kraepelin’s early work seems to be established on better and certainly more ethical research than much of what followed. Early 20th century treatments were crude and very under researched, many of which lacked any scientific evidence. Combine these Frankenstein-

like experiments with the asylum conditions reported in 1946 by Albert Q. Maisel in *Life* magazine, and one is able to recognize just how far we have come in the way of treating mental illness. One testimony from the *Life* issue came from a mental hospital in Ohio:

“An attendant and I were sitting on the porch watching the patients. Somebody came along sweeping and the attendant yelled at a patient to get up off the bench so that the worker-patient could sweep. But the patient did not move. The attendant jumped up with an inch-wide restraining strap and began to beat the patient in the face and on top of the head. 'Get the hell up...!' It was a few minutes -- a few horrible ones for the patient -- before the attendant discovered that he was strapped around the middle to the bench and could not get up.”

Prefrontal Lobotomies

Prefrontal lobotomies were introduced by Egas Moniz in Portugal in 1936. Moniz would somehow win a Nobel Prize in medicine for the introduction of the procedure. A paper from the Journal of American Medical Association in 1943 notes that the lobotomy, even at the height of their popularity, were considered a radical procedure that should only be utilized on cases where all other treatment had failed (Bennett, 1943). Bennett claims that a “definite change in personality occurs after lobotomies, noting that patients become “more extraverted, less self-conscious, no longer aggressive, and responsive to abnormal emotional drives, thus becoming a socially acceptable individual.” Patients are also described as “childlike and cheerful” after the procedure. The descriptions of socially acceptable, childlike, and cheerful violently contradict the outcomes of these procedures, as patients were basically rendered completely helpless. A procedure that utilized a modified ice pick, hammered into the eye socket and moved side

to side to separate the frontal lobes from the thalamus should never have been described as having “beneficial results” (Bennett, 1943).

The paper delivers 5 case studies of the procedure, all with varying degrees of horror. One recounts a middle aged teacher who tried to stab her nephew. The case report describes her as being “restless and resentful of hospitalization” (Bennett, 1943). She was given 12 rounds of electroshock therapy before the lobotomy was performed. The postoperative report reads, “The patient was untidy at first. Her belligerent and spiteful remarks have changed to sarcastic and witty ones.” Her follow-up report comments, “She has absolutely no insight but is able to make good social adjustment. She has been spared institutional life and is a fairly useful, capable person.” One should be left wondering how a person with “absolutely no insight” could ever be even “fairly useful”. The remaining 4 case reports described in the paper are similar to this, often reporting “carefree nature”, loss of memory, and weight gain.

Bennett’s report also describes the recovery period of these patients as regressive to infantile levels, as the patient had to be fed and moved about in bed and they, quite understandably, gave no attention to excretory functions. This despicable science continues, “Some patients carry our repeated acts indefinitely, but gradually the patient becomes more tidy” (Bennett, 1943). Contradicting most of these prior statements, Bennett goes on to report, “the patient must be forced out of bed, taken to the toilet at regular intervals, removed from the toilet, forced to bathe, dress, and feed himself.” The paper concludes by suggesting that the degree of recovery cannot be determined until at least 3 years after the procedure and that further research and study is needed for aid in normal living post surgery. One can only imagine what life was like for, not only for the

patient, but also the caregivers after those 3 years came and went. Thankfully, the use of prefrontal lobotomies vanished with the advent of antipsychotics in the early 1950s.

Electroshock Therapy

Electroshock therapy was introduced in the 1940s, with the idea that inducing seizures might improve psychotic symptoms. A 1946 case report from Harvard University notes that, “judging from the patient’s behavior, the trembling, the profuse sweating, and the impassioned verbal pleas for help and release, it would appear that most patients find the treatment unpleasant.” If this is not disturbing enough, the report continues by presenting this theory, “it has been suggested, for example, that the treatment threatens the patient with death and offers him an opportunity of rebirth cleansed of previous fears, anxieties, and confusions; or that, the treatment is a form of punishment which absolves the patient from overwhelming guilt feelings.” This report seems to keep true to the scientific theme of the time period, little evidence and a lot emphasis on inducing punishment and guilt.

Following the introduction, the report gives a detailed, first hand account of a 24 year old manic college student’s experience with the therapy. Before the procedure began he recounts, “there was another scream and a gurgling coughing groan, and the patient ahead of me was moved down the hall into the dormitory, with arms, legs, and head flopping around.” Not only was he able to witness the prior patient’s experience, but he was also able to remember in striking detail his own experience:

“One held my right arm and pressured hard with the elbow just inside my shoulder muscle. The other had the other arm. Another climbed the wagon and laid across my knees--all three holding me down during my convulsion. The

theory was: the more severe the convulsion, the better the results. I heard the doctor give the pretty blonde nurse a set of numbers, and I knew she was setting the dials. Very deliberately, very slowly a black shade came up over my eyes. I woke up sometime later and could think of nothing” (Alper, 1946).

From the patient's account, we can gather that electroshock treatments were also a monstrous attempt at treatment. Often times they were only truly successful at causing large, blatant gaps in the memory of many patients.

Psychoanalysis

The 1950s were dominated by psychogenic theories of etiology. These were theories that projected the idea that schizophrenia was caused by early problems in interactions between a child and their parents. Psychoanalysis was often the go to treatment, and at the time it was thought to be curative. Unlike today’s therapies that act to change a patient’s thinking or behavior, psychoanalysis was based on assumptions and projections to which there is no conclusive evidence to support. Below I present some of the belief systems and models that helped guide this belief in a paper entitled, “*Parents of Schizophrenic Children Workshop, 1958.*” This paper makes one wonder if those who put any stock into the claims of psychoanalysis might have been just as delusional as the schizophrenics they were attempting to treat.

The authors begin the paper by establishing that ego (the part of our personality that prevents us from acting on our basic urges) disturbances within schizophrenic children can be caused by either emotional or organic processes. The work further asserts that in all the cases diagnosed as childhood schizophrenia, there was severe emotional impairment arising from the emotional disturbances in the parent child interaction. The

authors further claim, “that the schizophrenic child presents himself as a fragment of an identity with distorted relation to reality” (Kaufman et al., 1958).

After establishing that the child’s schizophrenia arose from the disturbed relationship between them and their parents, the authors further assert psychogenic ideas by claiming that the parents of schizophrenic children had severe ego disturbances that prevented them from mastering their fear of being overwhelmed by their primary anxieties (Kaufman, 1958). This perspective believed that this fear was associated with a fixation at the oral level of development, before the ego had developed “the usual appropriate methods to cope with this threat.” According to Kaufman et al., psychosis was supposed to protect the individual from being overwhelmed by anxiety, but it also resulted in a loss of self-esteem. The idea here was that these defenses by the parents created the range of parental personalities seen in schizophrenic children (Kaufman, 1958).

This paper suggests that regardless of external behavior (which can appear adequate, i.e. they were actually good parents), these parents had a core ego disturbance associated with “primitive destructive wishes and fears at the oral level of development.” The authors assert that a diminished ego confused one variety of tension for another, attempting to block life experiences to keep their anxiety at a tolerable level. Somehow this was supposed to indicate that there was lack of differentiating between self from nonself, and by not being able to distinguish between self and nonself, the parents “projected their disturbance onto the child who became schizophrenic” (Kaufman, 1958). According to this theory, the child became the embodiment of the parent’s underlying

pathological process, and the child's behavior exhibited the overly psychotic mechanisms with which the parents were supposedly struggling.

As if the explanation for the child's psychosis was not perturbing and far fetched enough, the principal concept of treatment of these parents was the therapist's attempt at "maintaining an awareness of the core disturbance and recognizing and dealing with its expressions." The authors note that this could be a long-term process that required "a great deal of patience and understanding of how fearful and threatened these parents are." It is of particular interest to note that when the parent's tried to present themselves as adequate parents, the therapists took the parents' pleas as defenses of their self-esteem. This behavior by the parents was allowed early on in treatment, but it was eventually unpermitted by the therapist in order "to ensure it would not blot out the eventual discussion of pathologic material," which was essentially the therapists trying to make a feeble attempt at analyzing the early childhood of the parents. The language used by these authors was unbelievably dense, as is evident from what has been presented. One might question if this language was deliberately dense at an attempt to disguise just how nonsensical it actually sounds: "schizophrenic children are the result of the inability of their parents to deal with anxiety that arises due to the possibility of being weaned too early as an infant."

From what I have presented, schizophrenia's history has been established as particularly turbulent. However, it is important to understand the history of this disease for several reasons: first, it is useful to look back and establish what treatments have not been successful to ensure that ideas for new treatments draw from new ideas; to recognize how far we have advanced, from bloodletting and exorcisms to dopamine

antagonists; and finally, to understand how stigma might have been created so that it might some day be fully eradicated.

From the Darkest Night: Diagnosis

The inner workings of madness cannot be fully understood without looking to first-hand accounts, experiences, and journeys. Therefore, I will recount three stories of obtaining a the same verdict: schizophrenia. Each story is unique, not only in its details, but in the symptoms and demographics of its sufferer. First, I will recount Elyn Saks's journey from *The Center Cannot Hold*. Elyn's account is unique because of her brilliance and determination, as she has managed to acquire numerous academic degrees and is a professor of law. Her primary symptoms are delusional thoughts. Next, I will deliver Lori Schiller's story from *The Quiet Room*. Admittedly, *The Quiet Room* is not an easy story to read, as it recalls, with tremendous detail, the horrors of Lori's thoughts and auditory hallucinations. Next, I will draw from Kurt Snyder's narrative from his book entitled *Me, Myself, and Them*. Kurt recalls his suffering of severe paranoid delusions. Each diagnosis was unique, but the ultimatum associated with the diagnosis was all the same.

Elyn

Elyn Saks grew up in Miami, Florida to an affluent family. Her father was a lawyer, while her mother stayed home, taking care of every want or need from her and her two younger brothers. Elyn speaks very positively of her childhood, noting that it was full of laughter, music, and movies. However, she notes that when she was about eight years old, she began to do things a little differently, claiming that she developed "quirks." She goes on to say that not long after the quirks developed, she was joined by nights filled with terror. She recounts that she knew someone was "just outside the window, just

waiting for the right moment, when we were all sleeping, with no one left on guard. *Will the man break in? What will he do? Will he kill us all?*” (Saks, 2007). Elyn details that her first feelings of estrangement from the world began in high school, “I grew somewhat quiet and withdrawn--’in myself’, as I came to call it. Unless spoken to, I didn’t have much to say; I wasn’t sure I even deserved to be heard. I started to believe that speaking was actually ‘bad.’”

As I mentioned earlier, Elyn was gifted with magnificent intelligence. She began her education at Vanderbilt in Nashville. It was during her freshman year that Elyn described the symptoms of schizophrenia “rolling in like a slow fog.” She notes that she often forgot to take care of herself, meaning she went for long bouts of time without showering or brushing her teeth, as the mind forgets to ask those sorts of questions, “have you eaten today?” or “how often do I need to change clothes” (Saks, 2007). She speaks of social isolation in the early days at Vanderbilt, eating many meals alone. She notes that while she was at Vanderbilt she was constantly fighting to keep her shell strong, not allowing any signs of illness break through. Fortunately, this strategy worked at this point in her life, as she made straight A’s, taught herself to read Aristotle in the original Greek, and she was awarded the Marshall scholarship to get a graduate degree in philosophy at Oxford.

Elyn’s psychosis began to break through the shell she had managed to create while at Vanderbilt in the first few weeks upon arriving at Oxford. She came extremely delusional, citing that she often had difficulty differentiating between what was real and what was not real. Elyn’s delusions were often on her unworthiness, “*I am bad, not mad. Even if I were sick, which I’m not, I don’t deserve to get help. I am unworthy*” (Saks,

2007). She had difficulty adjusting to the structure of Oxford courses, and she inevitably fell behind. Consequently, she grew very depressed which led her to seek help. This was the first time that she was suggested to be hospitalized. At first, she rejected the idea. Then, recognizing that she was very depressed, checked herself into the day program at Warneford Hospital so that she could get better in order to continue at Oxford. She notes that at this time she felt, “only desperation and a profound isolation that every day seemed to burrow more deeply inside me. What a waste of oxygen it was for me to draw breath. Suddenly, the solution presented itself: killing myself. It seemed the best option” (Saks, 2007).

Thankfully, Elyn got the help she desperately need at Warneford. She was quickly admitted as an inpatient. She began to get consistent therapy and show signs of improvement upon agreeing to take antidepressants. Nevertheless, she was eager to get back to her studies at Oxford, and she left the hospital against medical advice. Unsurprisingly, her symptoms returned and she was hospitalized for a second time. During her second hospitalization is when her psychosis became much more severe. She recounts,

“I began to feel I was receiving commands to do things--such as walk by myself through the old abandoned tunnels that lay underneath the hospital. The origin of the commands was unclear. In my mind, they were issued by some sort of beings. Not real people with names or faces, but shapeless, powerful beings that controlled me with thoughts (*not* voices) that had been placed in my head. *Walk through the tunnels and repent. Now lie down and don't move. You must be still. You are evil.*” (Saks, 2007)

Her illness began to command her to hurt herself. She burned herself with lighters, cigarettes, electric heaters, even boiling water. Like many schizophrenic individuals, Elyn did not tell the hospital staff about these symptoms. She adverts to a deep feeling of embarrassment and shame. Elyn's symptoms did not improve during her second hospitalization; nevertheless, the hospital staff discharged her on the bounds that she would begin a regimen of "serious talk therapy." She was recommended to see a psychoanalyst by the name of Elizabeth Jones, whom Elyn referred to as Mrs. Jones. Elyn quite blatantly establishes that Mrs. Jones was not a counselor but an analyst full of intellectual rigor. Personally, I cannot understand how one can find any solace in psychoanalytic treatment for schizophrenia; notwithstanding, it worked very well for Elyn and whatever works for someone, well *works*.

Once Elyn left the hospital and began to see Mrs. Jones regularly, she was able to finish her philosophy degree at Oxford with her thesis receiving excellent reviews from her examiners. Elyn stayed in England for a year after she graduated in order to continue her analytic treatment with Mrs. Jones. During this year she actually volunteered at Warneford Hospital and also decided to go to law school, landing on none other than Yale.

It was during her first year at Yale that Elyn would have, what I would consider, her most dramatic psychotic break. She not only dealt with the stress of a new environment, but she refused to take any sort of medication. The fog of madness soon settled in. While visiting a professor during his office hours, she climbed out his window, onto the roof of Yale Law School. She recalls the outburst of madness,

“I spent the next hour or so laughing and singing and gibbering away on the roof of the Yale Law School. I found several feet of loose telephone wire up there, and made myself a kind of belt. I picked up all sorts of metal objects lying around on the roof and attached them to the belt. The best find was a rather long nail, six inches or so. I put it in my pants pocket, just in case I needed protection” (Saks, 2007).

While with the same professor, she called an old friend from Vanderbilt’s neurologist husband, Richard. The conversation went a little something like this, “Elyn, is something wrong?”, Richard asked. “Come to the Florida sunshine tree,” she replied.

He asked, “What do you mean?”

She babbled back, “Fresh tasting lemon juice naturally. There’s a natural volcano. They put it in my head. It’s erupting. I’ve killed lots of people. I’ve killed children. There’s a flower on the bookshelf. I can see it blooming. Have you killed anyone, Richard? My teacher is God. I used to be God but got demoted. Do you think it’s a question of Kilimanjaro?”

Richard urged her professor to take her to the hospital immediately and soon after she was taken to Yale-New Haven Hospital, before she was quickly transferred to Yale Psychiatric Institute.

While in YPI, Elyn reports in astonishing detail the atrocity she experienced: being placed in restraints for hours on end with medicine being shoved down her throat. One should speculate *how* on earth this kind of treatment happened was allowed to happen in the United States? Like any human being who is forced to succumb to such blatant, detrimental treatment, Elyn fought back against the restraints in anyway she

could--screaming, humming Beethoven, begging and pleading. She also made several escape attempts, landing her right back in the restraints. Elyn received her first formal diagnosis during this hospitalization--*chronic paranoid schizophrenia with acute exacerbation*.

Lori

Much like Elyn, Lori Schiller was a bright, young girl when she was thrown into war with madness. Lori notes an exceptionally happy childhood, filled with love, comfort, and friendship. She adds that when growing up she had always felt special, she was the oldest and the only girl. She loved attention, and to get it she strove for the highest achievement. Lori describes her onset of psychosis as having a pleasant beginning, "Sometime during that summer things began to change. At first, the change was pleasant. Somehow, without my quite knowing why, everything seemed much nicer than it had been before. The lake seemed more blue, the paddlewheels bigger and the sailboats more graceful than ever before" (Schiller, 1996). She was at summer camp, and everything seemed right with the world. Nevertheless, her "mood began to shift, and the brightness of the world began to darken. Then came the dreadful thoughts." She recalls that her mood began to "turn black," and a dark haze settled around her. Perhaps the most enlightening account of the experience, "Then, in the middle of this chaos, a huge Voice boomed out through the darkness. 'You must die!'" Upon returning home from camp, she kept the voices to herself. During her last year of high school the Voices came and went without warning, but ultimately she had more good days than bad during this infantile stage of psychosis.

She enrolled in the prestigious Tufts university, and on the surface everything was going well for her. She had lots of friends, a vibrant social life, and did well in her classes. On the inside, however, the Voices were becoming louder and faster. She adds that they filled her with anxiety, and she was always tense. She laments,

“What tortured me more than anything was when the Voices laughed at me. It was kinds of hysterical laughter, as if I was the target of everyone’s jokes. I didn’t know why they were making fun of me so viciously but I hated myself for being the sitting duck of ridicule. I became extremely self-conscious in front of everyone for fear that they too would nail me to a taunting cross” (Schiller, 1996). During this time Lori’s “highs were higher” and her “lows lower.” Self harm entered her mind: “I thought increasingly about hurting myself. I sat in the library, up all those flights of stairs, and considered jumping” (Schiller, 1996). At this point, she clearly knew something was wrong, “something was about to snap.” She began meeting with a counselor at Tufts and a psychiatrist every week in a private practice, but she never brought herself to mention the Voices.

After graduating from Tufts, Lori moved into an apartment in New York City with a close friend. She had a great job in the city, but things began to unravel quickly, and within months she had attempted suicide for the first time. Her psychosis quickly spiralled out of control. She was hospitalized for the first time, but with the help of her concerned father, she was released quickly without any evidence of a psychotic break on her record. Even so, when Lori returned home she was unable to retain a job, her friend had moved out, and her apartment became a disaster. She smoked cigarette after cigarette to pass the time, to ease the pain of psychosis. Soon, her parents enrolled her in a clinic

that specializes in psychopharmacology, essentially a program that prided itself in loading up its consumers with potent drugs. Soon after this development came her second suicide attempt and second hospitalization.

Upon her second hospitalization, Lori was transferred to The Payne Whitney Clinic of New York Hospital, a place that specialized only in short-term psychiatric problems. Her father remembers that the first time he saw the hospital he understood that it “was no ordinary place where ordinary people came to get well.” When Lori’s father saw her in this environment for the first time he bemoans, “I saw my daughter. But it was not my daughter. The Lori I knew was gone. And in her place was a stranger, a person who seemed to be living only partly in this world, and partly in some faraway world of her own making... The illness had captured her, and was apart of her” (Schiller, 1996).

The Quiet Room offers insight from medicinal notes on Lori from this time period. A primary therapist penned,

“Patient remains agitated and intermittently actively hallucinating. It became clearly evident in discussion how tormented she is by these voices and how hard she is fighting to resist their commands. Much of her treatment resistance appears to stem from fear of the repercussions of revealing these hallucinations to staff.

‘They’ll kill me if I tell.’”

Lori’s mother visited her daily while she was in the hospital. Her mother remembers Lori was often in a fog, “as if a curtain were drawn between us.” Her mother expresses frustration in *The Quiet Room* that after Lori had in the hospital for quite some time, they still were not aware what was wrong with her and they had tried every cocktail of antipsychotics imaginable: “They gave her enough medicine to fell a cow. But nothing

was working.” After Lori had been at Payne Whitney for two months and had even surrendered to electroshock therapy, the doctors believed they had a better idea of what Lori’s problems were. They soon delivered her diagnosis to her parents, “Because of the combination of her severe mood swings and her hallucinations we think that Lori has something called schizo-affective disorder. She’s got some symptoms of schizophrenia and some symptoms from manic-depression” (Schiller, 1996). Her parents, quite understandably, were devastated by her diagnosis. They had difficulty accepting that she would never get better, so they fiercely contested the doctors’ opinions. One young doctor in the room took it upon himself to fire back at them, “Schizophrenia is a serious illness. It may be a very long time--if ever--before she will get better. She will probably never be able to live on her own again. It would be better for both of you if you faced facts” (Schiller, 1996).

Kurt

Kurt Snyder also had a normal childhood. He tells of his parents’ kindness and encouragement in everything he did, from school to sports. He incites that he did not have many friends his age, but had a few close friends and that was enough for him.

Like the others, Kurt's symptoms arose with the stress of college. He recalls, “everything fell apart during my second semester, when I became disorganized, lost in my concentration during class, and failed to complete many of my homework assignments. I ended the year with very poor grades” (Snyder, 2007). He remembers that during his first year he began to believe that he was going to discover a new mathematical principle that would transform the way we view the universe. Of the experience he says, “I told myself I was always on the verge of discovery, and I hadn’t

discovered the right idea because I just wasn't thinking hard enough. I knew it would take a genius to solve some of the problems I was thinking about, but I didn't realize that I wasn't a genius" (Snyder, 2007). Kurt began to struggle academically, and soon dropped out of college. He planned on starting his own business, but that failed as well. He further established himself as a "professional handyman."

During this stage he began to feel more and more self-conscious, wondering if people were watching him while he worked. He remembers that the paranoia began mildly, meaning that he only *wondered* if his clients were watching him. It would not be until he fell deeper into illness that he actually *believed* they were watching him. He recalls that it was also during this time that he began to fear security cameras, especially the ones found in large megastores, banks, and convenience stores. He elicits, "They made me feel like I was being watched exclusively. As more time passed, I started to get a general feeling that I was being observed wherever I went, by the general public. I felt exposed and self-conscious in every public place--like I was on display at the zoo" (Snyder, 2007). Kurt soon sought the help of a psychiatrist but was unable to allow himself to speak about his paranoia.

Several months later, Kurt got a new job as a maintenance man at a high-security building. This obviously led to more frequent feelings of paranoia, and soon his mind filled with delusions. He attests, "I quickly became convinced that the FBI had hacked my computer, and they were somehow working with the facility to keep me under surveillance. I certainly didn't have any real evidence of this, but evidence isn't required for a delusion" (Snyder, 2007).

Kurt's psychotic break happened quite abruptly. He unravelled quickly. On the way home from his brother's house one Sunday he remembers that he started to think about religion and began to believe that he had "joined the universe on some metaphysical level." He wondered if he was God. On the drive back from his brother's house he also began to believe that FBI agents were following him as well. He soon drove to his place of work, looking for his boss whom he believed was in on an operation with the FBI against him. When he arrived at his place of work, he had an altercation with the security guard on duty and ran away on foot. For there he recounts,

"I called a taxi at a nearby hospital. My plan was to take public transportation back to Maryland from DC, and call my folks to come pick me up. I was highly agitated and suspicious of everyone. When the taxi driver arrived, I gave the driver very specific instructions--I was even suspicious of him. He drove for about 5 miles before turning off the route that I had given him. 'Where are you going?' I asked 'I know a shortcut,' he said. I began to think, 'He must be with Them! He's kidnapping me!' I asked, 'Are you going to kill me?' Not waiting for a response, I bailed out of the cab at the next stoplight" (Snyder, 2007).

After getting out of the cap he began to walk along the highway. Soon his boss found him, as the security guard had notified him that Kurt was frantically looking for him. At this point, Kurt definitely thought that his boss was working against him, and he ran away from him. Kurt's boss called the police and soon sirens were behind Kurt while he was walking along a highway. Quite understandably, the police wanted to know why he was running along the highway and why he left his truck at his work place. Kurt somehow

convinced the police that he was fine and managed to get them to take him to the airport so he could catch a bus at the metro station.

Kurt got on various buses and trains thereafter, attempting to “lose the agents” that he believed were following him. He eventually arrived at a train station in Maryland and called his parents to pick him up. They had already spoken to Kurt’s boss, and were finally fully aware of the extent and severity of his mental illness. They scheduled an appointment with a psychiatrist, but Kurt told him very little about his delusions. This psychiatrist incorrectly diagnosed Kurt with bipolar disorder, a not uncommon occurrence.

An Abnormal Reality: Symptoms

As evidenced from the previous accounts, there is no *single* symptom that is found only in schizophrenia. This makes diagnosis particularly tricky, hence the psychiatrist who incorrectly diagnosed Kurt. The DSM criteria for schizophrenia requires that there are two or more of the following symptoms present for a significant portion of time during a one month period: delusions, hallucinations, disorganized speech, catatonia, restricted affect, and asociality. In this section I will describe the most common symptoms of schizophrenia: alterations of senses, inability to interpret and respond, delusions and hallucinations, changes in emotions, and anosognosia. Positive symptoms are considered those that add to the disease, hallucinations and delusions. Negative symptoms are those that disease takes away, inability to interpret and respond. I will highlight an example of some of these symptoms that were experienced by either Elyn, Lori, or Kurt in order to illustrate how that symptom might appear in an individual context. The descriptions of the symptoms are taken from *Surviving Schizophrenia* by E. Fuller Torrey. Torrey is a psychiatrist who has written numerous books, conducted loads

of studies, and prepared numerous reviews on all things schizophrenia. It is worth mentioning that his sister suffered from illness as well. With that being said, if anyone understands the symptoms of schizophrenia, it is Torrey.

Alterations of the senses

Torrey asserts that alterations of the senses are especially prominent in the early stages of breakdown in schizophrenia, and that they can be found in almost two-thirds of all patients. In some instances visual alterations improve the appearance of something. Sometimes both hearing and visual sensations are increased. This is evidenced by Lori Schiller's onset of schizophrenia, "Everything seemed much nicer than it had been before" (Schiller, 1996).

Closely related to the over acuteness of the senses, is the flooding of the senses with stimuli. It is not only that the senses become more sharply attuned but also that schizophrenics see and hear *everything*. Under normal circumstances our brain screens out most incoming sights and sounds, allowing us to concentrate on whatever we choose. This screening mechanism is impaired in many people with schizophrenia, thereby releasing a flood of sensory stimuli into the brain simultaneously (Torrey, 2013). Kurt experiences this symptom. He recalls in *Me, Myself, and Them*, "I became acutely aware of certain stimuli that normal people don't notice. Noises like fans, people coughing, car horns, machinery rattling, tires screeching, wind blowing, engines running, and birds chirping became very disturbing to me" (Snyder, 2007) On this experience Ely Saks also relates,

"Consider this: The regulator that funnels certain information to you and filters out other information suddenly shuts off. Immediately, every sight, every sound,

every smell coming at you carries equal weight; every thought, feelings, memory, and idea presents itself to you with an equally strong and demanding intensity.

You're receiving a dozen different messages in a dozen different media--phone, e-mail, TV, CD player, a friend knocking on the door, ideas inside your head--and you're unable to choose which ones come to the front and which are regulated to 'later.' It's the crowd at the Super Bowl, and they're all yelling directly at you" (Saks, 2007).

Furthermore, it obviously becomes difficult to concentrate or pay attention when so much sensory data is rushing through the brain. One study reported more than half of the people with schizophrenia reported impairments in attention (Torrey, 2013). Elyn sights this experience in her early days at Oxford, "I could not concentrate on my academic work. I could not understand what I was reading, nor was I able to follow the lectures. And I certainly could not write anything intelligible" (Saks, 2007).

Another aspect of the over acuteness of the senses is a flooding of the mind with thoughts. It is as if the brain is being bombarded both with external stimuli and with internal stimuli. A variation of flooding with thoughts occurs when the person feels that someone is inserting the flood of thoughts into his or her head. This is commonly referred to as thought insertion and when present is considered by many psychiatrists to be almost certainly a symptom of schizophrenia (Torrey, 2013). This phenomenon happened to Kurt just before his first psychotic break,

"My imagination was running wild. I was thinking about how computer systems had become more powerful in the last 50 years. What impossible tasks would become possible in the future thanks to computer technology? I wondered if

computers would be able to send data directly to the brain by some type of neural connection. Could such a computer generate a virtual reality? Maybe I was hooked up to one right now! Maybe my whole world was actually generated by a virtual reality machine connected to my brain” (Snyder, 2007).

However, some patients describe the sensitivity as pleasant in the earliest beginnings of the disease. Many patients interpret their newfound sensitivity within religious framework, that they have been touched by God.

It is worth noting that sensations are not always enhanced. They can be blunted, as well. Blunting often occurs late in the disease and is often described as “a heavy curtain drawn over the mind.” One sensation that can be blunted in some rare cases is pain. Some attribute pain blunting to antipsychotic medication side effects, but the effect has been described long before the advent of these medications (Torrey, 2013).

Inability to Interpret and Respond

Torrey cites that in normal people, the brain functions to sort and interpret stimuli, followed by a correct response being selected and sent out. Most responses are learned and often include basic logic like simply being able to predict outcomes of your actions. A hallmark of schizophrenia is the inability of the brain to sort, interpret, and respond (Torrey, 2013). Many schizophrenics also struggle with putting auditory and visual stimuli together. Recall that not being able to select an appropriate response to stimuli was the symptom that supposedly led Eugen Bleuler to coin the term schizophrenia, as he attributed these odd responses to a split mind (Torrey, 2013).

The schizophrenic thought process is often described as disconnected. The terms loosening of associations, impairment of logic, thought blocking, and ambivalence are often used by psychiatrists. Patients often put together several disconnected ideas that a

normal functioning brain could not come up with. Often schizophrenics make vague connections between their jumbled thoughts, this is what is referred to as loose associations (Torrey, 2013). Kurt's symptoms often took the form of loose associations. For example, Once while sitting on a bench, a large bug landed on a bush near by him, causing him to think about hidden microphones being called "bugs" and about insects being called "bugs." He quickly began to believe that the bug that had landed nearby was a surveillance device designed by the CIA to look like a real insect (Snyder, 2007).

Another uncommon but dramatic form of thinking in schizophrenia is called word salad; the individual just strings together a series of totally unrelated words and pronounces them as a sentence. Elyn Saks's symptoms frequently appeared as word salad. Once when on the phone with one of her therapists she said, "Yes, but it's Sunday. The Sundance kid. Bearded and gray. With all the interferences" (Saks, 2007). Naturally, he knew she was having a psychotic break and he urged her immediately into his office.

Another classic symptom of schizophrenia is flattened affect. Often these individuals seem very aloof and often respond incorrectly or abnormally. A classic example of this comes to my mind via my Aunt Linda. When her siblings called to inform her that their eldest brother had passed away, she simply responded with, "Well, I guess that happens sometimes," and hung up the phone.

Torry asserts that it is an unusual patient who does not have some form of thinking disorder. Some psychiatrist even question whether schizophrenia is the correct diagnosis if the person's thinking patterns are completely normal.

Delusions and Hallucinations

The best known symptoms of schizophrenia are hallucinations and delusions. They are dramatic and are often the behaviors usually focused on when schizophrenia is being represented in popular literature or movies (Torrey, 2013). It should be remembered that these symptoms are not essential to schizophrenia, as there are many schizophrenics who suffer from other symptoms like thought disorder that do not report hallucinations or delusions. These symptoms are also found in other brain diseases aside from schizophrenia; therefore, they are not complete hallmarks of the disease. It is also important to understand that most delusions and hallucinations are distortions of the body boundaries; they are direct outgrowths of over acuteness of the senses and the brain's inability to interpret and respond to stimuli. They are the culmination of the other problems the brain is experiencing.

Delusions are a false idea believed by the patient but not other people in his/her culture and that cannot be corrected by reason. They are usually based on some sort of sensory experience that the person misinterprets. One common delusion is that random events taking place around the person all relate in a direct way to him or her. The sufferer often knows his or her delusions to be true with *absolute* certainty; therefore, trying to reason with them often leads nowhere (Torrey, 2013). Elyn Saks had delusions that she had killed thousands of people, even children, with her thoughts. Kurt Snyder suffered from the common delusion that he was being watched by the FBI. Paranoid delusions, like Kurt's, can sometimes be dangerous, as a paranoid person may try to strike first if they feel a threat. Many delusions are grandiose. This often leads to the person believing he or she is God, Jesus Christ, the President, etc. These delusions can be dangerous but often only to the sufferer, as they might believe they are indestructible. Lori Schiller was

convinced that she had the ability to fly: “I know you’re not going to believe this, Daddy, but I can fly. I can fly. Really, Daddy. I can” (Schiller, 1996). She then began to demonstrate her flying by jumping off sofas and even suggested her father take her to a window to show her new found ability to fly.

Hallucinations are very common in schizophrenia and are at the very end of that spectrum that begins with a slight sensitivity to the senses. In true hallucinations the brain makes up what it hears, sees, feels, smells, or tastes (Torrey, 2013). Auditory hallucinations are the most common form. The hallucinations may be heard occasionally or continuously. In a vast majority of cases, the voice are male and very unpleasant. Visual hallucinations often occur in conjunction with auditory hallucinations. Lori Schiller's hallucinations in *The Quiet Room* absolutely qualify as unpleasant. She recalls that The Voice’s power over her was “too fierce.” She says that there was nothing on the outside that could pull her away. They were the most powerful thing in her world (Schiller, 1996). She asserts that the closest she ever got to hearing a friendly voice was what she referred to as the narrator:

“He described my actions instant by instant, not leaving out even the tiniest, most insignificant thing. A hundred times a day, he commented on my movements.

‘She is now walking through the door. She’s wiping her feet, little ass. Wiping her feet on the rug in the entryway. She’s going into the kitchen. Ha! Ha! You fat piece of lard, of lard. Go to hell. Ha! Ha! You look sad. You look like a shit. You are shit’” (Schiller, 1996).

She tells that sometimes that voices instead joined together in a large congregation, always reminding her that she was going to hell. As Lori's disease progressed she also suffered from visual hallucinations. Again in vivid, bone-chilling detail she remembers,

“But the worst torment in those days was not the things I heard but the things I saw. I saw fire, lightning, colored bolts of light. I saw people hanging in the window, and body parts hanging from the trees. I saw fire around people and walls and faces. Sometimes I felt I had projector eyeballs, shooting things and shapes and colors straight ahead of me. Sometimes I saw things that looked as real as my bed or my lamp or my tennis shoes.

I couldn't sleep at night because of the creatures in my bed. I sat at my desk writing in my journal one night because I was afraid to go near my bed.

‘There are four of them sitting on my bed,’ I wrote. Usually I saw creatures with faces that were like the scariest Halloween mask ever made or creatures with big, blubbery, hairy, slippery green faces” (Schiller, 1996).

Changes in Emotions

According to Torrey, changes in emotions is often one of the most common characteristics. In the early stages of the illness, depression, guilt, fear, and rapidly fluctuating emotions may all be found. In the later stages, flattening of emotions are more common, often resulting in people who cannot feel emotions at all. Elyn, Lori, and Kurt all experienced very deep depression and fear at the onset of their illness. Often many schizophrenics have difficulties in assessing emotions in other people. The most characteristic changes in emotions in schizophrenia are inappropriate or flattened emotions (Torrey, 2013).

Anosognosia

Some people with schizophrenia are aware of the malfunctioning of their brain; this is called awareness of illness or insight. As the disease becomes fully manifest this awareness is lost. This is often *why* patients stop taking their medication. They begin medication, start feeling better, then stop believing that they are sick, like this illness is some kind of flu virus. They are certain they are fine, when in reality the medication is lifting the fog of psychosis. It took Elyn Saks a very long time to understand this: “If I’d had a broken leg and a crutch was required, I’d have used it without ever thinking twice. Was my brain not worth tending to at least as much as my leg? The fact was, I had a condition that required medicine. If I didn’t use it, I got sick; if I used it, I got better. I don’t know why I had to keep learning that the hard way, but I did” (Saks, 2007).

The Method Behind the Madness: Theories of Etiology

The etiology of cancer is well understood. It is, with a few exceptions, a disease of old age. The etiology of schizophrenia, on the other hand, has been a minefield of discovery. First, genetics were believed to be the sole cause. Scientists once, almost laughably, believed that a single gene could be responsible for such a complicated disease. Next, came the neuropathology hypotheses, then neurotransmitter theories, then developmental theories, then bacterial theories, and so on. Finally, scientists have reasoned that the rise of schizophrenia is most likely the culmination of all the implications of these theories working in combination with environmental variables to create this bemusing illness that has eluded scientists for centuries. To put it simply, the etiology of schizophrenia is complicated. Next, I will only scratch the surface while summarizing the genetic, neurochemical, pathological, developmental, and infectious properties of the disease.

Genetic Theories

While there is a lot of dispute as to the number of genes involved in this process and their subsequent effects, there is little to no doubt that there is a genetic component of schizophrenia. Relatives of patients with schizophrenia have a 5-10 times increased risk for developing schizophrenia compared with the general population. Heritability for schizophrenia has been estimated at 80%, and linkage or gene-disease associated studies have found some loci that are likely associated with schizophrenia on chromosomes 1, 2, 3, 5, 6, 8, 10, 11, 13, 14, 20, and 22 (that is 12/23 chromosomes). Data from association and linkage studies have confirmed that schizophrenia is NOT caused by a single gene. Instead it is what geneticists refer to as polygenic. Each gene responsible has a small effect, and can even have different or contrasting association results (Olgiati et al., 2009).

Endophenotypes have emerged as promising targets of psychiatric genetics. The term endophenotype is currently used in psychiatric research to define a measurable biological or psychological trait that is common to both individuals with a disease and their non-affected relatives and that may predispose to illness onset (Olgiati et al., 2009). Researchers Olgiati et al. defined an endotype as a neuropsychological or neurophysiological character associated with schizophrenia. They are heritable, stable, and trait related. These researchers report that schizophrenia-related endophenotypes are cognitive functions such as attention, working memory, executive functions, and visuospatial memory that are impaired in schizophrenic patients and that can also be impaired in their non-schizophrenic relatives. Below are descriptions of the most common genes associated with schizophrenia: COMT, Neuregulin 1, DISC-1, DTNBP1,

and GRM3. The effects of these genes and their endophenotypes are described for both schizophrenic individuals and their non affected relatives.

Catechol-O-methyltransferase (COMT)

This enzyme terminates catecholamine activity in the brain by degrading neurotransmitters. The gene that encodes COMT has a functional polymorphism (Val 108/158 Met) that moderates dopamine availability in the prefrontal cortex in an allele dependent manner. The Met allele, which has 3-4 times lower enzymatic activity, has been associated to higher dopamine levels in the prefrontal cortex (the region of the brain most associated with planning complex cognitive behavior). There have been a number of studies that have indicated this COMT polymorphisms as a risk factor for schizophrenia. The Val-allele has higher enzymatic activity, resulting in lower prefrontal cortex dopamine levels (Olgiati et al., 2009). The Met-allele has lower enzymatic levels, resulting in higher dopamine levels. Altered dopamine levels have long been associated with the pathology of schizophrenia and can also have effect on non affected relatives.

Neuregulin 1 (NRG1)

The neuregulins (NRGs) are cell-cell signalling proteins that are ligands for receptor tyrosine kinase family (proteins that regulate normal cellular processes) of the ErbB family (improper signalling in this family has been associated with the onset of many neurological disorders). NRG1 proteins have been shown to play important roles during the development of the nervous system. Therefore, these findings help support NRG1's involvement in the pathogenesis of schizophrenia (Olgiati et al., 2009).

Disrupted-In-Schizophrenia 1 (DISC-1)

This gene is disrupted by a balanced translocation that has been shown to cosegregate with major psychiatric disorders. The DISC-1 protein occurs in various

subcellular compartments, including the centrosome, microtubules, mitochondria, and the nucleus. Recent studies have established that DISC1 is a component of a neurodevelopmentally regulated protein complex that has different functions in the developing and the adult brain. In the developing brain, DISC1 has been implicated in neuronal migration and in outgrowth and extension. In the adult brain, DISC1 has been identified in multiple neuron populations, particularly in structures that specialize in synaptic function (Olgiati et al., 2009). DISC1 is also present in many of the brain regions that are known to be abnormal in schizophrenia--the prefrontal cortex, hippocampus, and thalamus. Mutant DISC1 may contribute to schizophrenia susceptibility by altering neuronal architecture and migration. DISC1 single nucleotide polymorphisms have been shown to be connected with sustained attention and working memory deficits in schizophrenic families (Olgiati et al., 2009).

DTNBPI

Dysbindin is an evolutionarily conserved gene. An evolutionarily conserved gene or protein has remained essentially unchanged throughout evolution. Conservation of the gene indicates that it is unique and essential: there is not an extra copy of that gene and changes in such a gene are likely to be lethal. Significant dysbindin reductions have been found at presynaptic levels in glutamatergic afferents of the several brain regions such as the hippocampus in schizophrenic brains. Sequence variations in *DNTBPI* determine reductions in dysbindin messenger RNA levels in the prefrontal cortex in schizophrenics and mediate the risk of schizophrenia by lowering dysbindin expression. In samples of schizophrenics, carriers of the *DNTBPI* have been associated to early visual processing deficits and a significantly lower spatial working memory performance. Genetic variation

of *DNTBPI* within carriers has also been shown to influence general cognitive ability (Olgiati et al., 2009).

GRM3

A large body of evidence supports the involvement of the glutamate system (a fast-signaling system that is very important for information processing in neuronal networks of the neocortex and hippocampus in particular. Glutamate is also very much involved in the process of long-term potentiation, which is a neuronal model of memory) in schizophrenia (Olgiati et al., 2009). For example, glutamatergic NMDA receptor antagonists (often drugs used to induce and maintain anesthesia) can cause psychotic symptoms in healthy people and exacerbate psychosis in schizophrenics. *GRM3* is a metabotropic glutamate receptor modulating synaptic glutamate, and it has also emerged as a promising candidate gene for the onset of schizophrenia. Variations in the gene mediate glutamate release in the prefrontal cortex and aid cognitive functioning in psychotic patients (Olgiati et al., 2009).

Epigenetics

Epigenetics was born out of the terms “genetics” and “epigenesis”--referring to the study of causal relationships between genes and their phenotypic effects, but more recently associated with the changes in gene activity independent of the DNA sequence (Petronis, Paterson, & Kennedy, 1999). Often these modifications are a direct result of environmental influences: obstetric complications, viral infections, stressful life events, and substance abuse. It has been very clearly established that this gene-environment interplay is critical for the onset of psychosis.

The fundamental difference between epigenetic systems and DNA sequence-based heredity factors lies in the ability of epigenetic systems to change rapidly in comparison to DNA sequence-based heredity. Epigenetic factors may only exhibit partial stability when transmitted from parent to daughter cells, while DNA sequence demonstrates nearly complete inheritance (Petronis, Paterson, & Kennedy, 1999). For example, DNA methylation patterns undergo major reorganization during gametogenesis, development, and aging. Numerous cellular functions are controlled and mediated by DNA methylation: the regulation of gene activity, genomic imprinting, and genetic recombination. In mammals, DNA methylation patterns are subjected to major changes due to environmental influences. Therefore, epigenetic mechanisms such as methylation have the potential to cause subtle changes in brain development such as those observed in schizophrenia (Petronis, Paterson, & Kennedy, 1999).

Neurotransmitter Theories

Neurochemical theories for schizophrenia have been prominent for quite some time. Many of these theories evolved in response to observed effects of antipsychotics, antidepressants, or psychotomimetics such as amphetamines, and can now be tested by examining gene, RNA, and protein expression (Torrey et al., 2005). Although schizophrenia was once seen as a disease affecting only a few key brain regions and regionally discrete neurotransmitter systems such as dopamine, more recent findings implicate widespread cortical and subcortical dysfunction (Javitt, 2010). While originally thought to be independent of one another, it is now evident that these neurotransmitter systems affect each other and are very much interdependent.

GABA

A 2005 study done by Torrey et al. sought to confirm neurochemical markers in schizophrenia. To conduct the study, Torrey and his team examined post-mortem brains, looking for abnormalities in neurochemical markers. They found that twenty-three neurochemical markers (23% of the total) were significantly abnormal in one or more brain region. As a percentage of the total markers included in this study, the developmental/synaptic (10/22 were found to be abnormal) and GABA (3/7 were found to be abnormal) neurochemical systems had the most abnormalities.

The major finding to emerge from this study is the involvement of the GABA system in the pathophysiology of schizophrenia (Torrey et al., 2005). GABA is the major inhibitory neurotransmitter in mammalian brains and used by approximately 40% of all neurons. Abnormalities in the GABA system in schizophrenia have been identified in the GABA transporter, receptors, and in neuron density. The GABAergic system is also thought to interact with the dopamine system and with the excitatory glutamatergic system (Torrey et al., 2005).

Perhaps the most interesting finding is that almost all abnormalities represent decreased expression compared with the unaffected control subjects. The direction of such findings suggests a widespread failure of gene expression in the major psychiatric disorders, leading to malfunctioning neurotransmitter systems, as evidenced by these findings on GABA (Torrey et al., 2005).

Dopamine

Dopamine is a neurotransmitter produced in the substantia nigra, a portion of the midbrain that has an important role in reward, addiction, and movement. Dopamine alterations are related to schizophrenia. The “original dopamine hypothesis” states that

hyperactive dopamine transmission results in schizophrenic symptoms. The positive symptoms of schizophrenia include hallucinations and delusions as a result of increased subcortical release of dopamine, which augments D2 receptor activation, resulting in these over perceived sensations (Brisch et al., 2014).

There is ongoing discussion regarding the involvement of D1 and D2 receptors in cognition in schizophrenia patients. Cognitive discrepancies and working memory deficits in the prefrontal cortex are associated with an increase in dopamine and D1 receptors in the prefrontal cortex in schizophrenia patients (Brisch et al., 2014).

Glutamate

While dopaminergic models of schizophrenia take only positive symptoms into account, glutamatergic models help to account for the negative and cognitive symptoms (Javitt, 2010). The first alternative to the dopamine hypothesis was first proposed in the early 1990s, based on the observation that ketamine-like compounds induced unique behavioral effects by blocking neurotransmission at N-methyl-D-aspartate (NMDA)-type glutamate receptors. The ability of these compounds to imitate key symptoms in schizophrenia by blocking NMDA receptors led to the concept that symptoms in schizophrenia may reflect underlying dysfunction or dysregulation of NMDA receptor-mediated neurotransmission (Javitt, 2010).

Interestingly, when patients with schizophrenia are exposed to ketamine, they also show increases in positive symptoms, as well as negative symptoms, suggesting that NMDA antagonists affect a brain system that is already vulnerable in schizophrenia. As opposed to ketamine, administration of dopamine agonists such as amphetamine does not reproduce the pattern of deficit observed in schizophrenia. New treatment approaches

based upon glutamatergic approaches are only now reaching the clinical stages and will serve to further elucidate and refine these models over upcoming years (Javitt, 2010).

Neuropathological Theories

Finding the neuropathology of schizophrenia has been one of the major, failed quests of biological psychiatry for over 100 years. In fact, one American neurologist called the disease the “graveyard of neuropathologies” (Harrison, 2008). Two early researchers, Alzheimer and Southard, were the first to describe any neuropathological changes in the brain’s cortex in the late nineteenth century. They found volume loss of the cortical gray matter, different vulnerabilities of cortical neurons toward injury, low glial to neuron ratios, and abnormalities in the position of neurons within the cortical layers. Interestingly, many of these original findings are still being investigated (Heckers, 1997). Even so, a major challenge of neuropathological studies that examine postmortem tissue is controlling for the tissue changes after death, particularly brain shrinkage. This tissue shrinkage creates issues in the two most-studied parameters, brain volume and cell density (Heckers, 1997).

More modern neuropathological techniques have been made possible thanks to brain imaging. MRIs now show clearly that there are structural brain differences in living patients with schizophrenia. Neuropathological alterations are present in patients experiencing their first episode, and some also in at-risk subjects, indicating that there is pathology that cannot be explained as simply an artifact of the chronic illness itself or antipsychotic medication (Harrison, 2008). There are two important findings from neuropathological studies done thus far. First is that the neuropathology of schizophrenia is not neurodegenerative, as there are no structures that indicate such findings. Next, is

that although early studies have often made the claim, there is no gliosis (proliferation of the supporting cells in the brain) in schizophrenia. Gliosis is a sign of inflammation, injury, etc.; therefore, the lack of gliosis is often taken as a sign that the disorder is likely to be neurodevelopmental in origin (Harrison, 2008).

There are many studies that have attempted to examine the neuropathology of schizophrenia. Unfortunately, only one theme has emerged from them: the inability to replicate. Nevertheless, there are a few promising studies that have found an increased packing density of neurons within the cerebral cortex (Harrison, 2008). This finding has contributed to the understanding of schizophrenia as a disease of neural connections. Many studies in neuropathology of schizophrenia have found a decreased size of pyramidal neurons. The size of a neuron is related to the volume of axons and dendrites that it can support--therefore its synaptic activity. Therefore, schizophrenic neurons are making fewer or less active connections within the brain (Harrison, 2008). The thalamus, a collection of nuclei that serve as the primary relay station to the cerebral cortex is also an area of particular interest, and it has been found to be smaller and contain less neurons in several schizophrenia studies. Conclusively, there is *some* evidence for a neuropathology of schizophrenia; however, the details of these changes remain elusive.

Developmental Theories

There are several lines of evidence that indicate early brain development in the onset of schizophrenia. This evidence is consistent with the view that the neurodevelopment of schizophrenia is partly present from birth and may therefore explain motor language, and social affective delays and other deficits in neurocognitive functioning in children who later develop schizophrenia (Cannon et al., 2003).

The timing of certain environmental factors linked to increased risk for schizophrenia does provide perhaps the most direct evidence to date that early neurodevelopmental disturbances are involved. For example, prenatal complications like hypoxia (deficiency in amount of oxygen in tissues) appears to be a risk factor in about 30-40% of pregnancy complications. Therefore, complications that are associated with fetal hypoxia are of particular interest, as they represent a very plausible mechanism for explaining many issues that are found in adult neuropathology (Cannon et al., 2003).

In addition to issues that arise because of hypoxia, there is now sufficient evidence from both animal and human models that have implicated influenza during gestation specifically as a concern for the increased risk of several psychological disorders, including schizophrenia. In fact, one study that examined schizophrenics born between 1959 and 1966 found that the presence of maternal antibodies for influenza during pregnancy created a four to seven fold increase in risk for psychiatric diseases (Short et al., 2010). Unsurprisingly, concern arose from this study and experiments replicated on animal models followed. Researchers soon found that decreased cortical thickness and smaller hippocampal volumes in the brains of rodents occurred when they were exposed to the influenza virus (Short et al., 2010).

Microarray (method to detect DNA or RNA that can potentially be used to create proteins) analysis from the rodent models have helped to indicate that these neuropathological differences might be caused by both up- and downregulation of genes for the structural proteins such as reelin--a protein known for its importance in neural migration and several other important neural processes. The study conducted by Short et al. sought to determine whether infection in late pregnancy would result in

persistent brain changes that are large enough to be viewed with neuroimaging techniques (MRIs) in rhesus monkeys. Rhesus monkeys were chosen as a model to investigate given the similarities between their placental functioning in relation to humans.

The results from this study indicated that even in the absence of an effect on gestation length and birth weight (controlling for the rodent models), prenatal infection can significantly reduce brain size in the young monkeys (Short et al., 2010). Based on these results, the researchers inferred that these alterations would persist, as reduced volumes are evident at a stage in the monkeys that are equivalent to late-childhood in humans. The nature and extent of the brain volume reductions seen in the monkey bore the most common with the structural abnormalities frequently found in schizophrenics, indicating that exposure to influenza virus in gestation can predispose one to schizophrenia (Short et al., 2010).

Infectious Theories

There are several aspects of psychotic disorders that suggest a possible infectious origin. One is the observation that people who experience microbial diseases often express the symptoms of schizophrenia and other psychological disorders such as bipolar disorder and depression (Torrey & Yoken, 2008). There is a long list of infectious agents that have been suspected culprits, including: the bacteria that causes syphilis, the bacteria known for causing Lyme disease, the herpes virus, measles, mumps, rubella, polio, retroviruses such as HIV, and the protozoan *T. gondii*. Nevertheless, when one considers epidemiological aspects many of these candidates become very unlikely suspects. For example, schizophrenia like symptoms have been described for over 200 years, but HIV has only recently infected human populations, ruling it out as a probable cause (Torrey & Yoken, 2008).

One particularly interesting epidemiological aspect of schizophrenia is that there has been a seasonal birth predominance in the winter and spring months. Interestingly, over 200 studies have demonstrated this finding, and it is actually the most consistently replicated findings in all of schizophrenia research. If an infectious agent is responsible for the seasonality, it would be one that occurs throughout the year but that has a modest seasonal predominance. The infectious agent would also be one that does not vary in its occurrence from year to year--the flu virus, perhaps? (Torrey & Yoken, 2008). Another epidemiological factor that must be considered is that being born and raised in urban environments compared to rural environments can sometimes double the risk of later being diagnosed with schizophrenia. A dose-response relationship has been proposed in that the longer one lives in an urban environment, the higher degree of risk they adopt (Torrey & Yoken, 2008). The exact mechanism is, of course, not fully understood; however, the greater population densities found in cities is often associated with the transmission of a wide array of microbes.

Toxoplasma

T. gondii or toxoplasma is a protozoan whose definitive hosts are felines, particularly the domestic cat. Toxoplasma can only complete the sexual part of its lifecycle within the feline host. Toxoplasma oocysts (a cyst containing a zygote formed by a parasitic protozoan) are excreted through feline feces. The oocysts may then be released into the air to infect humans who might be changing the litter box, gardening, etc. Cat feces are also often found in the ground of or in animal feed troughs of barns, domestic animals then eat it, the oocysts are produced in their muscles, thus later affecting humans who might consume undercooked meat (Torrey & Yoken, 2008). Feline

feces can potentially contaminate water supply as well. Toxoplasma is found world wide as has been found to affect anywhere from 10-80% of the adult population, with about 25% of adults projected to be infected in the United States.

Toxoplasma is a known neurotrophic--infecting both neurons and glia. Until recently there was little to no understanding of the molecular biology behind toxoplasma's mechanisms of infection. However, protozoan genomes have since been sequenced and an increased understanding of toxoplasma infections has followed. Researchers have recently discovered that toxoplasma's protein kinases (proteins involved in cellular signaling) alter signal transductions in infected hosts, compromising immune function (Torrey & Yoken, 2008).

One might be wondering: why would a microbial agent lead to the altered behavior that is present in schizophrenia? To answer simply, toxoplasma alters the host behavior in order to enhance its own survival and reproductive success. Mechanistically, felines are the natural hosts for toxoplasma; therefore, if toxoplasma enters a cat, it can fulfill its entire lifecycle. However, if toxoplasma infects another animal, toxoplasma faces a problem, as it cannot complete its lifecycle in any other animals besides felines. Nevertheless, if toxoplasma can get its host to be eaten by a feline, it will be able to complete its lifecycle within its feline host (Torrey & Yoken, 2008).

All felines are carnivores; therefore, the possibility that another animal will be eaten by them is conceivable, especially if said animal is a small rodent. Recent research has uncovered that toxoplasma is successful at altering small rodent behavior, ensuring that these small rodents lose their inherent fear of cats. Therefore, this adaptation of the parasite ensures that the hosts are consuming more rodents infected by toxoplasma,

allowing toxoplasma to infect its proper host and complete its lifecycle (Torrey & Yoken, 2008).

You might be thinking: humans are not normally consumed by domestic cats. This means that toxoplasma effects seem to be vestigial in humans. However, two studies have demonstrated that young adults that are infected by toxoplasma have an increased rate of automobile accidents, a behavior very much associated with risk (Torrey & Yoken, 2008). True to most research on schizophrenia, the exact mechanisms as to how toxoplasma creates such psychosis are currently unknown. However, researchers believe that one possibility is the direct effect of the organism on neurons and glial cells cited earlier. As of yet, it is known that toxoplasma may affect signal transduction pathways and that it also encodes proteins that might interfere with the synthesis of dopamine in human hosts, a known cause of schizophrenia symptoms (Torrey & Yoken, 2008).

Each respected theory of causation has its strengths and weaknesses. While I have presented each separately, for organizational purposes. They should be taken together to create a holistic model of causation. In addition, every theory may not apply to one individual. For example, there are plenty of schizophrenics that are not infected with toxoplasma. When taking all of the respected theories into account, one wonders how scientists could have even dreamed to believe that a single gene was the culprit of the illness.

An Enigmatic Legacy: Evolutionary Theories of the Schizophrenia

A textbook on evolutionary psychology says that the evolutionary approach to mental illness is characterized by a variety of attempts to classify and explain the disorders in terms of the fundamental architecture of the brain as laid down by natural and sexual selection. The textbook goes on to summarize that since it's inception,

evolutionary psychology has stressed that the cognitive and emotional systems we possess, were designed to cope with problems that are much different from those present in the modern world. According to Darwin's *Origin of Species*, the process of natural selection preserves genetic variants associated with survival and reproductive advantage, while genetic variants associated with low fitness are eliminated from the gene pool. Therefore, why has natural selection not eliminated genes that might predispose one to psychiatric disorders like schizophrenia? This question has been addressed by many and is commonly known as the evolutionary paradox of psychiatric disorders. In relation to schizophrenia, those affected show lower reproductive success and suffer from greater mortality from both natural and unnatural causes like suicide than the greater population. Therefore, why has the disorder remained prevalent, consistently affecting about 1% of the world's population (van Dongen & Boomsma, 2013)? As I previously mentioned, it was this question that gave me the inspiration to write this thesis, and I found its explanations just, if not more, fascinating than the question posed. Next, I will review some of the theories that have been suggested, taken from a recent review by van Dongen and Boomsma, *The Evolutionary Paradox and the Missing Heritability of Schizophrenia*.

First the researchers cite a study entitled *Schizotypy, Creativity, and Mating Success in Humans* by Daniel Nettle and Helen Clegg. This study first notes that many prior studies have found a relationship between those active in the creative arts and schizotypal traits. The authors pose, why would artistic creativity contribute to reproductive success? They note that some researchers have argued that human artistic qualities have origins in the costly displays of quality. Therefore, the function of such qualities is similar to that of the quintessential peacock's tail (Nettle & Clegg, 2005).

Nettle and Clegg's study examined the relationship between schizotypy and mating success in a large sample of British adults.

The results of the study showed a significant positive relationship between unusual experiences and mating success. Nettle and Clegg hypothesize that in the case of unusual experiences, the relationship is mediated by creative activity; those high in unusual experiences produce poetry or art more seriously, and this in turn increases mating success. They found that increasing this trait lead directly to an increased number of sexual partners. Therefore, these results are consistent with the view that schizotypal traits are maintained in the human population at significant levels because the negative effects in terms of psychosis are offset by enhanced mating success. The study notes that first degree relatives of schizophrenics are often "strikingly creative individuals."

Therefore, this theory asserts that mating success given from schizotypy traits like creativeness could be the reason that schizophrenia has persisted. Although I am a second degree relative to my schizophrenic Aunt Linda, I would dare say that I have been given my fair share of creative traits, and I can attest that they did not come from my mother or father. Nevertheless, I am still waiting for them to land me a mate... Therefore, van Dongen and Boomsma stress that this explanation is not enough to explain the persistence of the genes associated with schizophrenia over the course of evolutionary history.

Still, others have suggested that schizophrenia may have arisen as an unfavorable but inevitable byproduct of the evolution of the human brain. Therefore, schizophrenia would represent an extreme end of the normal variation of cognition (van Dongen & Boomsma, 2013). It has been proposed that schizophrenia could be regarded as an outlier

on a normal continuum of social behavior and possibly even the toll that humans pay for the benefit that social skills have given. Another researcher proposed the link between schizophrenia, language dysfunction, and cerebral flexibility to hypothesize that schizophrenia reflects the extreme end of variation underlying language capacity. In other words, schizophrenia is the price we pay for being able to communicate. According to this theory, positive selection on cerebral flexibility was the associated variation in psychological functioning, resulting in schizophrenia at the extreme end of the spectrum (van Dongen & Boomsma, 2013).

Another important aspect of human brain evolution is the role of neural connections. According to the researcher Randall, the random establishment of novel pathways within the brain throughout its development can produce advantageous connections. However, in what some would call biological trial and error, that brain has mistakenly given rise to a range of behavioral variants like schizophrenia. Nonetheless, one must reason that if schizophrenia exists as a byproduct of extreme traits, the question as to why genetic variants that cause these extremes are maintained still exists (van Dongen & Boomsma, 2013).

Balancing selection refers to a situation where multiple alleles may be maintained in the gene pool if the genotypes are under different selection pressures. One example of balancing selection is presented by antagonistic pleiotropy, where the effect of the gene is associated with both advantageous traits and disadvantageous traits within the same person, making the gene neutral with respect to natural selection (van Dongen & Boomsma, 2013). Could this be the case in schizophrenia?

Another example of balancing selection is presented by polymorphisms, a topic already discussed in the genetic section. It has been proposed that genetic variants that predispose to fitness-reducing psychiatric disorders in homozygotes are maintained in the population because they are associated with a fitness-increasing trait in a number of carriers. This effect is evidenced by the COMT gene polymorphism. The valine allele is associated with lower dopamine levels, and the methionine allele is associated with higher dopamine levels. Some researchers believe that schizophrenia risk alleles could be maintained in the population because they provide beneficial cognitive or somatic traits in unaffected carriers of these alleles. For example, a met/val carrier might experience more reward because of an increase in dopamine. While schizophrenia may be present in homozygous (met/met) individuals. Heterozygous (met/val) carriers might experience superior social skills, creativity, and cognition. Nevertheless, most studies indicate that the fertility of relatives is not sufficient enough to outweigh the immense reproductive cost that schizophrenia inflicts on a population (van Dongen & Boomsma, 2013).

Another hypothesis along the same lines is called cliff-edged fitness. It refers to the increase in fitness associated with increased expression of a trait up to a certain point, above this point and increased expression is associated with a sharp drop in fitness (van Dongen & Boomsma, 2013). Are schizophrenia traits beneficial until a certain point where the illness sets in? The cliff-edged theory is not perfect, as it still does not make clear why natural selection would maintain harmful alleles that have the potential to lead to schizophrenia.

From an evolutionary perspective, all phenotypes can be regarded as compromises. Evolution does not strive for perfection. Rather, it drives traits towards an

optimum level where fitness and trade-offs are balanced. Therefore, the question arises: can schizophrenia be eliminated by natural selection without also losing valuable cognitive traits along with it? (van Dongen & Boomsma, 2013).

Another hypothesis proposed is called the triune brain concept. It is a model in which the human brain contains the evolutionary remnants of three ancestral brains: the reptilian brain (upper brain stem), the paleomammalian brain (limbic area), and the neomammalian brain (cortical region). According to this model, each successive brain area that was introduced incorporates and modifies previous functions. The researcher Millar proposed that the introduction of each successive brain feature may have come with difficulties in connecting pre-existing areas to novel parts. Millar hypothesized that schizophrenia may reflect a failure of integration between different parts. Nevertheless, the benefits of having a more complex brain have allowed for novel functions such as language, thus outweighing the disadvantage of a design prone to maladaptive error (van Dongen & Boomsma, 2013). But the question remains, why do errors in brain development only lead to problems in some individuals but not everyone? (van Dongen & Boomsma, 2013).

Schizophrenia has been suggested to represent an adaptively programmed phenotype that is induced by environmental adversity. Many organisms express variable morphologies in response to variable environmental conditions encountered during development, many of which are thought to represent alternative survival or reproductive strategies (van Dongen & Boomsma, 2013). The phenotypic plasticity hypothesis states that exposure to adverse environmental cues during early development may induce alterations in the expression of genes, resulting in a phenotype that is better suited for a

stressful or deprived environment, some of the core symptoms of schizophrenia that predict social and vocational disabilities in modern times, such as the inability to calm instinctual drives, ignore arousing stimuli, and inhibit transient desires may represent a “defensive, vigilance-based behavioral strategy that alerts the organism to salient, potentially informative stimuli and permits it to be more impulsive and vigilant” (van Dongen & Boomsma, 2013). Therefore, schizophrenia must be related to physiological and behavioral characteristics that created a fitness advantage in our ancient ancestral environment under conditions of nutritional scarcity and severe environmental stress. What's more, schizophrenia is linked to stress exposure during development.

The model of genetically inherited epigenetic variation may explain how the persistence of disadvantageous traits like schizophrenia may be stimulated by the pressures of variable environments, and at the same time have a heritable basis (van Dongen & Boomsma, 2013).

For example, sexual conflict arises when the two sexes of a species have conflicting optimal reproductive strategies, leading to an evolutionary arms race between males and females. In mammals, the mother is predominantly responsible for providing resources to offspring pre- and perinatally. As a result, the fitness of maternally derived alleles favors smaller demand on maternal resources, anticipating the survival of future offspring. Paternally derived alleles are associated with high fitness if offspring exploit as many resources from the mother as possible (van Dongen & Boomsma, 2013).

Imprinted genes or genes whose expression is determined by the parent that contributed them, the expression pattern of an allele depends on its parent of origin. Typically, one allele is expressed, while the other is transcriptionally silent. The kinship

theory of imprinting states that the evolution of imprinted gene expression originates from the conflict of interests between maternally and paternally derived alleles (van Dongen & Boomsma, 2013). Paternally derived alleles favor higher growth rates of offspring and greater demands on maternal resources than maternally derived alleles. Therefore, growth promoting loci are often maternally silenced through imprinting, whereas loci that suppress growth are often paternally silenced (van Dongen & Boomsma, 2013).

Researchers Badcock and Crespi suggest that the “genetic war” at imprinted genes for brain development may give rise to mental disorders if expression is pushed too far towards the benefit of one parental alleles. Paternally biased expression of genes involved in brain development may give rise to a self-oriented child that is highly demanding to its mother-- extreme cases of this behavior are cited as autism. Badock and Crespi hypothesized that small deviations in imprinted gene expression towards a maternal bias may lead to offspring that are energetically “cheaper” and easier behaviorally to mothers, less demanding and better capable of interpreting and understanding the mental states of others. As such, large maternally biased deviations may lead to psychosis. Badock and Crespi also suggest that several characteristics of autism and psychosis may be regarded as opposites in the context of parental demand. Autism spectrum conditions are characterized by deficits in theory of mind skills, whereas psychotic spectrum conditions involve the opposite: “hyper mentalism.” For example, autistics are characterized by a defective detection of gaze and inability to appreciate what goes on in groups, while individuals with schizophrenia may experience

paranoid delusions of conspiracies and being watched by others (van Dongen & Boomsma, 2013).

Therefore, fluctuations in imprinted gene expression that result from the ongoing conflict between reproductive strategies of males and females may contribute to the persistence of fitness decreasing conditions such as schizophrenia. Because epigenetic mechanisms that regulate imprinting can be influenced by genetic variation, this theory is compatible with the persistence of heritable variation (van Dongen & Boomsma, 2013). Furthermore, the genomic conflicts that arise can cause natural selection to drive phenotypes away from their optimum values, resulting in a maladaptive, but selectively favored evolutionary trajectory. Therefore, mental disorders that occur at high frequencies despite reducing individual fitness, such as schizophrenia, may be related to effects of imprinted gene expression in the brain (van Dongen & Boomsma, 2013). While many of the hypotheses presented are both interesting and logical, most fail to account for the genetic component of natural selection. Therefore, the hypothesis of imprinted genes is the strongest presented, as it gives an explanation for the heritability of schizophrenia.

The Battle Behind the Forehead: Treatment Strategies

From the vast array of theories that attempt to explain why schizophrenia exists and persists, I hope that I have left little doubt that the disease is complicated. Therefore, it should come as no surprise that treating the disease is, well, complicated. Nevertheless, recovery is no longer viewed as impossible, but it is now expected. In 2004 a major attempt at creating a working definition for what recovery from schizophrenia looks like happened at conference held by the Center for Mental Health Services Administration, a division of the US Department of Health and Human Services. While at the conference

family members, administrators, mental health professionals, and recovering schizophrenics agreed on a national consensus statement on mental health recovery: “Mental health recovery is a journey of healing and transformation enabling a person with a mental health problem to live a meaningful life in a community of his or her choice while striving to achieve his or her full potential.” Participants at the conference also agreed on the “The 10 Fundamental Components of Recovery.” They are as follows: self direction, the recovery process must be self-directed by the individual who designs his or her own life goals; individualized, there are multiple pathways to recovery based on an individual's own unique strengths and weaknesses; empowerment, individuals have the ability to speak for themselves about their needs and desires; holistic, recovery encompasses an individual's whole life--mind, body, spirit, and community; nonlinear, recovery is not a step-by-step process but one that is based on continual growth with some setbacks along the way; strength based, recovery focuses on valuing and building on the multiple capacities of an individual; peer support, sharing of knowledge and skills; respect, trying to eliminate discrimination and stigma; responsibility, taking steps towards one's own goals; and finally hope, ensuring a message of a better future (Frese et al., 2009). Today's recovery model is more effective than ever before. I will go over a few of the aspects of this model in detail, focusing primarily on antipsychotics and some varieties of psychosocial therapies. Finally, I will address the idea of early intervention, and how this model strives to bring better treatment results.

Antipsychotics

Upon diagnosis with schizophrenia, one will undoubtedly begin a routine of antipsychotics. Antipsychotics treat the psychosis of schizophrenia--delusions,

hallucinations, and disordered thought. Although these drugs are effective, they are undoubtedly controversial due to their often debilitating side effects. Elyn Saks loathed taking antipsychotics because of their side effects, she complains in *The Center Cannot Hold*, “Soaked in antipsychotic medication, with the psychosis actually clearing, I became profoundly depressed, and felt the brief flash of energy and focus leak right out of me. Suddenly, I couldn’t follow the simplest sitcom on the unit TV, or decipher the lines in a book I’d been reading days before... It’s not that I wasn’t trying--I just couldn’t function” (Saks, 2007). While antipsychotics available today are very helpful, they are far from perfect. There is still a long way to go in the way of research.

The first generation of antipsychotics was discovered in the 1950s by accident as a surgical anesthetic in France. Forty years later the second generation of antipsychotics became available. The first generation of antipsychotics are now referred to as typical antipsychotics. They work by antagonising dopamine receptors and are known for their side effects that cause the sensation that was just described by Elyn Saks. The D2 receptor is often referred to as the antipsychotic receptor. The second generation of antipsychotics gained populace in the 1990s for their novel addition of serotonin antagonism (Fitzpatrick, 2012). Antagonism of serotonin regulates dopamine release in the striatal, pituitary, and neocortical regions of the brain. This mechanism counterbalances the depletion of dopamine caused by typical antipsychotics. As antipsychotics go on and off the D2 receptor rapidly, DA is allowed to bind and release from the same receptors. This dynamic process seems to explain how atypicals are effective in producing an antipsychotic effect without the severe side effects of typical antipsychotics (Fitzpatrick 2012). Dr. Ted Bitner, an assistant professor of psychology at

DePauw University and practicing clinical psychologist, began practicing in 1991. Therefore, he has seen first hand the difference in treatment between typical and atypical antipsychotics, “The first generation didn’t allow any kind of life outside of the home or the hospital. Remember that at the end of the 1980s, we were just beginning to adjust to no psychiatric hospitals, so many of these people became homeless. But when the 2nd generation came along, people were given less restricted environments” (personal communication, April 7, 2016).

Some popular antipsychotics available include: aripiprazole, olanzapine, quetiapine, ziprasidone, and clozapine. Olanzapine, whose brand name is Zyprexa, was created by Eli Lilly, and was the first antipsychotic known for its side effect of serious weight gain. While these drugs might be effective for some, clozapine is considered the gold standard in efficiency for all schizophrenia patients--especially those at risk of suicide. The drug was discovered in the 1960s but continues to offer hope to treatment resistant individuals today. It has a low propensity for extrapyramidal side effects, even at high doses, but there are many life threatening side effects that disable it from being the first line of treatment. It is prescribed only when other antipsychotics fail.

Lori Schiller was one of these infamous treatment resistant patients. She recalls her experience with medications in *The Quiet Room*:

“I took pills for psychotic symptoms, pills for mood swings, and pills for anxiety. Because nothing had ever really given me long-term relief, the doctors were constantly trying me on something new. I went from one antipsychotic medication to another. Navane. Stelazine. Mellaril. Moban. Haldol. Nothing. Nothing. Nothing” (Schiller, 1996).

She recalls the nature of the side effects as well,

“I knew these medications mainly by their side effects. Some antipsychotic medications made me drowsy. Some blurred my vision. When I took Thorazine I felt like a zombie. My face looked like the frozen mask of someone who had been dead for weeks. I shuffled down the halls and my mind was a shadowy cloud...It gave me an appetite like a lumberjack and caused me to gain weight like crazy. My mouth was so dry that my lips would get stuck on my gums. Haldol didn't help the symptoms, and the side effects were horrible and scary” (Schiller, 1996).

Thankfully, Lori got the relief she so deserved when she signed up for one of clozapine's first clinical trials in the United States. She retells her initial experience with clozapine,

“Gradually, subtly, changes began creeping up on me. People began remarking on my changed demeanor. I was less impulsive, they said, and more thoughtful. I was looking brighter, more alive, they said. My parents said they saw beginnings of the sparkle back in my eyes again. The most striking thing I felt was a new sense of calm. For the first time in years, I slept... And the Voices? The Voices were growing softer... It was happening. I was being set free. I had prayed to find some peace, and my prayers were finally being answered” (Schiller, 1996).

Clozapine

The story behind clozapine, the gold standard, is a blend of medical misconceptions, public health scares, and accusations of company profiteering (Crilly, 2007). In 1953 the first clinical trials for the first antipsychotic drug had made their way to North America, and it was quickly approved by the FDA in 1954. The discovery of chlorpromazine in America created a huge boom in pharmaceutical research that ultimately led to the discovery of clozapine (Crilly, 2007).

In 1958 a group of tricyclic compounds were synthesized as antidepressants in a Swiss lab, one named clozapine was noticed to have similar properties to chlorpromazine but researchers noted how it differed in the lab in two important ways: first, it did not cause the loss of voluntary motion that was so common in chlorpromazine. Second, it demonstrated a higher pain threshold in rats. Interestingly, it was the second quality of the drug that led to further testing on humans. A number of clinical trials showed mixed reviews of clozapine, but a German lab finally confirmed the drug's antipsychotic properties. The German trial also famously and foolishly noted that the drug did not have any disabling neurological side effects. However, it was the extrapyramidal side effects that made antipsychotics so novel in the 1950s. In fact, many believed the more severe the extrapyramidal symptoms, the better the antipsychotic (Crilly, 2007). Therefore, clozapine was not met with the same enthusiasm that chlorpromazine was. Nevertheless, clinical trials continued in Europe, and the drug company Sandoz began to make plans to bring clozapine to the United States.

Drug development in Europe is much different than it is in the United States because of the role of the Food and Drug Administration. The FDA controls new drugs coming into the market as well as ensuring product efficacy. The FDA's role in the development in a new drug begins when a pharmaceutical company seeks to test potential therapeutic effects on humans. An "investigational new drug" application is submitted at this stage. After this, the three phases of clinical trials can officially begin. Upon completion of phase III, the company must submit a "new drug application" to the FDA for approval. Once approved, the drug can at last be brought to market. In 1973, Sandoz began this process in the United States for clozapine. By 1974, Phase II trials had begun

on male volunteers in prisons. However, it was at this phase that the hypotension side effect was discovered. Some of the men were quite literally crashing from the drug. The researchers reasoned that the hypotension was due to high doses given too quickly. The solution--a dosing regimen, start low and build to effective dosage levels (Crilly, 2007).

Momentum of getting clozapine to the US market slowed to a complete halt in 1975 when 18 patients in Finland on the drug developed severe blood disorders, and 9 lost their lives. Sixteen of these patients developed the now known side effect agranulocytosis. Agranulocytosis suppresses the production of white blood cells in bone marrow, leaving one vulnerable to infection (Crilly, 2007). Upon these deaths, several European governments ordered clozapine to be removed from the market. When these deaths were investigated, the researchers found that it was due to secondary infection due to agranulocytosis. They reasoned that the agranulocytosis was not diagnosed early enough so that clozapine could be withdrawn immediately. Following the Finnish incidence, there was mandatory implementation to weekly screening white blood cell counts for patients using clozapine (Crilly, 2007).

Unsurprisingly, the FDA in the United States was concerned about what had happened in Finland. Consequently, stage III of the clinical trials soon proved to be the most difficult hurdle to jump. To understand why, one must consider the political climate of the early 1980s. The Reagan administration pushed for ideals of competition, free enterprise, and greater involvement by the private sector in the healthcare field (Crilly, 2007). However, the appeal of clozapine was that it treated mental illness, a cohort that was mostly reliant on Medicaid. It was also during this time that the FDA helped pass the Drug Price Competition and Patent Term Restoration Act of 1984. These acts began to

allows generic drugs to proceed to market with minimal delay--guaranteeing only a 5 year period of exclusivity for the brand name drug following FDA approval. This policy gives pharmaceutical companies incentive to profit as much possible during those 5 years--forcing Sandoz to organize a plan to maximize its profits from clozapine in the US market (Crilly, 2007).

In the 1980s state governments were looking at any possible way to decrease state hospital stays for schizophrenic patients and create venues for less expensive treatment. Sandoz knew that clozapine could play a truly pivotal role in this operation (Crilly, 2007). Therefore, the FDA was supportive during stage III trials for clozapine. However, the FDA forced Sandoz to test subjects who had been failed by every other possible treatment--clozapine was their last chance. The trial compared clozapine's effectiveness to chlorpromazine. Although Sandoz was given the toughest crowd to work with, clozapine showed a clear superiority. It was approved by the FDA, despite lingering worries of agranulocytosis.

Clozaril, clozapine's brand name, entered the US market in 1990. The cost was stifling--almost \$9,000 per patient. Many families had to pay for the medication out of pocket, as neither Medicaid nor private insurance was willing to pay for the drug and the weekly tests for agranulocytosis (Crilly, 2007). Naturally, the outcry against the outrageous cost came loudly and quickly. By December of 1990, 29 states had brought the issue to federal court. A hearing convened in March of 1991 that sought to address Medicaid's inability to pay for the drug. Several physicians spoke at the hearing, making a case that the drug could be marketed at just \$200 per year, per patient. The final ruling came in May of 1991, and both Sandoz and the states were found at fault for barriers to

access to Clozaril (Crilly, 2007). State Medicaid programs were thereafter mandated to pay for the drug and the required blood tests.

Throughout the 1990s Clozaril was described as ‘new’--atypical even (Crilly, 2007). Therefore, it created a second scramble by drug companies to develop their own atypical antipsychotics--Zyprexa by Eli Lilly, Geodon by Pfizer, and Risperdal by Janssen to name a few. Nevertheless, clozapine remains the most effective antipsychotic on the market, particularly for treatment resistant cases. However, the fact that a nearly 60 year old drug remains to be the gold standard is troubling. There is little excuse for such lack of development, especially when so many face unrelenting psychosis coupled with horrific side effects of the antipsychotics available today.

Despite its demonstrated efficacy in psychosis and the expanding indications for its use, widespread use of clozapine has been limited by the potential for adverse effects. Adverse effects limit the rate at which the dose can be increased, as well as the maximum dose that can be tolerated by some patients. For many patients, clozapine is their best hope for successful treatment of a disabling mental illness. However, full benefit of the drug can only be achieved if the adverse effects can be controlled (Young, 1998). Below the adverse side effects of clozapine that must accounted for are recounted by Young. Some of them are found only in clozapine but several are common to most antipsychotics. They include: agranulocytosis, seizures, sedation, tachycardia, orthostatic hypotension, weight gain, and gastrointestinal problems, urinary incontinence, and akathisia.

Agranulocytosis is defined by low granulocyte count. The risk of agranulocytosis is highest in the first three months of clozapine treatment, and 95% of the cases occur

within the first 6 months. Agranulocytosis predisposes patients to neutropenic sepsis and has a mortality rate of 3 to 4%. Mortality rate can be significantly decreased if agranulocytosis is discovered before signs of infection and if clozapine is immediately discontinued. The pathophysiological mechanism is uncertain, but there is evidence of an immunological basis. Therefore, treatment with clozapine requires a weekly white blood cell count.

Seizures tend to occur during the upward titration phase of treatment or at exceedingly high doses. In Europe, lower total doses have generally been used with equal efficacy and fewer seizures. The risk of seizures ranges from 1 to 3% at low doses and up to 5% at high doses.

Sedation is the most frequent reported adverse effect of clozapine, occurring in approximately 39% of patients. It appears early in treatment and patients gradually develop tolerance, usually within 4 to 6 weeks of treatment.

Tachycardia or rapid heart beat from clozapine occurs in approximately 25% of patients. Tolerance generally develops within 4 to 6 weeks but may limit the rate at which the dose can be raised.

About 9% of patients receiving clozapine experience orthostatic hypotension. It usually occurs at the initiation of treatment or with dosage increases, and patients gradually develop tolerance, usually within 4 to 6 weeks of treatment. Patients with orthostatic hypotension secondary to clozapine may describe dizziness or lightheadedness and are prone to syncope. It can be monitored by taking sitting and standing blood pressures regularly when therapy is initiated or when dosage increases.

Weight gain is a frequent side effect of neuroleptic treatment. Recent studies have suggested that the weight gain associated with clozapine is more common than previously thought and may be even more common than with conventional neuroleptics. One study found that 75% of patients gained at least 10 pounds over a 6 month period. Over a 3-year period, more than 80% of patients increased their weight by 10% and over 38% increased by at least 20% percent, thus posing a significant long-term health risk. The mechanism of clozapine-related weight gain is uncertain, but as with conventional neuroleptics, its effects are likely to be multifactorial. Clozapine affects the histaminergic, cholinergic, endocrine, and metabolic systems, all of which can affect weight. In addition, serotonin antagonists stimulate carbohydrate cravings and cause weight gain.

There are a number of adverse gastrointestinal effects of clozapine including sialorrhea, constipation, and nausea. Sialorrhea or excessive salivation, occurs to some degree in most patients treated with clozapine, and tolerance does not usually develop. Although hypersalivation is generally a benign side effect, patients sometimes describe a choking sensation at night and may even aspirate excess saliva. In addition, drooling can be socially embarrassing. The pathophysiology of sialorrhea is not clear. Constipation occurs in 14% of patients treated with clozapine and can be severe. It is most likely due to the anticholinergic properties of clozapine. Fiber supplements or unprocessed bran are safe and effective in cases of mild constipation. Nausea tends to develop later in the course of treatment and affects 11% of patients. The pathophysiological basis for this symptom is also uncertain.

Estimates of urinary incontinence have ranged from 0-30%. The mechanism is unknown, but suggestions include excessively deep sleep related to clozapine sedating properties, and urinary retention with subsequent overflow related to clozapine's anticholinergic properties.

Akathisia, a state of agitation, distress, and restlessness, has been reported in 6-39% of patients treated with clozapine. This stands in contrast to reports of the use of clozapine as an effective treatment for patients unable to tolerate traditional neuroleptics because of severe akathisia.

For many patients, clozapine offers new hope for successful pharmacological management of a disabling mental disorder. However, up to 17% of patients must discontinue treatment because of adverse effects. Therefore, managing these unwanted effects is essential to a therapeutic outcome (Young, 1998). Furthermore, compliance with clozapine can be significantly enhanced if patients are completely informed by their healthcare providers about the nature and risks of its adverse effects.

Antidepressants in Schizophrenia

Because of the harsh side effects of clozapine, polypharmacology of antidepressants and antipsychotics is often utilized rather than simply taking clozapine. Rather than relying on clozapine, due to the side effects, many clinicians prefer pharmacological treatments that are used in addition to antipsychotics including lithium, anticonvulsants, sex hormones, glutamatergic drugs, acetylcholine esterase inhibitors, and antidepressants. Antidepressants as adjuncts in schizophrenia are particularly under extensive study and are, despite contrary evidence regarding their efficacy, already in wide clinical use (Terevnikov & Joffe, 2013).

The American Psychiatric Association suggests that antidepressants should be used with caution in schizophrenia because of possible risk that sometimes an antidepressant may exacerbate psychosis. However, recent research data shows that in actual practice clinicians tend to widely use antidepressants to overcome the co-occurring depression, posttraumatic stress disorder, anxiety, or schizoaffective disorder, negative symptoms, and cognitive deficits of schizophrenia .

Antidepressants may reactivate neuroplasticity that is lost in schizophrenia. Although antidepressants do not have antipsychotic properties, they may contribute to the improved treatment outcomes in different phases of schizophrenia. Many researchers believe that this might be an underlying mechanism of positive results in a recent study. In that study, antidepressants prevented conversion to psychosis in subjects with prodromal schizophrenia symptoms more effectively than did atypical antipsychotics. Another study found that add-on antidepressant treatment may become a useful option in treatment of negative symptoms of schizophrenia. Specifically, the receptor-blocking antidepressant has inspired more confidence than do antidepressants from other groups, since their effect on negative symptoms seems to be rather consistent (Terevnikov & Joffe, 2013).

Unfortunately, depressive symptoms are common in schizophrenia, and comorbid depression significantly elevates the risk of suicide and negatively influences patients' quality of life and level of functioning. Furthermore, at chronic stages of schizophrenia, depression is associated with a higher risk for relapse. Typical antipsychotics are known to even worsen depressive symptoms, and a considerable amount of atypical antipsychotic treated patients with schizophrenia suffer from depressive symptoms as

well. Therefore, antidepressants become an obvious pairing to combat the dangerous side effect of depression (Terevnikov & Joffe, 2013).

Naturally, drug interactions must be considered when mixing antipsychotics and antidepressants. Some drugs may inhibit certain enzymes, often causing unpredictable, drastic, or even toxic increase in blood concentrations of medications metabolized by these same enzymes. The role of these enzymes must be kept in mind when combining antidepressants and antipsychotics. The most common mechanism of the interaction is augmentation of the same neurotransmitter pathway. Another possible mechanism is competition at receptor sites and a direct effect on an organ/system's physiological functioning (Terevnikov & Joffe, 2013).

The most common pharmacodynamic interaction side effects between antipsychotics and antidepressants are: anticholinergic effects, extreme sedation, weight gain, extrapyramidal symptoms, cardiac effects, vascular effects, and proconvulsive effects. Although there are some side effects, it remains clear that antidepressants play an important role for some schizophrenics.

Obstacles to Clinical Research and New Drug Development in Schizophrenia

Treatment of schizophrenia remains the arena in which all our science and all the skills of clinicians are put to the ultimate test. Research to develop new treatments holds enormous promise for the future (Kane, 1991). However, one cannot help but wonder why clozapine, wrapped with all of its side effects, remains the gold standard in antipsychotics. Why has a better, more effective drug not come to pass? Unsurprisingly, there are difficult obstacles in this kind of clinical research.

Given the fact that schizophrenia is an illness that affects insight, judgement, and other aspects of cognition, the ability of patients to give informed consent is often a

legitimate concern (Kane, 1991). A related issue involves the fact that some patients do not understand or accept the fact that they are ill or are in need of treatment and are ultimately hospitalized against their will. To what extent can or should such patients participate in research?

Not only do researchers deal with the challenge of finding samples, but challenges inherent in drug development also exist. For example, during the early stages of the testing of a particular drug, patients may be offered acute treatment in an experimental protocol, but if they improve on the drug, they may not be able to continue taking it due to restrictions laid out by the FDA. Therefore, an enormously challenging tension exists between the scientific aspects of establishing drug efficacy and the increasing demand and expectation for the availability of experimental compounds outside treatment protocols (Kane, 1991).

Furthermore, there is a selective premium that is placed on subjects whose central nervous system neuronal receptors have not already been influenced by treatment, particularly chronic treatment, with other agents--schizophrenics who are often not so easy to find, due to the immediate prescription upon diagnosis (Kane, 1991).

Furthermore, the availability of accurate treatment histories can be an enormous problem in clinical research. The inclusion in clinical trials of patients who, for whatever reason, are unresponsive to treatments in general can also be a particular problem. This problem is accentuated by the natural tendency for clinicians to want to offer experimental treatments to those patients who have failed to benefit from other treatments. These patients are often encouraged to join these trials by added incentives (Kane, 1991). The fact that most pharmaceutical industry-sponsored trials provide financial support on a

per-patient basis can also foster the temptation to bend inclusion-exclusion criteria, sometimes leading to skewed results (Kane, 1991).

The very nature of schizophrenia also has an impact on the clinical resources, facilities, and ancillary personnel necessary to maintain an environment conducive to treatment research. In some psychiatric conditions, after an acute treatment phase, patients may improve to such a degree that they require little follow up care. However, this is rarely the case with schizophrenia. During or following participation in clinical treatment trials patients normally require psychosocial and vocational therapies, supportive living environments, family treatment, social clubs, jobs, and other support--all things that are often very expensive. While these problems are not an excuse for lack of development, it is now easy to understand why scientists have found strife in developing new drugs.

Psychosocial Treatments

While medications are very effective, the best treated individuals are those who combine medicinal therapy with some kind of psychosocial treatment. There is a huge variety of therapies available to choose from; therefore, the patient must choose what is right for them. As I was previously noted, Elyn Saks relied heavily on a psychoanalyst for treatment. Is this treatment right for everyone? Absolutely not, but for Elyn it worked wonders. Again I reiterate, individuals must be able to choose what is right for *them*. In this section I will present examples of psychosocial treatment models used today. They include: shared decision making, cognitive behavioral therapy, assertive community treatment, and familial involvement.

Shared Decision Making

One summer during her time at Yale Law School Elyn Saks worked together with her best friend at a halfway house for mental patients. It was here where Elyn became passionate about mental health law. She worked for mental patients and children that summer and from the experience she noticed:

“Psychiatric patients always have someone (or a whole chorus of someones) telling them what they’re supposed to do. In my own experience, I had discovered that it is much more effective to be asked what *I’d* like, e.g. ‘If you could arrange things your way, what would that look like and how do you think we could help you get there?’” (Saks, 2007).

Noncompliance of medication has been defined as, “a deviation or cessation of a medication regimen that is less than what was recommended by a doctor.” Compliance interventions are often designed for patients to conform to a *doctor’s* view of optimal treatment. However, shared decision making diverges from this view and it assumes that there are two experts, the patient and doctor, who share their respected information to determine an optimal treatment option (Deegan & Drake, 2006). Shared decision making with respect to medication is upheld by the values of recovery proposed by the Center for Mental Health Services Administration in 2004.

The use of antipsychotics is often referred to as a journey, as patients often approach these medications like scientists conducting a lay assessment of medication effects--not just on their symptoms but on their quality of life as well. Therefore, the compliance model, with its simple emphasis on obedience, is too basic to address the complex process that is used to find an appropriate medication (Deegan & Drake, 2006). When medications support or enable one to pursue activities such as employment,

parenting, and returning to school, they are perceived by clients as a valuable asset to the recovery process. On the other hand, if medication interferes with personal activities, medications are then viewed as blocking the recovery process and are often rejected by the patient. Therefore, insistence on compliance in this sort of situation is unhelpful. However, shared decision making allows the patient to share with his or her physician on what works for them and what does not (Deegan & Drake, 2006).

Like most things in life that involves the opinions of people, sometimes there is tension between these perspectives. For example, a doctor may think that a medicine is working well but the patient may no longer feel like themselves. This often results in conflict where the doctor insists on compliance while the patient discontinues the medication. Shared decision making allows the two parties to work together to achieve a consensus to ensure a more positive and effective recovery (Deegan & Drake, 2006).

Cognitive Behavioral Therapy (CBT)

CBT is time limited and collaborative therapy that involves an individualized case formulation approach based on functional analysis, routine monitoring of outcomes, and assigned home practice between sessions (Marcus, 2016). CBT for psychosis was developed with the underlying assumption that symptoms are maintained by both appraisal and behavior and can be modified using CBT techniques. For example, delusions are thought of as an attempt to make sense of negative affect and/or anomalous sensory experiences, which result from an interaction between biology, cognition, and behavior (Marcus, 2016). While not explicitly stated that they were using CBT techniques, Lori Schiller's therapists used several techniques of CBT on Lori before she found full relief from clozapine.

Within CBT, distressing hallucinations are conceptualized as products of a person's mind that reflect a propensity to access memories, internalized language, and negative core beliefs. Impairments in source monitoring or the ability to monitor the origin of one's own thoughts, may play a role. The associated distress is amplified by the experience of these phenomenon as alien, rather than coming from within one's own mind. Therefore, the goals of CBT include fostering a curious attitude about symptoms, improving the sense of personal control over symptoms, decreasing distress, and improving functioning and the acceptance of living with a difficult disorder (Marcus, 2016). Lori Schiller's therapists used this idea on Lori:

“Dr. Fischer and Dr. Doller explained that, there weren't really voices that other people could hear. It was just my own hostile thoughts getting blown up out of proportion inside my brain. I listened. I thought about it. No way, I thought at first. I don't have horrible thoughts like that. Those thoughts aren't me. It's those Voices who are the crazy demons, not me... It seemed impossible to me that they were simply figments of my own imagination. But gradually, with Dr. Fischer and Dr. Doller leading the way, I began to test the waters... Once I began to be able to tell my doctors what the Voices were saying about them, they began to help me look more closely at what the Voices were saying and why” (Schiller, 1996).

Special attention to the engagement process of CBT is needed with individuals suffering from psychosis. First and foremost, the clinician must lead with empathy, convey expertise, and express hope that therapy can be helpful. Lori's therapist, Dr. Doller, did exactly this. Dr. Doller shares her philosophy of treatment in *The Quiet Room*,

“We concentrated our efforts on getting inside the heads of our patients. Many other treatments focused on using medication to alleviate symptoms. These treatments were considered a failure if the patient was still hallucinating. We felt otherwise. We felt that, hallucinations or not, there was still a person inside there—and that we could reach that person if we tried” (Schiller, 1996).

Many schizophrenics are often very secretive about their psychotic symptoms, motivated by fear of involuntary hospitalization or of an increase in antipsychotic dosage or even from direct messages from the voices that order them not to be spoken about to the clinician. Furthermore, paranoia often makes it difficult to form a relationship for the patient to form a relationship with a clinician. This is also evidenced in *The Quiet Room*, “Dr. Diane Fischer, my new therapist is trying to kill me” (Schiller, 1996). Therefore, CBT recommends at the onset of therapy, clinicians encourage clients to share suspicious thoughts when they arise. In the early stages of therapy it is also important that the clinician adopt a curious attitude towards the patient’s experience to help elucidate the reinforcement behind a certain way of thinking about the world (Marcus, 2016). This is also evidenced by Lori and Dr. Fischer’s relationship, “When I began to tremble, and shake with fear, Dr. Fischer gently asked me to tell her what was on my mind. ‘Tell me what you are hearing, Lori,’ she said” (Schiller, 1996).

One of the first aspects of implementing CBT are interviews that are conducted to assess symptoms to create a case formulation. Assessment continues throughout treatment, but the initial assessment typically comprises three or five of the first sessions where the the patient and therapist work towards a shared case formulation. A basic care formulation might include common triggers, thoughts, and behavioral responses

associated with either the onset of or response to psychotic symptoms (Marcus, 2016). The assessment phase also entails the development of a problem/goal list that provides the blueprint for the goals of CBT. Often socially oriented goals are important, as social support often decreases psychotic symptoms, preventing relapse (Marcus, 2016). Dr. Doller also utilized this method for Lori, “First, we had to help her develop relationships with people around her. People with schizophrenia are filled with an essential longing. They have a longing to explain what is happening to themselves. And they have a longing for a connection, for some a relationship that will give them a pathway back toward the world they have lost” (Schiller, 1996).

In CBT, psychoeducation of symptoms, medication, and early warning signs can help to reduce the feelings of powerlessness, provide needed clarity, and provide a needed alternative for the reattribution of symptoms (Marcus, 2016). Dr. Doller also implemented this strategy, “We felt that by trying to understand too. And by helping them to understand, we could help them feel less overpowered and less terrified by their symptoms” (Schiller, 1996).

Optimizing coping strategies for psychotic symptoms is a focus of CBT treatment. In order to bolster coping, it is important that the clinician and client have a clear understanding of how the client has typically responded to his or her psychotic symptoms, the effectiveness of those strategies in reducing associated distress, and whether these coping strategies have been associated with any problems (Marcus, 2016). For example, to cope with command hallucinations is to comply with the instructions. This strategy is often problematic for obvious reasons--self harm, legal problems, and acting in a manner that goes against one’s values and goals. Therefore, the CBT therapist

must conceptualize the behavior as maintaining the distress the patient feels, and together with the patient identify ways to change these behaviors that are inhibiting normal functioning (Marcus, 2016). The brilliant Dr. Doller used this method to help save Lori's life:

“Take Lori's out-of-control behavior. Her record suggested that earlier doctors had believed she was breaking things, smashing walls and running away on purpose. They believed her behavior was under control. They believed she was manipulative, attention-getting, and locked in a power struggle with staff that could be handled only through strict discipline. I chose to believe otherwise. I saw her behavior as an understandable response to her scary inner world. As I observed her, I could see how she would get. As she spiraled out of control, we could begin to talk to her... But she couldn't hear. This woman wasn't playing mind games. This woman was in genuine distress. And how would she have any idea how to handle that distress without being taught?” (Schiller, 1996).

Dr. Doller saw her job as teaching Lori to recognize her symptoms as phenomena, and to seek help immediately before the symptoms became too much for Lori to bear. This is precisely the aim of cognitive behavioral therapy.

Assertive Community Treatment and Familial Involvement

Assertive community treatment (ACT) provides a comprehensive range of treatments, rehabilitation, and support services through a multidisciplinary team based in the community. Basic characteristics of ACT programs include assertive engagement (delivery of social services that is effective even for the hardest to reach clients and families), a multidisciplinary team approach, continuous responsibility, staff continuity

over time, caseloads with high staff-to-client ratios, and brief but frequent contacts (Scott and Dixon, 1995). ACT teams also provide a close liaison with the client's support system and a treatment focus on alternative activities. There is strong and consistent evidence that ACT reduces the rate and duration of psychiatric hospitalization. ACT programs reduce psychiatric symptoms, improve social functioning, and promote residential stability and independent living. ACT has also been shown improve compliance with treatment and is well accepted by patients (Scott and Dixon, 1995). When I spoke with Dr. Bitner, he noted that assertive community treatment has been very successful with his patients. On the effectiveness he remarked, "These patients cannot help themselves without the community behind them. Treatment today takes the whole village" (personal communication, April 7, 2016).

Family psychoeducation is an evidenced-based practice that has consistently been shown to reduce relapse rates for schizophrenia (Weisman et al., 2006). For instance, a meta-analysis conducted in 2001 strongly indicated that relapse rates for patients with schizophrenia can be drastically reduced if patient's family members are included in the treatment. However, most mental health facilities still lack family treatments of any type. During my conversation with Dr. Bitner he was sure to emphasize that families of his patients can also be very ill. He noted the importance of family support groups so that family members get a chance to air their grievances is often crucial to the success of the patient's treatment (personal communication, April 7, 2016). Expressed emotion is a widely used measure of the emotional climate of a household, specifically assessing relatives' critical, hostile, and emotionally overinvolved attitudes towards patients. Not surprisingly, a mounting body of research indicates that the course of schizophrenia is

highly correlated with the familial atmosphere. Many studies have indicated that returning home to a hostile, critical, or emotionally overinvolved environments can substantially increase chance for relapse, hence the importance of familial support groups (Weisman et al., 2006).

Early Intervention

Today's treatment model for schizophrenia is the best it has ever been. However, prominent researcher on early intervention, Patrick McGorry, asserts that stigma and pessimism have dominated therapeutic efforts to ensure that treatment efforts become delayed and inconsistent. On the contrary, McGorry cites that early intervention techniques in treatment have seen an explosion of sorts in research and effort.

The advent of preventative thinking in psychosis has required a shift in the way schizophrenia and other disorders are viewed. Rather than seeing them as having inevitably poor prognoses with deterioration in social and functional outcome as the norm, more recent thinking backed up by evidence from large international studies views the course of these disorders as being very fluid and malleable (McGorry et al., 2008). Examination of the risk factors which can influence outcome has revealed that many symptoms may be reversible. For example, disruption of peer and family networks and social dropout typically occur around and even before the onset of a first psychotic episode. Attention to these areas as part of treatment has the potential to limit or repair the damage (McGorry et al., 2008).

Early intervention can be a potentially confusing term. There are no known flexible causal risk factors that predict the onset of psychosis. Therefore, primary prevention is out of reach. Therefore, all prevention in early intervention is actually

secondary (McGorry et al., 2008). Early intervention in psychosis can be defined as comprising three stages: ultra-high risk, first episode, and the recovery or critical period. This is called a clinical staging model. It helps to provide a framework that allows for the development and evaluation of broad and specific interventions as well as the study of the variables and processes underlying the evolution of psychiatric disorders (McGorry et al., 2008). Put simply, clinical staging is a more refined form of diagnosis. It differs from conventional diagnosis practice in that it defines the extent of progression of disease at a particular point in time, and where a person lies currently along the continuum of the course illness. It enables clinicians to select treatments relevant to earlier stages, and assumes that such interventions will be both more effective and less harmful than treatments delivered later in the course (McGorry et al., 2008).

Defining discrete stages according to progression of disease creates a prevention-oriented framework for the evaluation of interventions. The key positive health outcomes are prevention of progression to more advanced stages. This requires an accurate understanding of those broad social, biological, and personal risk and protective factors which influence progression from one stage to the next. Risk factors for each stage can begin to be identified, and while some risk factors may operate across all stage transitions, others may be stage-specific, for example, substance abuse and stress may be especially harmful in triggering the onset of the first episode of illness, but can be less toxic in later stages (McGorry et al., 2008).

A key aspect related to early intervention methods is beginning to be more recognized: it is that mental disorders are the chronic diseases of the young. Most adult type mental disorders like schizophrenia have their origins in late adolescence and early

adulthood (McGorry et al., 2008). Therefore, developed countries need to recognize the public health importance of untreated and poorly treated mental disorders. McGorry further emphasizes that early diagnosis and intervention can change many of the issues faced by schizophrenics during their diagnosis and treatment process.

Invisible Wounds: Issues faced by Schizophrenics

Unfortunately, we live in a society that does not take kindly to mental illness, and schizophrenics, in all of their suffering, often face more suffering, as society struggles to understand their illness. Moreover, early intervention techniques are still in their infancy in the United States; therefore, many schizophrenics continue to suffer in their diagnosis and treatment. When the disease arose in the early 20th century in literature and in the public, state mental hospitals were implemented to care for people who could not care for themselves alone. While there is no hiding the fact that the early history of these hospitals was grim, horrible even, they served an important purpose for society. With the rise of deinstitutionalization, came an increasing number of homeless and criminalized schizophrenics, as public policy officials did not follow up on their promises to create more community based treatment centers. If schizophrenics are left untreated, they are at an immensely increased risk of drug abuse and suicide, as Lori Schiller's story so chillingly depicts. Increased criminality, drug use, and homelessness, are all issues related to schizophrenia that are the result of uneducated and misinformed societal choices. In addition, these problems further contribute to the stigma that has infiltrated mental illness for much of the 20th century and now, a far too large, portion of the 21st century. Most of society relates this stigma to the "inherent violence" associated with the mentally ill. When in reality, stigma ultimately relates solely to the *inherent ignorance* of society.

Deinstitutionalization

State hospitals were once the most prominent components of the United States public mental health systems. A major focus of mental health policy over the past fifty years has been to close these facilities. These efforts led to a 95% reduction in the country's state hospital population (Fisher et. al, 2009). Eliminating state hospitals continues to be a goal despite the enduring importance of the services they provide. Forces that worked for the changed role of state hospitals often called for more community based care or solutions (Fisher et. al, 2009). However, often the role of state hospital's has been to provide care for those people deemed inappropriate for placement in an alternative mental health setting for behavioral reasons (Fisher et. al, 2009).

The closures of state hospitals began with president John F. Kennedy's Community Mental Health act that was enacted in 1963. Where President Kennedy's legislation went wrong was that it failed to recognize the role that state hospitals had in managing the population of people that has been deemed inappropriate for normal societal expectations and settings. These patients, often schizophrenics, were often labeled "difficult to discharge." With nowhere else to turn at the closure of a hospital, these schizophrenics are often left on the streets or in jail (Fisher et. al, 2009). Since the 1970's, attention has been focused on the increasing rates of arrest and incarceration among people with mental illnesses who use state hospitals and other mental health services. A recent Massachusetts study of the arrest patterns of the state mental health agency's clientele found that nearly 30% of a cohort of nearly 14,000 people experienced state hospital stays, and they had been accused of recent charges, including mostly drug and nonviolent offenses. These charges likely complicated their housing arrangements,

employments, and other aspects of successful hospital discharge and community integration (Fisher et. al, 2009).

In line with President Kennedy's plan, in the late 1990's many state hospitals diverted their funds to expansions of community-based services. However, if schizophrenics do not successfully enroll in community-based treatment they remain at an increased to fail to comply with their antipsychotics, thus placing them at a higher risk to wind up on the streets or in jail.

Homelessness

A 2009 survey by the National Institute of Mental Health found that 20-25% of all homeless people in the United States suffers from mental illness. There has been an association with schizophrenia and homelessness for decades, and such an association does not solely exist in the United States. It has transcended into the United Kingdom, Europe, and Australia (Timms, 2005). Homelessness can take many forms. At one end of the spectrum are those sleeping outdoors, sleeping in abandoned buildings, or using hostels and night shelters. At the other end of the spectrum are the 'hidden homeless' who seek refuge with family and friends. (Timms, 2005).

The earliest scientific literature connecting schizophrenia to homelessness was a survey from one a police warehouse to a local asylum that revealed a large number of homeless men and women were also afflicted with schizophrenia. Unfortunately little progress has been made on this issue, as the most recent studies have found that homeless people with schizophrenia tend to have no current contact with mental health treatment services (Timms, 2005).

There are many explanations for the connection between homelessness and schizophrenia. Most notably, the United State has the deinstitutionalization of state hospitals to blame. While these social issues are certainly a part of the problem, the untreated symptoms of the disease themselves contribute to the problem of homelessness as well (Timms, 2005). For example, social withdrawal is often one of the early symptoms of schizophrenia. This withdrawal reduces the ability or likelihood that family or friends will lend the necessary support. Symptoms of schizophrenia also interfere with an individual's ability to cope with everyday life. For example, auditory hallucinations and delusions have the potential to make a schizophrenic individual believe that he or she was meant to be or deserves homelessness (Timms, 2005).

To add to this already unfortunate issue, the schizophrenic homeless population has become much younger, and many researchers have found higher rates of alcohol and drug abuse within the population. Nevertheless, actions are being taken to ensure that the connection between schizophrenia and homeless dissolves in due time. These changes have most importantly included implementing early intervention tactics, thereby reducing the number of new arrivals on the street (Timms, 2005). Volunteers in the United States, the United Kingdom, Europe, and Australia are also working to prevent dropout from mental health services. This is being done by establishing assertive community treatment. Volunteers often seek out patients who do not turn up for appointments, ensuring they do not fall through the cracks that lead to homelessness. Volunteers are also now working with programs that engage with those who are already homeless through mediums like street outreach teams, community hospitals, and temporary accommodation (Timms, 2005). The relationship between homelessness and

schizophrenia undoubtedly remains a problem today. However, the issue is being seriously addressed by updated, more effective treatment models. Nevertheless, these treatment models have to get the needed funding to stay in operation, often a major impediment for many communities.

Criminality

As was evidenced in Kurt's story, situations can arise when schizophrenics wander the street with their delusions and hallucinations leading them astray. As evidenced by Kurt's account, dramatic things can happen, like his breaking into someone's home while on vacation in Montana after he suffered from a cat scratch that he believed was "a poison dart inserted by bad CIA agents" trying to kill him. He broke into a home with the idea of finding something sharp to remove the "dart" with. Kurt did not believe anyone to be home, but he turned out to be very wrong. The residents were home, and they called the police. Thankfully, both the residents and the police understood that Kurt was very mentally ill, and they took him to a hospital, rather than jail (Snyder 59-62). Other schizophrenic individuals are not so lucky, and their criminalizing delusions like this land them in jail time and time again, often for similar nonviolent crimes.

Researchers Modestin and Ammann conducted a study that examined schizophrenia and criminality in males afflicted with schizophrenia to better understand the connection between crime and the disease and how it might be resolved in the future. The researchers cite prior studies that note correlations between criminality and lack of active treatment, delusions, noncompliance with antipsychotics, substance abuse, homelessness, and prior arrests (Modestin & Ammann, 1996). In a general population

birth cohort study, men with a major mental disorder, including schizophrenia, were found to be 2.6 times more likely to have been convicted of a criminal offense than healthy men and were registered for more crimes. Mentally ill offenders are more frequently arrested and shifted into the penal system because of unavailability of mental health care, more rapid discharge from psychiatric hospitals, refusal of these hospitals to admit dangerous patients, lack of beds, lack of resources, etc.

Notably, Modestin and Ammann found that differences between the patients and the controls appeared when individual crime categories were considered. For example, patients were 2.5 times more likely to have been convicted of crimes against property and almost 3 times more likely to have violated drug laws. The researchers also found that 59% of the patients with a criminal record had been criminally convicted before their first psychiatric hospitalization and 41% afterward; therefore, the beginning of the criminal behavior was quite equally distributed around the time of the first psychotic episode (Modestin & Ammann, 1996). Unfortunately, issues that schizophrenics face often lead them to becoming confined to a jail cell, rather than obtaining proper mental health care.

Substance Abuse

As the researchers Modestin and Ammann found, illegal substance abuse leads too many schizophrenics behind bars. This means that many schizophrenics not only deal with the mental stress of the disease but also deal with the mind games of drug addiction. Comorbid substance abuse disorders have emerged as one of the greatest obstacles to the effective treatment of persons with schizophrenia (Warner et al, 1994). Lori Schiller's battle with cocaine began soon after she was released from the hospital after she landed a waitressing job. She laments,

“Those little pills or a quick snort on the other hand could make me feel incredibly relaxed when the Voices were making me tense. But drugs had never been a big part of my life until I got out of the hospital... All I was trying to do was feel better. The medications they gave me in the hospital were useless... And the Voices still raged away at me, mocking the drugs, the doctors and me. Cocaine, on the other hand, helped me ignore the Voices” (Schiller, 1996)

Lori’s only goal was to get high and avoid the crash that would inevitably come.

The odds of having a substance abuse diagnosis were found to be 4.6 times higher for persons with schizophrenia than for the rest of the population; the odds for alcohol disorders have been found 6 times as high (Warner et al, 1994). Researchers Warner et al. uncovered that schizophrenics have been found to have a heavier pattern of use of marijuana, LSD, and amphetamines. Previous studies argue that drug dependent schizophrenics, like Lori, use substances to self-medicate painful affective states and other psychiatric symptoms. Other researchers reported that patients with major mental illness may try to use a variety of substances to control depression. For example, schizophrenic inpatients interviewed during a 1990 study reported that various substances were effective in reducing depression and anxiety, increasing energy, or alleviating negative symptoms of schizophrenia, but that these substances could also increase suspiciousness and hallucinations (Warner et al, 1994).

Previous studies have noted that substance abusing mentally ill patients appear to be less compliant with medication use, a difference that may contribute to the worse pattern of illness. Therefore, researchers Warner et al. developed a study to evaluate substance use in an outpatient population whose medication compliance was well

controlled by assertive community treatment. To conduct this study, seventy-nine psychotic patients were randomly selected from the caseload of a comprehensive community mental health center. Most patients were afflicted with schizophrenia or bipolar disorder. A confidential interview was conducted. The results from the Warner et al. are staggering. They found that the lifetime prevalence of use of marijuana in the study sample was 89.1%. For hallucinogens and stimulants it was 61.8% and 60%, respectively. The researchers noted that hallucinogen use by the mentally ill sample was substantially greater than the general population sample. In addition, nearly 90% of subjects had used marijuana, compare that to the college-age control sample of 61%. The same is true for hallucinogens (over 60% versus 15% of college-age subjects), cocaine (49% versus 24%), and other stimulants (51% versus 27%) (Warner et al, 1994).

Although these findings are quite dreadful, the researchers found that substance use may become less frequent when mental illness is better controlled, and improvements in this sample may be related to provision of intensive case management and daily monitoring of medication, prevention of relapse, money management--restricting the opportunity for patients to purchase large amounts of drugs or alcohol (Warner et al, 1994). Lori's story can also attest to these results; when her illness was controlled, he turned her back on cocaine. She testifies in the epilogue of *The Quiet Room*, "As for drugs, real drugs, street drugs, I have never taken up the offer to get high with anyone since the cocaine incident at [the halfway house] years ago. I've come too far and accomplished too much to waste it all by sliding back into the shadowy world of drugs" (Schiller, 1996).

Like Lori, the subjects in the Warner et al. study cited social interaction, relief of unpleasant affective states, and boredom as the principal reasons for substance use. Many subjects also noted a feeling of improved self-esteem. Subjects who preferred hallucinogens, as they were cited to help the subjects “stay awake” and to “feel better physically”. The effects of antipsychotic medications were also said to be relieved or lessened with the use of hallucinogens (Warner et al, 1994). Subjects who preferred marijuana used it to reduce anxiety and boredom, and these subjects were often found to have little structured daily activity. This observation indicates that work and activity programs may be helpful in reducing marijuana use among the mentally ill (Warner et al, 1994). As evidenced by Lori’s account and by the results from this study, drug use is a problem that persists in society. It is also a large contributor of jail time for schizophrenics as well; therefore, drug use needs to be more fully addressed in our society so that schizophrenic can get effective treatment they need to live more fulfilling and safer lives.

Suicide

Unfortunately, there are situations in which some or all of the issues of schizophrenia I have listed above compound on a single individual until that person feels totally hopeless. Not only did Lori Schiller deal with drug use, she attempted suicide a staggering four times. When Lori was home with her parents in between her major hospital stays, suicide was often on her mind, “My head swarmed with suicidal fantasies” (Schiller, 1996). She shares many of her suicidal thoughts in the memoir. They are vivid, fascinating, but mostly very disturbing:

“I didn’t want to just take an overdose or slit my wrists. I wanted something powerful that would reflect the despair that haunted me every day of my life. As the pressure of those thoughts built, my imagination went wild seeking ways of accomplishing my aims. I thought about jumping in front of a car, or better yet, a truck; or even better yet, a train. I thought about jumping out of a moving car onto the highway. I thought about standing on a bridge, pouring a can of gasoline over my head, lighting a match, and jumping in the flames to my death. Splat. Rocks in a bathing suit, then the ocean? How about jumping into a vicious animal’s cage in the Bronx Zoo” (Schiller, 1996).

It is nearly impossible to fathom what it must be like to have such terrifying thoughts. One wonders what might even cause such thoughts. Lori’s Voices were pure evil, “The Voices would alternatively chant, ‘To die! To die! To die!’ and then ‘Peace! Peace! They are waiting to give you peace!’ There was only one route to peace. The pressure was building. Finally, it became unbearable. I had to act” (Schiller, 1996).

Living with such Voices is inconceivable to most; therefore, it comes as no surprise that many schizophrenics, like Lori, cannot deal with their illness and feel that their only escape is suicide. It is estimated that about 5% of schizophrenia patients commit suicide and a considerably greater portion attempt suicide at some point of the illness, with estimates varying in the range of 20%-40% (Kjelby et al, 2015). The most established clinical risk factors for suicide in schizophrenia include depressed mood, hopelessness, previous suicide attempts and fear of mental disintegration. Other risk factors, some shared with other psychiatric disorders, include younger age, early stage of illness, good premorbid adjustment of functioning, male gender, married status, living

alone, unemployment, substance abuse, higher intelligence, having awareness of/insight into disease, recent loss, access to lethal means, poor adherence to treatment and recent admission to hospital.

Researchers Kjelby et al. found that the evidence concerning the influence of positive psychosis symptoms such as hallucinations and delusions, on suicidal behavior is not extensively described in current literature. However, hallucinations are prominent and often dramatic symptoms during the acute phases of schizophrenia. Much of the current literature also indicates that delusions are not a substantial risk factor for suicide; nevertheless, there are numerous case reports and a few studies that describe completed suicides and very serious self-harm in the extreme psychotic and delusional states. Therefore, taken together, researchers Kjelby et al. assessed that roles of hallucinations and delusions remain unresolved in schizophrenia-related suicidality research.

To conduct their study of the relationship between suicide and hallucinations and delusions, 124 patients suffering from schizophrenia were assessed. The primary aim was to determine the risk attributed by hallucinations and delusions on suicidal ideation and plans and attempts. In their analysis they found that depression, drug use, hallucinations, and negative symptoms were associated with suicidal ideation. Only depression and hallucinations were significantly associated with suicide plans. Increased suicide-risk in a hallucinating patient may be related to the hallucinations devaluing the patient or voices encouraging self-harming behavior. These results indicate a relationship between hallucinations and suicidal ideation and suicide plans (Kjelby et al, 2015). As expected, depression was the factor in the study that increased the chance of suicidal ideation and suicide plans most markedly. Depression, drug use, and hallucinations increased the

chance of suicide symptoms and negative symptoms reduced the change of suicidal symptomology. This study aimed to warn against underestimating the risk contributed by hallucinations on suicidal ideation, plans and attempts. The suffering and human cost due to suicide attempts may be substantial and the prevention of suicide ideation and attempts in themselves is in its own term highly important (Kjelby et al, 2015).

Researchers Jovanovic et al. created a study to explore the association between psychopathology and suicidal behavior in patients with schizophrenia, acknowledging three groups: suicide ideators, suicide attempters, and participants without suicide ideation and behavior. A previous study showed that during the first year after suicide attempt, the active form of the disease was present in 78% of participants, depressive symptoms in 64%, previous suicidal attempts in 71%, and command hallucinations in 10% of participants.

Jovanovic et al. found that a higher percentage of suicide attempters were unemployed or retired and a much lower percentage complied with the treatment in terms of regularly using their antipsychotics. There was a higher percentage of men in the groups with suicide ideation or attempt. Suicide ideators were on average somewhat younger than those without suicide ideation. Suicide attempters had greater mean numbers of institutional admissions than the other two groups. They were also more depressed than suicide ideators. Jovanovic et al. also found that only negative symptoms were found to discriminate among the patients. In comparison to the group of patients without suicide attempt or ideation, some of the negative symptoms were slightly higher in the groups with suicide ideation or attempt (e.g., social withdrawal and passive/antisocial withdraw) (Jovanovic et al, 2013). This study's results are in

accordance with other studies that depict suicide behavior in schizophrenia not as related to psychotic symptoms such as delusions and hallucinations as it is to depressive mood. The majority of suicide attempts in the study happened during an ongoing psychiatric treatment and the odds of suicide attempts increased with the number of prior hospitalizations. This might indicate that these participants suffered from a greater burden of symptoms but it might also be argued that if these patients tend toward suicide behavior, this tendency itself would increase the likelihood of hospitalization independent of illness severity (Jovanovic et al, 2013). Like many of the issues listed above, suicide is very much preventable with proper treatment.

Stigma

“Mental Illness comes with stigma attached to it, and that stigma can set off a negative reaction, even from the nicest people, with good intentions and kind hearts. Even for many of these people, those with mental illness are other; they're not like ‘us.’” -Elyn Saks

In light of recent events in the United States, most Americans have an outlook on the mentally ill that, in all honesty, cannot be further than the truth. People believe that schizophrenics are violent, dangerous, wacko, insane, crazy, etc. When most schizophrenics live a life much like my Aunt Linda, in a group home, medicated, and in reality, quite tame. Nevertheless, I can almost assure that if young people in the United States were to name a face of schizophrenia, people like my Aunt Linda would not come to mind. Instead, they would point to those like James Holmes, the man responsible for the movie theater massacre in Aurora, Colorado. First, I want to firmly reiterate that the violence that Holmes committed is inexcusable, unforgivable, and heartrending. Be that as it may, I would like to take a further look into James Holmes’s mind to illustrate that societal stigma and lack of treatment are a deadly combination that *must* change.

First, I would like to establish some of the evidence that lead me to believe that Holmes suffers from schizophrenia symptoms. I should point out that his formal diagnosis from the court-appointed psychiatrist on his case is schizotypal personality disorder. Nonetheless, the World Health Organization recognizes it as a disorder that that is very closely associated with schizophrenia. The disorder is differentiated from schizophrenia by extreme social anxiety. According to a CNN article on Holme's psychiatrist, his first visit was scheduled under reference of none other than extreme social anxiety. I would also like to point out evidence to schizophrenia symptoms from an ABC news article that came out in the early summer of 2015 in light of his trial: he suffered from both auditory and visual hallucinations. He claimed to see "flickers" of shadows of axes and guns out of the corners of his eyes. He also claims to have heard voices, though these voices did not tell him what to do, instead they were "talking about him in another room." To which I wonder if his voices were anything like Lori Schiller's Voices? What if he had had a therapist like Dr. Doller who took extreme, concerted action to change his disorganized thought process?

Holme's journal, which he entitled his 'Course of Life,' recounts this life, ideas, and thought processes up until the massacre in July of 2012. The journal was made public in the summer of 2015, following his trial. Upon reading the contents of the journal, one is vehemently aware of his illness. In combination with a broken mind, this man was suffering from a multitude of horrors that included extreme social isolation. His childlike writing composes page after page of his self diagnosis of a broken mind. Some of his self diagnosed symptoms are undoubtedly some of the insignias of schizophrenia: catatonia, social isolation, inability to communicate with others, difficulty concentrating, "odd"

sense of self, and finally delusional thoughts on homicide are mentioned to which he wrote, “the obsession to kill.” He follows his self diagnosis with narrative that says, “And finally, the last escape, murder at the movies. Obsession onset > 10 years ago. So anyways, that's my mind. Its broke. I tried to fix it.” The journal continues with bizarre and complicated loose associations, made up symbols, 7 entire pages of the word ‘why?’ written over and over again, and finally his plan of mass murder. After finishing Holmes’s journal, which was by and large the most disturbing piece of writing I have ever read, my only response is, why has our society allowed someone to feel like their only escape is mass murder? Why are individuals with schizophrenia like, Elyn, Lori, Kurt, and finally Holmes so afraid to admit their symptoms to people who can offer treatment or help? To address the seven pages of ‘why?’ in the journal. I need only conclude with one word: stigma.

Many are quick to point out that Holmes had received therapy up until about three weeks before he committed the shootings. Many have posed, if he was receiving treatment, why did this happen? To which I would respond, “Was he diligently taking medication?” According to the ABC news article that came out just after the trial, he was not on medication the night of the murders. In his journal he jots that his psychiatrist immediately prescribed antidepressants but he felt “no effect.” According to the CNN article on his previous psychiatrist, Lynne Fenton, when she wrote his prescription, she got his name wrong--prescribing it to “James Hughes.” This was most likely extremely detrimental considering Holmes already had deep feelings of hatred towards society. Who could even imagine how Holmes felt after even his *therapist* could not remember his name? By now, I am sure that it is obvious that the events of July 20, 2012 might not

have unfolded had James Holmes been on a steady regimen of antipsychotics, and the correct antipsychotic at that. What if he had been on clozapine? Would he have responded as miraculously as Lori Schiller? In addition, how could he have benefitted from programs like assertive community treatment? I can only guess that it might have significantly improved his feeling of social isolation, and it would have definitely ensured that he was getting effective medicinal treatment.

Similar to Lori Schiller, Holmes was described as hostile, anxious, and “not very forthcoming” by his psychiatrist, Lynne Fenton. She notes that from his very first session with her he described homicidal thoughts, but she never believed he would act on them. In fact, she told police that she tried to form an alliance with Holmes to ensure that he would continue counseling and continue taking his medication. This sounds quite rational. However, during their last session even she grew to fear him, noting an “angry edge” according to CNN. Instead of the police, she called his mother who responded with, “Yes, he's been troubled for many years.” Which leads me to wonder why the only prior psychiatric help before Fenton he had was a family therapist in childhood, despite his showing of early warning signs that include childhood night terrors and obsessions with homicide beginning as a teenager. In his journal, Holmes wrote that “He revealed nothing to the family therapist, so that he would not appear weak to his family.” One can only assume that had James Holmes felt comfortable sharing his madness earlier, his parents might have further employed the basis of early intervention with respect to his psychosis, and the events of July 20, 2012 might not have come to light at all. Likewise, what if Holmes had not fallen victim to the deceitful idea that mental illness means weakness?

Despite efforts to get help, Holmes was not treated effectively. Holmes did not act randomly, his actions were premeditated down to the last detail. Making one wonder how he even slipped through the cracks to pull this off? Regardless, in his broken mind he is now “known for something” thanks to the media frenzy that surrounded his case. The mentally ill fascinate us, so the media preys on them, thus further perpetuating ideations of violence and criminality associated with them. The day after it happened, when my family was caught up in the media’s circus, my mom look one look at Holmes’s large, expressionless eyes and dyed orange hair and said, “That man is a monster.” While she is not wrong, I must pose the question: what created the monster?

I presented Holmes’s case to illustrate that our society is the monster that needs to change so that we can begin to accept the realities of mental illness and get these people the help they need before they end up homeless, in jail, addicted to drugs, committing suicide, or in a very small percentage of cases, mass murder. First and foremost, I would like to inquire why mental illness policies are consistently called into question upon mass shootings? Politicians are quick to deflect gun control conversation to mental illness, but are not so quick to actually draft appropriate legislation to fix the problems the mentally ill face. In the words of John Oliver of the satirical political program *Last Week Tonight*, “It seems there is nothing like a mass shooting to suddenly spark political interest in mental health.” Oliver goes on to condemn former governor of Arkansas, Mike Huckabee, for his comment on CNN in light of the recent shootings at an Oregon community college during the early stages of the 2016 presidential race. Huckabee incited “Do we need to do a better job in mental health? You bet we do.” Oliver rebuttals, “It’s worth noting that Governor Huckabee’s state got a D- in mental health care when he

was in office, and you can't lecture people in something that you got a D- in." The real issue, as presented by Oliver, is that the entire political system that encompasses mental illness needs a complete overhaul. True to most topics related to schizophrenia, such an undertaking will not be easy due to the fact that there are hundreds of programs involved: medicaid, 8 federal agencies, 112 different programs associated, and the social services programs in each state. Oliver finishes the segment by citing the effectiveness of assertive community treatment programs: "Doing everything it takes for people to stay healthy is a lot better than what we've been trying, which is nothing, not anything, very few things, not much, and prison." Unfortunately, assertive community treatment and many other programs I have cited are in danger of budget cuts; however, these programs often pay for themselves. Consequently, Oliver emphasizes that, we as a society must figure out how to pay for them, because they save lives.

Moreover, we must ask ourselves how often the mentally ill are actually related to gun violence. Although it may seem like a lot, due to the attention from the media and opportunistic politicians, in reality fewer than 2% of mentally ill individuals are considered violent (Choe et al., 2008). Oliver contests during the same segment that when mental illness is talked about in light of mass shootings, it is seriously misleading-- compare the 2% of violent crimes attributed to the mentally ill to the 40% of arrests for serious violent crimes that belong to the cohort of males under 24 (Choe et al., 2008)

In a recent study that examined this perpetuation of violence, researchers Choe, Teplin, and Abram suggest that data must be collected on the mentally ill who are not receiving treatment. These researchers also cite the need to disentangle the causal relationships between severe mental illness, victimization, and perpetration. Choe,

Teplin, and Abram then created a model whereby social disorganization and poverty, a phenomenon common among many persons with severe mental illness, increase a person's vulnerability to victimization and their propensity to perpetrate violence. Repeated victimizations may lead to suspicion and mistrust, which in turn may lead to conflictive and stressful situations--creating the vicious cycle of victimization and perpetration (Choe et al. 2008).

The researchers recommend the following steps to stop the cycle: encourage mental health centers to assess the risk of victimization and perpetration in their communities and create interventions that provide information on substance abuse, homelessness, medication adherence, and conflicted relationships to help the mentally ill equip themselves with the proper tools to enhance personal safety and reduce conflict (Choe et al., 2008). The researchers conclude by voicing that the media has an enormous power to reduce stigma, rather than perpetuate it, through a plethora of mediums: TV, magazines, social media, etc., because at this point, there is no excuse for keeping the public uneducated and misinformed (Choe et al., 2008).

Personal Stigma

Stigmatization cannot be fully understood without taking into account the subjective perspective of people with mental illness, as they very clearly play an active and important role in this process (Switaj et al., 2014). The subjective perspective is often referred to as personal stigma, as opposed to public or societal stigma. Three main aspects of personal stigma are distinguished: perceived stigma, experienced stigma, and internalized stigma, the process of the internalization of stigmatizing societal attitudes,

resulting in fear of discrimination, social withdrawal, feelings of shame, guilt, and hopelessness, and a decrease in self-esteem(Switaj et al., 2014).

Elyn Saks writes eloquently and often about how stigma has affected her life in *The Center Cannot Hold*. She recounts,

“There were whole parts of myself that I tried desperately to keep hidden. I knew, for instance, not to share my ongoing delusions of evil, in particular the part about my being evil and my total certainty that I was capable of horrible acts of violence. Not that these thoughts were wrong; I believed everyone thought this way, but just knew better than to talk about it.” She continues, “One of the worst parts of schizophrenia is the profound isolation--the constant awareness that you’re different, some sort of alien, not really human. Other people have flesh and bones, and insides made of organs and healthy living tissue. You are only a machine, with insides made of metal” (Saks, 2007).

Elyn’s words are dismaying. Therefore, we must ask ourselves how we can change individual thoughts and actions; we must ask how we can be a *friend* to help raise self esteem and self worth in order to save *lives*.

A study conducted by Switaj et al. sought to examine what socio-demographic and clinical variables are most prominently related to the intensity of stigma reported by people with mental illnesses and to determine how various aspects of personal stigma contribute to the impact of stigma. The researchers found that the participants most often felt internalized stigma followed by perceived stigma followed by actual stigmatizing experiences. Within the sample, the factors most associated with greater impact of stigma were a schizophrenia diagnosis (as opposed to any other mental illness), current inpatient

treatment, experienced stigma (bullied, denied rights, etc.), and internalized stigma (social withdrawal) (Switaj et al., 2014).

This study's findings add to the extent of literature demonstrating that self-stigma and experienced stigma can negatively influence personal and family life. Hence, counteracting their pernicious effects is crucial for the improvement of the well-being of people with mental health problems. The researchers recommend that the interventions to reduce internal stigma need to enhance an individual's self-esteem, empowerment, and help-seeking behavior.

Stigma in Healthcare

Next, I would like to draw attention to the last clause of the previous section that states interventions need to reduce stigma to improve help-seeking behaviors. Stigma negatively impacts almost every aspect of the lives of the mentally ill, including whether or not they seek treatment from healthcare professionals. Upon reading the stories of Elyn, Lori, and Kurt, I was taken aback by how each of them were treated in healthcare settings. The language that they use to describe their hospitalizations is frightening, leading me to surmise that their unjust treatment while in the hospital was due to misunderstanding and stigma from people who were likely just trying to *help*, but were unable to perceive their actions as problematic. Stigma in psychiatric healthcare leads to biased punishments and treatments that are ineffective, painful, and emotionally damaging.

During Lori Schiller's first extended hospitalization, she incites the inception of her hatred,

“Everything about the hospital infuriated me. I didn’t know why I was there. I didn’t know how I had gotten there. All I knew was that I was trapped. I felt like a prisoner doing my time. I looked out the window every single day and waited for my freedom. Outside was so inviting. I begged for a walk on the hospital grounds. I was always so grateful for outside air. I hated being locked up... I wanted to get out to be normal again” (Schiller, 1996).

There are a few very concerning remarks within this passage that tell me that Lori was not receiving the most effective care. First, she did not understand how she had gotten to the hospital. No one took the time to explain to her the mental illness is very real sickness, and that was the cause of her suffering, not her actions. Second, no one explained to her that this was essentially her new normal. Without this information, it is easy to assess why Lori loathed the hospital from the start.

After her third suicide attempt, Lori was hospitalized for the second time. Upon her second hospitalization, Lori was very ill. Her Voices were in complete control. She recites that from the very first days, the days inside the hospital here “painfully empty.” She saw a psychiatrist three times a week: “I didn’t like my doctor I was assigned. He didn’t understand me. I didn’t care if I understood him or not. So our sessions were a dreary waste of time” (Schiller, 1996). Lori let her Voices run the show during this hospitalization. She began cheeking her medicine, i.e. not swallowing it and made frequent visits to her memoir’s namesake, *The Quiet Room*. She describes her idea of the room in great detail,

“The Quiet Room was supposed to be a safe and tranquil place, a place where patients could be alone, free to relax and calm themselves down after a crisis.

Some walked in voluntarily... Me, I was usually carried there. I hated it. It was almost a routine. I'd hear the Voices, would feel the need to do something destructive, and be sentenced to the Quiet Room" (Schiller, 1996).

When Lori was in the Quiet Room her voices went wild:

"Far from calming me down, the very emptiness of the Quiet Room became the screen on which terrible fantasy projected itself. The Voices spoke to me through cracks and vents in the walls. The overhead light transmitted messages to me. I couldn't breathe. My skull was coming undone and the Voices became megaphoned until I was sure they would deafen me. I had to make them stop. I had superhuman energy, superhuman strength. I literally punched a hole in the wall. I pounded my hands across the safety screen on the windows, opening my knuckles and fingers till the bones showed, and blood ran down my arms. I was beyond even the Quiet Room" (Schiller, 1996).

This passage is perhaps the most disturbing that Lori shares. It is literally terrifying, absolutely hair-raising. Nevertheless, the punishment she received from these actions is *almost* scarier,

"I was going to be cold-wet-packed... The idea behind cold wet packs was to chill the patient thoroughly. As the body struggled to warm itself, it would use energy. And as the person tired from the effort to get warm, he or she would calm down, ultimately relax, and, it was hoped, fall asleep. I was wrapped securely in sheets that had been soaking in ice water. They wrapped me tight as a mummy... Cold pack protocol mandated a full two hours as this freezing mummy. When the two hours we up, a decision had to be made. Was I calm enough to be unpacked? If

not, an order had to be made for an additional two hours. After the two hours were up, I had usually recovered enough of myself to be self-conscious about what had transpired... I'd be freezing, wet and cramped, and feeling embarrassed, degraded and demeaned by the whole process" (Schiller, 1996).

While Lori was violent and undoubtedly needed to be calmed down, why was, what I would consider a form of torture, used as a punishment for someone who was so terrified of everything around her? The idea was not only ineffective, as she cites more than one cold-packing, but degrading in such a way that reiterated that her psychosis made her different, more violent than others, thus worsening self stigma, self-esteem, and pushing her deeper in psychosis. The health care that was supposed to help her, made everything much worse.

Elyn Saks's first stay in an American psychiatric hospital also harkens images of medieval torture. She chronicles her horror,

"Within seconds, The Doctor and his team of goons swooped down, grabbed me, lifted me out of the chair, and slammed me down on a nearby bed with such force that I saw stars. Then they bound both my legs and arms to the metal bed with thick leather straps." Next, they forced medicine down her throat, and as you can imagine this was a terrifying and belittling experience. The next day she was forced into restraints once more to which she recounts, "No single hallucination, no threat of demonic forces or impulses I couldn't control had ever held me hostage like this. No one I knew, no one who ever loved me, knew that I was here, tied to a bed with a net over my body. I was alone in the night, with evil coming at me both from within myself and from without. It was unimaginable to

me at that point that the ancient meaning of the word ‘hospital’ is ‘shelter.’
Refuge. Comfort. Care. *No*” (Saks, 2007).

She pleaded with her doctors about her detest for the restraints. “*Please, I’ll do anything.*” The doctors persisted, citing that she was very psychotic. She brilliantly defends in *The Center Cannot Hold*, “If they couldn’t tolerate what was in my head, why were any of them in this business?”

Fortunately, Elyn was able to take her frightening experience and do something about it. She wrote a Note for *The Yale Law Journal* inciting a beautiful argument. The Note first establishes,

“A patient should be deprived of his liberty only when failure to do so either presents a risk of serious physical harm to himself or others or prevents medical treatment which has clearly been shown to be effective. Second, a patient should be deprived of his liberty only to the extent necessary to achieve the desired goal. Third, a patient’s right to choose among treatments should be protected whenever possible. Fourth, when a patient must be deprived of liberty, a set of strict procedures should be imposed to ensure that the infringements upon his liberty and dignity will be kept to an absolute minimum. The law’s current treatment of restraints substantially departs” (Saks, 1986).

Not only does she flawlessly establish the rights of individuals with mental illness, her counterarguments are perceptive, concise, and perfect. For example, she takes down the argument that psychiatric patients cannot appreciate their actions in the same way that other individuals can, meaning we should therefore be less concerned about protecting their liberty by instancing two reasons. The first is that nothing even remotely suggests

that psychiatric patients value their freedom as least as much as anyone else does. Second she establishes that creating a “standard of liberty based upon an individual’s level of functioning” would open pandora’s box about the weights and merits of the liberties of different individuals within society. Elyn argues that rather than having to ask ourselves these questions, we must ensure that these individuals maintain the little freedom they have while also giving them a right to choose how they are treated (Saks, 1986).

The Note continues by advancing on medical professionals, “The physician's superior medical knowledge does not vest him with a unique ability to make these collective, ethical choices. The questions are properly social, not medical, and the answers should be properly supplied by social mechanisms” (Saks, 1986). Elyn brilliantly makes clear that holding someone in restraints is not a medical issue but a violation of human rights that must be addressed by legislation. Elyn remarks in *The Center Cannot Hold* that lawyers have used her note as inspiration to create lawsuits that challenge the use of restraints. Elyn’s determination and brilliance is incredible, and her fight against stigma in mental illness had only just begun with this note.

Today cold packing has, thankfully, fallen out of favor and is no longer used as treatment. However, in a 2010 paper affiliated with Harvard medical school, “concerns of patient safety” are cited first for justified use of restraints. While safety is a legitimate concern, as patients who are severely ill can remove IVs and the like, I would question how often someone on a psychiatric ward would have an IV? This article also cites staff safety as a concern, but I must bring back Elyn’s words that question why someone would work in this environment if they are not trained to deal with the minds and actions of the mentally ill? Lori and Elyn’s experiences with psychiatric healthcare are

indefensible. No human should ever undergo such treatment. To further prevent this from happening to others, society must take our prior, ineffective policies and use their torment as implementation of education and *change*.

The Sun will Rise: Conclusion

While there are plenty of issues that continue to persecute the mentally ill, particularly those with schizophrenia, I must mention that there are plenty of reasons to hope. This disease was once regarded as being possessed by demons, and while I am sure that Elyn, Lori, or Kurt would not argue against that when their illnesses were at their worst, we now understand that it is a brain disease, one of broken connectivity. This discovery alone has allowed treatment to be revitalized in nearly every aspect: from electroshock and psychoanalysis to antipsychotics, cognitive behavioral therapy, assertive community treatment, etc. Being able to control symptoms with more effective treatment methods means that societal issues that individuals with schizophrenia are forced to bare, namely stigma, can be researched and further understood. Education can be provided to the masses, ensuring inaccurate beliefs, opinions, and interpretations are corrected-- putting an end to the plight of stigma.

The Ever After

I must conclude the stories that I have shared throughout. Thus far, I have shared mostly the low points from the memoirs of Elyn, Lori, and Kurt. To be fair, the majority of the pages of these books are rife with madness, struggle, and strife. Nevertheless, each author includes an epilogue, the final chapter of hope.

After Elyn's break in law school she went on to find her passion in mental health law, as evidenced by her note on restraints. That note was the beginning of something

wonderful for Elyn. After establishing that working at a stressful law firm was not ideal for her, Elyn began to teach law. Today, Elyn teaches at USC Law School where she has achieved tenure. She continues to educate others on living with schizophrenia. Of her recovery Elyn shares,

“My life today is not without its troubles. I have a major mental illness. I will never fully recover from schizophrenia. I will always need to be on antipsychotic medication and in talk therapy. I will always have good days and bad, and I still get sick.

But the treatment I have received has allowed me a life I consider wonderfully worth living. My good fortune is not that I’ve recovered from mental illness. I have not, nor will I ever. My good fortune lies in having found my life” (Saks, 2007).

Lori Schiller lost everything in the midst of her illness. Therefore, she faced a long recovery. She had to recreate her life in almost every respect: losing the inherent fear of people was the top priority, followed by learning to be social, then taking on budgeting, cooking, cleaning, even taking her medications on her own. She recognizes that the process took a while, and she spent three years in a halfway house to gain the skills she needed to live on her own. Interestingly, Lori touches on what it was like to live without the Voices that were so cruel to her for so long. She recalls, “I found to my surprise that I missed them... My head felt empty. Without them I felt lonely... The Voices had dominated my head for so long that they had left no room for any other thoughts” (Schiller, 1996). This I found completely heartbreaking. Lori had become a victim of Stockholm syndrome. Nevertheless, with the help of support groups and

counselors Lori was able to create a life without the Voices. Today, Lori works at an outpatient treatment center, she notes that she is a staff member who understands both sides of the fence. Lori lives happily in Florida with her husband.

Kurt Snyder recognizes that medications have helped him reclaim his life from the depths of paranoid psychosis. On his recovery he reports,

“The improvement in my mental health status has been gradual but continuous, and I have not had any clinical symptoms of schizophrenia for at least four years. I also seem to be progressing in in other ways that are not easily classified by a doctor. My productivity and outlook on life all seem to improve as time passes. Most people who meet me now would find it hard to believe that I ever had a mental illness. Many are surprised when I tell them I have schizophrenia” (Snyder, 2007).

Kurt continues by citing the importance of social support in his recovery: “Most of my friends and family who knew about my mental problems were still very close to me--they never abandoned me. This was truly a blessing” (Snyder, 2007). Today Kurt works as a database administrator and president of his volunteer fire department in Maryland.

Aunt Linda

After reading the testimonies of Elyn, Lori, and Kurt and upon my visit with my Aunt Linda, I began to wonder about the details of her life. When did she first show signs of illness? Did she experience a dramatic psychotic break? Upon talking with her sister Carol, my Auntie Carol, I began to puzzle the pieces of her life together. She was apparently always “odd” in childhood, consistently speaking of a ghost around their home she named Alabaster. She went on to study business at Ball State University, but

became delusional in her third year with regards to a certain professor whom she had apparently fallen in love with. She returned home, believing that he would follow her. She lived at home with her mother for a while before moving away again. Naturally, she was never able to hold down a job, so she was forced to return home to live with her mother once more. It was around this time when she received her diagnosis. Interestingly, but not really surprising, is the fact that the doctor had to make a house call, as she refused to go to the hospital defending, "there was nothing wrong with her." Eventually she was able to move into a small apartment on her own in town after my Grandma Hawkins had taken ill. Throughout this time period she was not on antipsychotics or receiving any treatment. Consequently, her delusions ran wild. She brought Christmas presents for her deceased father during the holidays and firmly believed her mother to be dead, when in reality Grandma Hawkins was suffering from severe Parkinson's. Nevertheless, my Auntie Carol continued to manage her finances and take her to get groceries with her every Saturday. I would often join them on their trips to the grocery. In fact, some of my very earliest memories are grocery shopping with Auntie Carol and Aunt Linda at the JayC food store in Paoli, IN.

Aunt Linda continued to live in her small apartment in Paoli for many years. She apparently had consistent catatonic spells, only occasionally going outside to walk or interact with others. As I previously mentioned, she was not taking antipsychotics, and she lived alone. While this somehow worked out for several years, her symptoms inevitably began to flare. My Auntie Carol would get calls from neighbors that she had, without invite, walked into their house and was rummaging through their belongings. These symptomatic flare ups kept happening, and Auntie Carol was forced to have her

committed to the psych ward in nearby Jasper, IN. From there she was relocated to her current group home. She is now on a steady regimen of antipsychotics and lives in a place where she is surrounded by others. She also joins the family on Christmas and Thanksgiving and sends each of her six nieces and nephews a birthday card every year.

While Aunt Linda's life is probably much better than before, I wonder what other symptoms she might have suffered from. Did she endure threatening, evil voices? Did she ever fear for her life due to incessant paranoia? I have only visited her one time in the 13 years that she has lived in Jasper. Two of her siblings visit occasionally but not often. I am saddened that she lives a very lonely life, duely filled with boredom. Therefore, I think I have a responsibility to begin making it a priority to visit her more often with my Auntie Carol. We can rekindle our conversation on Plato and Aristotle, or maybe just discuss the plot of a Danielle Steel novel. It is my duty, now that I consider myself somewhat of an expert on Aunt Linda's illness, to correct the insensitivities from the rest of my countrified, Southern Indiana family. Linda is not "batshit crazy." She is mentally ill and deserves respect from each of us. Because when someone sends a birthday card every year, they are not crazy. They care. The least we can do is the same.

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*“Of course it is happening
inside your head, Harry, but
why on earth should that
mean that it is not real?”*

-Albus Dumbledore, Harry Potter and the Deathly Hallows