The Creative Advantages of Schizophrenia

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The Muse and the Mad Hatter

By

Paul Kiritsis

Cambridge
Scholars
Publishing

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This book first published 2019

Cambridge Scholars Publishing

Lady Stephenson Library, Newcastle upon Tyne, NE6 2PA, UK

British Library Cataloguing in Publication Data
A catalogue record for this book is available from the British Library

ISBN (10): 1-5275-3165-1
ISBN (13): 978-1-5275-3165-9

For Christos Stamboulakis

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ACKNOWLEDGEMENTS

Fig. A-1. Author Paul Kiritsis and his mentor Dr. Brooke Schauder.

First and foremost, I wish to thank my family for their unwavering and unrelenting support over the years. None of this would have been possible without you. Special mention to my doctoral mentor and methods expert, Dr. Brooke Schauder, for her *bona fide* input and constructive criticism, support, and communication during the rigorous dissertation process. Moreover, an expression of gratitude to my other two dissertation committee members, my chairperson Dr. Marilyn Schlitz of Sofia University, and my content expert Dr. James Kaufman of Connecticut University, for their guidance and encouragement.

I couldn't have asked for a better team–a clinical neuropsychologist, a social anthropologist, and a cognitive psychologist. Eternal gratitude and special thanks to Dr. Mary Gregerson, my editor, for rendering this manuscript fit for publication. A special mention to Francesca Lalli, my colleague, for her support and friendship during this laborious albeit rewarding period of my life. And of course, my work colleagues at CIP– Cindy Jepson, Lena Grintsaig –for keeping me sane along the way. To my friend Ethan Carr, thank you for your emotional countenance and guidance. Finally, I wish to thank my talented cousin Chris Stamboulakis for impelling me to grow psychologically and spiritually, have the courage to confront my shadows, fears, and vices, and be the best version of myself that I can be.

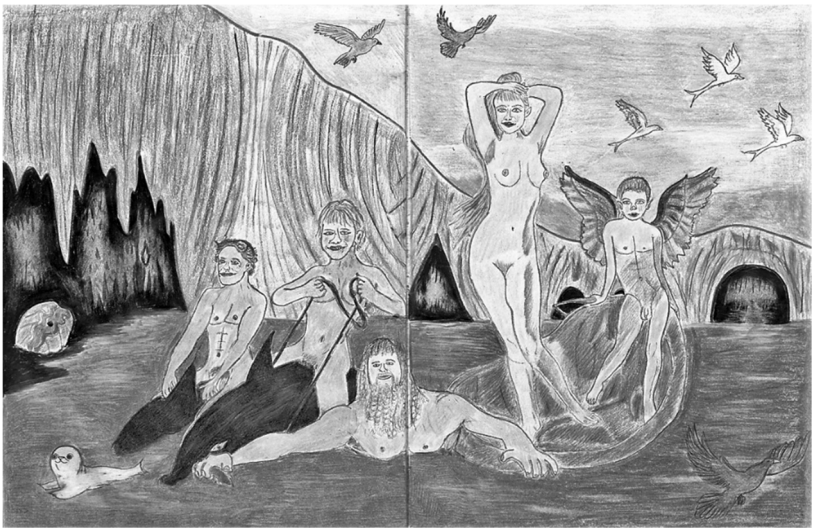

Fig. A-2. The Dissertation Committee. From left to right: Fred Luskin, PsyD chair, as a water nymph; chairperson Marilyn Schlitz, PhD, as a water nymph; committee member James Kaufman, PhD, as Poseidon; committee member Brooke Schauder, PhD, as Aphrodite; and Paul Kiritsis, as Eros.

FOREWORD

The theme of this book is the interface of psychopathology and creativity, a narrative that goes back to at least the time of Aristotle on a sociohistorical, collective level and to my exuberant childhood years on an individual, personal level. In Problemata XXX, the great Hellenistic philosopher broached the topic for discussion when he questioned why melancholic individuals would so often concern themselves with the expressive arts. As a youngster, I, too, asked myself a rudimentary, unsophisticated version of that question after observing, time and time again, my beloved cousin as he endured ungrounding throes of psychotic decompensation interspersed with arduous bouts of creative, task-focused productivity. It was so hard to make sense out of what was happening to him; the periods of confusion, dysregulation, imaginative hyperassociation, and ebullient emotion, juxtaposed by equipoise, clarity, and lucid nonverbal narratives. It was all too confusing and overwhelming for my inchoate, prepubescent mind to understand. Everything felt like it was black or white around him; experience was never in shades of grey. He probably felt the same.

My cousin was moved by the natural world but more so by the preternatural and the sinister, murkier aspects of the human condition. At his best he was an inspired maverick however at his worst he was reckless and slightly anarchistic too. His visual art was always visceral, carnal, avant-garde, and colorfully evocative. Under the tutelage of his supportive parents and a cooperative inner muse, his hyperactive imagination spewed forth mythological statuettes, ornaments, paintings, and variant genres. He graffitied onto trains and factory walls and allowed goblins, mythological creatures, and extraterrestrial beings which had erupted from his psychic unconscious to come life through visual sequences and narratives. He was extremely versatile and prolific as a creator, using his tutored hands to create an abundance of artistic works in naturalist, surrealist, and more abstract representational styles. I was not allowed entrance into that world unless I jettisoned the trumpet of judgement behind. For this experience I had to be completely naked– armed only with the tools of abandon and curiosity.

In his early adult years, perhaps around the age of eighteen, my cousin started experimenting with psychostimulants like cocaine and methamphetamines as well as depressants like cannabis. At heart an empathic, compassionate, and benevolent individual he found it almost impossible to refuse requests made by acquaintances to provide a homely and relatively spacious venue for communal drug use. From my father's assertions my cousin was an ill-fated victim of peer pressure. "He couldn't say 'no' to save his life," my father professes. "On weekends his parents would sojourn at their beautiful holiday house, and that's when the group festivities involving liberal and copious use of drugs would transpire. He and his friends would gather at the home on Friday and Saturday nights, either drinking or using drugs."

Unbeknownst to his parents, my cousin suffered an acute psychotic break not long after the inauguration of the drug use and it was not long before the benign whispers and driving melodies emanating from the wind and the non-conventional esoteric ideas surrounding creation morphed into poignant multimodal hallucinations and paranoid delusions. Once pregnant with collective meaning, the vivid imagination that had served as both a powerful ally against dis-ease and a viable source of income degenerated slowly into a self-sabotaging demon of faulty reality testing, an entity cocooning itself inside an artificial world with no yoke or thread to consensus reality. The drug use continued intermittently and as the years went by my cousin slipped further and further into the rabbit hole, ensnaring himself in a psychical dimension where the news reporter would communicate ideas and instructions to him directly, "The Others" would not allow him to depict certain scenes and images artistically, and the FBI had installed microchips and undetectable surveillance equipment in the house along with minute video cameras in the backyard in an attempt to discover the most intimate, microscopic details of his life and then use them against him. In the last two decades my cousin has been beleaguered by psychotic symptoms and chemical dependency, at times appearing like an insurmountable, stoic knight in shining armor and at others like an unassuming zebra caught in the lethal jaws of the dreaded African crocodile. All the while I was the silent, nonjudgmental, and seemingly detached third-person observer.

A whole lot of clamorous noise has been made about this topic, and it appears the crescendo may end up culminating as a sonic boom. Is the nebulous swamp that births demons, isolation, stagnation, and perturbation also the cloud that births muses, novelty, transformation, and evolution, and crisis in self-esteem? Do instances of and

propensities for exceptional ingenuity come at a cost? Must descent into the somnolent Osirian chambers be necessary to find gold nuggets with esemplastic powers? Is the barely coherent Mad Hatter sighted in the light of day the Muse which plucks "eureka" strings of revelation and weaves silent but inventive rhythms in the heart of night? Put simply, do I need to flirt with madness to create products that will be praised for their historical inventiveness, aesthetic beauty, unprecedented utility, and the sheer ineffable awe they're able to evoke in the sophisticated and layperson alike. The forbidden fruit which contains that seedy *gnosis* continues to dangle on branches above our heads, tantalizing us because the answers are seemingly within our grasp, but moving further and further away like a peripatetic rainbow each time we take a step towards this object of desire and philosophical interest.

As with anything ostensibly nomothetic and scientific the creativity-psychopathology theory, simply embodied by the "mad genius" archetype in the vernacular, isn't without its sceptics and detractors. In fact, there's no shortage of scholars who have made it their life's goal to discredit the theory altogether and relegate it to the same dustbin of pseudoscience the geocentric model of the universe, astrology, and phrenology now lay forgotten in. But behind every mental microscope is a mind colored by emotional subjectivity, and the reasons they have for clinging fiercely onto their sentiments probably has very little to do with any "objective" data in the stricter operationalization of the word and more to do with their own internalized ontological-epistemological position and psychical disownment of things that can't be reconciled with it. Of course, I, too, am by default hampered by this human limitation and thus guilty of partisanship.

From what I've read and from what I've experienced as both an untutored eyewitness and a burgeoning clinician trained in psychological theory and praxis, I am in favor of a veridical connection between the schizospectrum, bipolar, and substance abuse disorders and creativity [The Shared Vulnerability Model of Creativity and Psychopathology]. I believe existing clinical and biohistorical data fit neatly into this model with its profound explanatory powers.

INTRODUCTION

Not all people in this world are equally *creative*, and by the latter I mean having an eagle eye for the novel and unique. An act of creation entails developing a novel product within a sociohistorical context that is deemed to be both useful and beneficial, a novel product able to succeed an existing model and make because of its superior or more sophisticated design, its improved aesthetic appeal, or its augmented heterogeneity when it comes to general usefulness. It just so happens that a high proportion of these people, not all, but an incidence rate that piques human curiosity, happen to have schizotypal personality traits.

Schizotypal, of course, does not mean schizophrenic. Conceptually they're two different things. Schizotypy is a stable trait representing a theoretical continuum of personality characteristics and experiences whereas schizophrenia is a diagnosable psychiatric disorder that sits on the extreme end of that spectrum and frequently includes a constellation of symptoms like auditory verbal hallucinations, delusions, disorganized thinking, emotional blunting, and perseveration. A schizotypal individual may be highly imaginative, internally preoccupied, harbour idiosyncratic beliefs and metaphysical assumptions about the universe that are magical or supranormal in nature and be highly artistic and prolific when it comes to the creative enterprise. For example, many schizotypal individuals are prone to hearing voices in the wind and believe in extra-terrestrial life and contact. Typically, they're asocial and introverted, may appear apathetic, and may have salient periods of disordered, nonlinear thinking.

When perceptual excesses and aberrations like subtle microhallucinations (i.e., hearing one's name being called out when nobody is there) and synesthetic experiences or cross-modal percepts (i.e., a saccharine taste in the mouth each time one sees the letter U) predominate, then the schizotypy may be qualified as positive schizotypy–the variant that has been linked with creative productivity as well as artistic and scientific genius. A well-adjusted schizotypal person is a vehicle, instrument, and conduit for the inspired muses to possess, overcome, seize, and work through. These individuals march to the sound of their own drumbeat, sit at the peripheries of

convention, and peer beyond the horizon of culturally conditioned associations and rational analysis so as to make meaningful connections between two previously unrelated concepts through hyperassociation. The great polymath Leonardo Da Vinci, for instance, made a leap of insight by connecting a wood screw and the sail on his air machine to produce an aerial screw, rendering human flight possible. Schizotypal persons may be eccentric in nature, harbingers and martyrs of scientific and artistic change, and shatter existing paradigms. They are frequently cited by historians as being ahead of their time.

Thus far there is a growing body of theoretical, clinical, and experimental evidence corroborating a correlation between creative ideation and positive schizotypy. They may go hand in hand. In fact, it may sometimes be difficult to parse out where one ends and the other begins. But as the proverbial mad genius archetype intimates, people who espouse this kind of perceptual interface with reality are at an increased risk of becoming the Mad Hatter and falling prey to the demons of psychopathology. They usually don't suffer from psychopathology, but may capitulate under the auspices of a hostile, nonempathic, and unforgiving environment. I think we can subscribe to that connection without having to render the sublime and exquisite masterpieces of bygone artists–scintillating diamonds like the Sistine Chapel, *The Persistence of Memory*, and "The Rime of the Ancient Mariner"–as epiphenomena of an unquiet pathological mind, and hence devalue and indignify their sociocultural worth. Adopting a pathological stance doesn't celebrate the complexity and diversity of the human mind and condition.

Using common sense, one might promulgate more progressive views on the schizotypal position and claim that neurocognitive and psychosocial factors are predictive of creative ideation and productivity or protective against the development of fulminant schizophrenia and psychotic decompensation. One such empirical model that adopts this philosophical position is *The Shared Vulnerability Model of Creativity and Psychopathology*. Pioneered by Shelley Carson in 2011, it stipulates that for individuals with shared polygenic roots for both creativity and psychosis, turning towards the muse or the Mad Hatter depends, in part, on their general intellectual ability (IQ), working memory skills, and cognitive flexibility.

I vividly recall my punitive scepticism when I first stumbled upon this model. How could manipulating a variable like working memory protect an individual with a predisposition to schizophrenia from full-blown psychotic symptoms and at the same time harness creative

potential and activity? Was there any veridicality to Carson's model, did it in fact irradiate, evoke, or speak of an existing pattern in Nature intuited by romantic poets and writers of ages bygone, or was it merely the product of hope, wishful thinking, imagination, selection bias, and the practice of sloppy science [pseudoscience] all amalgamated into one? Had Carson been infected by the mad genius virus, just like Kay Jamison and Nancy Andreasen before her?

Strongly resisting the powerful urge to arrive at premature judgements and conclusions, I began researching the creativity-psychopathology link and swiftly found that the idea has persisted since the times of Plato. In reference to creativity, the eminent philosopher wrote that creative inspiration is like a divine madness… a gift from the gods themselves. I immediately became captivated by this profound transcultural impression and voraciously devoured everything I could find on the topic. It wasn't long after that I started to see how the adroit Carson had sewn together her model using existing empirical data from multiple domains of inquiry (i.e., genetics, cognitive neuroscience, psychology) as well as anecdotal accounts. According to Carson, a case could be made for a connection between creativity and three types of disorders: the schizospectrum disorders, the affective disorders, and the substance-abuse disorders. All the factors identified by her theoretical model–irrespective of whether they are protective or indicative of shared vulnerability–are substantiated by empirical data. Until now the emphasis and spotlight has been on neurocognitive factors, however there can be no question that psychosocial elements (i.e., effects of war, poverty, level of interpersonal attunement, emotional intelligence, visuospatial intelligence) also play an integral role in determining outcomes. These lines of inquiry are awaiting exploration and will probably precipitate another increment in the model's degree of conceptual sophistication.

Before long I had concluded that testing the empirical validity of Carson's eloquent *Shared Vulnerability Model of Creativity and Psychopathology* would be the topic of my doctoral dissertation. I espoused a longstanding interest and fascination with the schizospectrum disorders [schizophrenia], so it made sense to narrow and focus my efforts on that clinical population. One afternoon, while perusing some very impressive art in the surrealist tradition, an earth-shaking thunderbolt flashed in my mind's eye as I experienced the proverbial Eureka moment. "I know what I'll do! I'll subject a sample of individuals with a DSM diagnosis of schizophrenia to computerized cognitive training and see whether enhancements in working memory capacity

and mental flexibility are concomitant with improvements in mean creativity task performance as measured by pre and post psychometric tests of executive functioning and creativity." I surmised that if executive functions (i.e., mental flexibility, working memory capacity) were intimately linked with efficiency and effectiveness at expressing inherent creative potential, then what we should see in the aftermath of the cognitive intervention are significant improvements on all stipulated measures.

There were three specific hypotheses connected with my empirical investigation; each postulation conformed strictly to the empirical data and the implications about what should in fact occur in the aftermath of variable manipulation. The three interconnected hypotheses were: (a) riddled by cognitive deficits, the cohort of individuals with schizophrenia will perform much poorer than healthy controls on all measures for executive functioning and creativity before the intervention, (b) there will be significant post-intervention improvements for the cohort of individuals with schizophrenia on all measures for executive functioning and creativity, and (c) the cohort of individuals with schizophrenia will end up outperforming healthy controls on the aforementioned measures after they complete their computerized cognitive training program.

The computerized cognitive training sessions were done on a downloadable evidence-based program called Cogmed which the Vinogradov Schizophrenia Research Lab at UCSF promoted and helped commercialize. The cohort of individuals with schizophrenia received two sequential modules of active computerized cognitive training exercises, 1 hour per day over five days per week [until the full 50 hours of training was completed], while the control group comprised of healthy individuals did not receive the intervention. The rationale for not giving healthy controls the cognitive intervention should be blatantly obvious–the integrity of neural systems subserving their executive functioning is not compromised or impaired. Hence the exercises would be redundant, futile, and a lamentable waste of time and energy.

The training exercises themselves encompassed two specific modules: a module of auditory/verbal processing exercises and a combined module of visual processing exercises and emotion identification exercises. Contained in the latter was training in facial emotion recognition and theory of mind. Participants in this group spent approximately 33 hours of engagement with the auditory/verbal training exercises and approximately 17 with the rest. Trainings for the

computerized cognitive intervention involved a parametric and systematic increase in task difficulty as performance improved.

Naturally there would be profound clinical and social implications for individuals diagnosed with schizophrenia if my empirical findings ended up supporting the aforementioned hypotheses. Perhaps a positive result would aid in the war against the stigmatization of mental disorders or mitigate prejudice against individuals whose lives have been compounded by this psychodiagnostic label; perhaps it would announce the arrival of a collateral cognitive intervention, a possible adjunct to pharmacotherapy, psychoeducation, and psychosocial support, able to assist in the rehabilitation, remediation, and reintegration of the ailing individual into contemporary society. The possibilities were endless in light of auspicious data.

So, what were my findings, you ask? My preference is to keep you in suspense and so for that you will have to wait, at least until the chapter which describes the study results.

This book is divided into seven individual chapters. The first chapter, "Schizophrenia as a Social Construct," contains a general discussion about schizophrenia and its symptomology; the impact of schizophrenia on individual sufferers; and a brief history of schizophrenia. In this chapter the psychiatric disorder is operationalized using criteria outlined in the DSM-V.

The second chapter, "The Treatment of Schizophrenia," looks at treatment. It describes the pharmacotherapy tradition briefly before going on to cognitive therapies for schizophrenia. Special emphasis is given to computerized cognitive training programs, given the latter was the intervention for this research study. Following this will be a systematic examination of empirical studies that have used computerized cognitive training to improve working memory performance, self-relational processing, and cognitive flexibility or set shifting in individuals suffering from schizophrenia.

In the third chapter, "Creativity: Definition, Research, Measurement," the concept of creativity is operationalized and placed within a sociohistorical context. Empirical approaches to the study of human creativity are described with explicit emphasis on Guilford's dominant and orthodox psychometric tradition. Even though vast majority of creativity research has been conducted using much refined and comprehensive psychometric methodologies, the agenda of the present study remains faithful to Amabile's Consensual Assessment Technique (CAT), the "gold standard" of real-world creativity assessment.

Following this is the fourth chapter titled "The Muse Meets the Mad Hatter: Creativity and its Relation to Schizophrenia" where the proverbial mad-genius link–transposed as the creativity-psychopathology connection in our time–is explained in the context of the schizospectrum disorders. Creativity is identified as a trait with compensatory advantages able to account for the survival of schizophrenia in the gene pool. Then there is a brief look at some psychobiographical research linking creative luminaries and extraordinary persons to schizophrenia before moving onto the empirical literature: population studies, family and adoption studies, clinical studies, and psychometric studies in non-eminent and non-selected populations. Following this review, the disparities between definitions and criteria for psychiatric evaluation, methodologies, and conclusions across studies is discussed [methodological limitations]. This section concludes with theoretical implications pertaining to the consistency of empirical finds linking psychosis and schizotypal traits to creativity.

The latter encompasses a conceptual anchor to Carson's (2011) Shared Vulnerability Model of Psychopathology and Creativity, one interface model of the creativity-psychopathology connection which informed the rationale, hypothesis, and choice of intervention for the present empirical study. In this fifth chapter, titled "The Shared Vulnerability Model of Creativity and Psychopathology," cognitive flexibility, IQ level, and working memory capacity are identified as protective or vulnerability factors which determine whether an individual with polygenetic linkages to creativity, psychosis-proneness, and schizotypal traits will manifest the inner muse or inner fires of psychopathology. Moreover, theoretical particulars like the relationship between cognitive traits and joint vulnerability factors like attenuated latent inhibition, preferences for novelty, and neural hyperconnectivity are fleshed out in detail.

The sixth chapter, "The Golden Thread: The Marriage of Positive Schizotypy and Creativity" discusses the findings of my dissertation research. It also presents some caveats pertaining to the study's delimitations and limitations as well as viable avenues for future research.

Succeeding this section is "Clinical and Social Implications," the seventh and final chapter which offers a pithy description of the study implications. Several clinical, social, and personal applications are discussed here.

CHAPTER ONE

SCHIZOPHRENIA AS A SOCIAL CONSTRUCT

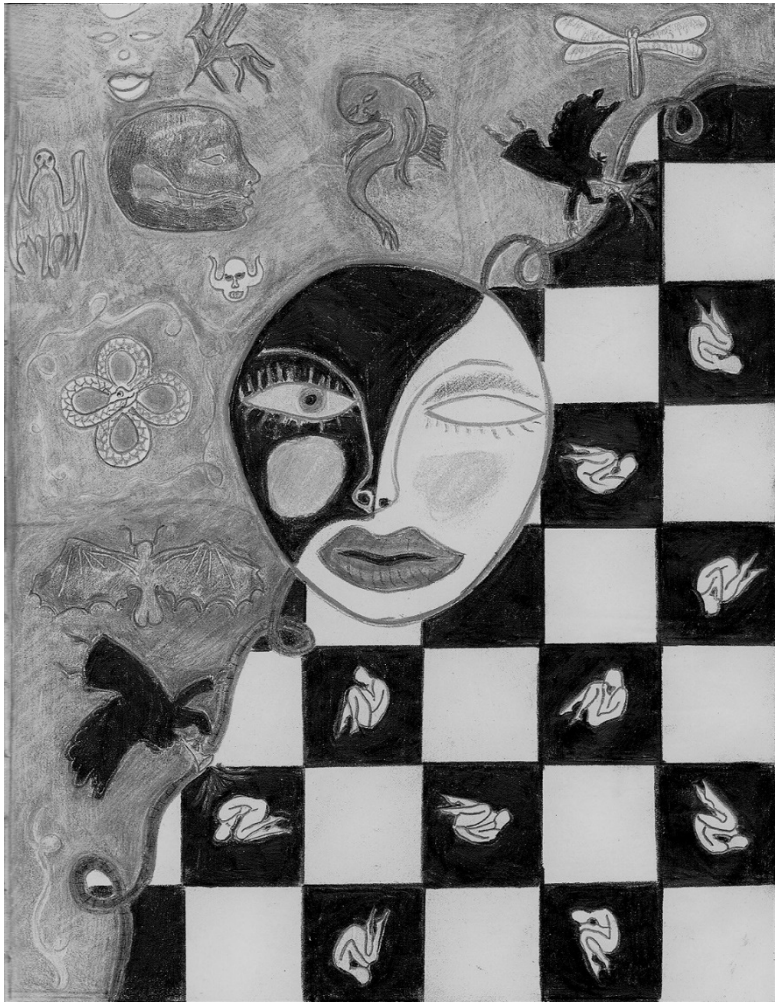

Fig. 1-1 Visual representation of reality as experienced by an individual with schizophrenia.

Derived from the Hellenistic words *skhízein* and *phrḗn* (literally, split-mind), schizophrenia is a complex heterogeneous disorder wrought by both genetic and environmental factors, relegating affected individuals to suboptimum levels of functioning in personal, social, and vocational domains. There is prodigious stigmatization and misapprehension connected with this psychiatric disorder and it is undoubtedly the most incapacitating in terms of the psychological, social, and financial damages it inflicts upon both those individuals unfortunate enough to develop any of its diagnostic hallmarks and their primary caregivers (Kingdon & Turkington, 2005). It affects approximately about 1.1% of the world's population and accounts for about 25% of all mental health care expenses, making it a leading cause of disability worldwide (SARDAA, 2018). About $32.5 to $65 billion are disbursed for the management and treatment of schizophrenia annually. Of the afflicted, 28% continue living independently, 25% are under the supervision of a guardian or caregiver, and 20% subsist in supervised cottages (schizophrenia.com, 2010). The rest are not as fortunate and rotate among the less-than-optimal spaces for convalescence offered by state hospitals, prisons, and shelters. Without a doubt, the greatest cost is not financial in nature but emotional—no monetary sum can compare to the disillusionment, demoralization, and emotional turmoil that patients with schizophrenia and their families have to endure.

Unlike the biased prerogative and opinion the aggregates would have us believe, chronic sufferers are not plagued by homicidal ideation nor prone to random bouts of violent enactment against others. In actual fact, threats stemming from possible self-deprecation and self-harm are much more veridical possibilities than threats motivated by homicidal impulse or being of danger to others (schizophrenia.com, 2010). When juxtaposed with the general population, individuals with schizophrenia exhibit a higher than expected mortality rate with heightened risk of attempting suicide accounting for at least part of the differential (Kingdon & Turkington, 2005). Recourse to suicide may be understood as an urgent, severe, and obviously ill-informed attempt at absconding the throes of depression, psychosis, and potential relapses that characterize the nosological trajectory of the disorder. Onset occurs during late adolescence or early adulthood with the incidence rate being minutely higher amongst men; men also tend to experience heterogeneous psychiatric symptoms developmentally earlier, usually 3 to 4 years before their female equivalents, who tend to develop it around the 25-year age mark (Oltmanns, Martin, & Neale, 2011). Apropos of gender differences in prevalence, the disorder is more

common amongst men and it is this demographic group that is particularly at risk of committing suicide. Increased risks have also been determined for individuals born and raised in urbanized areas (Mortensen et al., 1999), immigrants of color who have relocated to a predominantly white urban neighborhood (Boydell & Murray, 2003), and African-Americans (Bresnahan et al., 2007).

The prognosis is variegated and has remained stable over time. The pessimistic tendencies expressed by those clinicians who paint a disconsolate, hopeless, and grim picture of individuals suffering from schizophrenia and attribute a chronic deteriorating course to the disorder can be traced back to the 18th-century German psychiatrist Emil Kraepelin. Kraepelin was instrumental in developing the whole concept (then known as *dementia praecox*). His fixation with the then in-vogue idea of psychological degeneration played a seminal role in his theorization and conceptualization of the disorder, and he eventually emphasized deterioration of intellectual functions and mental disability to the exclusion of factors that may have suggested more optimistic prognoses (Bentall, 2003).

It would be imprudent to think that those early impressions have not shaped the conventional, orthodox teachings that a significant percentage of patients will suffer progressive deterioration in cognitive, social, and vocational functioning and continue to cycle through psychotic episodes throughout the remainder of their lives (Oltmanns et al., 2011). Nonetheless, opposing this pessimistic outlook is a body of evidence vindicating that about 20% to 25% of patients convalesce without subsequent indications of residual languishing (Heiden & Häfner, 2000). In many empirical investigations, including one entitled *The International Study of Schizophrenia* conducted by the World Health Organization (Hopper, Harrison, Janca, & Sartorius, 2007), it was found that roughly 60% of sufferers remain chronically ill with little remission whilst 25% make a complete recovery (Heiden & Häfner, 2000). Pharmacological and psychological interventions also impact clinical outcomes, especially if these are deployed during the prodromal phase where withdrawal and social isolation become especially salient (Oltmanns et al., 2011).

Juxtaposed with the developing countries, social outcomes for the schizophrenic disorder in the developed countries is generally poor, with the nosological consensus stressing chronic degeneration, mortality rates, and patterns of relapse. One feasible explanation attributes the discrepancy to sociocultural and religious factors; whereas the hegemony of the Western orthodox sciences has been to

identify the measurable with the veridical and objective, the complex nonphysical systems and models of mind-matter interaction endorsed by many non-Western societies emphasize the reality of nonphysical dimensions, at times giving them preferential status over the physical or material world. Informed by this epistemic framework, the developed Western world pathologizes idiosyncratic sensory perceptions like voice phenomena (considered to be a major hallmark of schizophrenia), in effect invalidating those individuals who experience them, whereas alternative cultural and religious models encourage and support these same individuals to develop healthy, meaningful relationships with the voices as to aid coping and distress tolerance (Jablensky & Sartorius, 2008; World Health Organization, 1973). Perhaps, then, outcomes for patients suffering from schizophrenia are better in the developing countries because their paradigms of mind and self attempt to understand and process these phenomenal experiences rather than slap the stigmatizing label of *madness* on them and then attempt to suppress them with neuroleptics.

Research in behavioral genetics has unearthed invaluable evidence supporting the contention that nature plays a significant role in determining who develops schizophrenia (Mitchell & Porteous, 2011). A review of 11 methodologically sound family studies indicated that the likelihood of developing the disorder is 10 times greater for first-degree relatives of patients with schizophrenia than for nonpsychotic controls (Sullivan, Owen, O'Donovan, & Freedman, 2006). Twin studies have spawned congruent results, with monozygotic twins manifesting a much higher concordance rate when it came to developing schizophrenia than dizygotic twins (Cannon, Kaprio, Lönnqvist, Huttunen, & Koskenvuo, 1998; Pogue-Geile & Gottesman, 2007). Lately, a more nuanced view of the genetic basis of schizophrenia has emerged with important thinkers in the field agreeing that susceptibility is polygenetic in nature, with candidate genes playing a cumulative role in the expression of its phenomenology (Sullivan et al., 2006). To date, two candidate genes, neuroregulin 1 and dystrobrevin-binding protein, have been implicated in the etiology of schizophrenia (Owen, Craddock, & Donovan, 2005).

Nonetheless, the dearth of absolute concordance rates amongst monozygotic twins, that is twins that share 100% of their genetic material, can only mean that liability for developing schizophrenia must also partly depend on environmental factors like social isolation and psychosocial stress, thus triggering epigenetic mechanisms to express those genetic mutations responsible for the known psychiatric

symptoms (Boydell & Murray, 2003; Gottesman & Hansen, 2005). One impressive study recruited more than 300,000 Israeli adolescents to scrutinize relations between population density and genetic risk factors, finding that genetic and neurobiological vulnerability somehow interacted with psychosocial stressors to produce schizophrenia (Weiser et al., 2007). Another quantitative analysis revealed that use of cannabis in adolescence precipitates the likelihood of suffering a psychotic episode in persons with a preexisting liability (Henquet, Murray, Linszen, & van Os, 2005).

Apropos of etiology, complementary research finds in twin, family, and adoption studies have substantiated the dynamic interaction between genetic and environmental factors in schizophrenia to the degree that researchers were left with no choice but to propound a hybrid theoretical model subsuming biological, psychological, and social conceptualizations. Armed with greater explanatory power than any of its domain-specific predecessors, it considers any particular psychotic symptoms and symptom clusters (i.e., voices, delusions, short-term memory deficits, poor attention, etc.) one might experience phenomenally to be determined by the idiosyncratic nature of one's own vulnerabilities and stress (Kingdon & Turkington, 2005). To give an example, an individual with vulnerabilities from genetic weighting, rigid cognitive schemas, and a characterological predisposition to rumination and engagement with magical thinking may become floridly psychotic after suffering successive environmental stresses including cruel rejection at the hands of a potential lover, loss of a secure job, and a traumatic incident like a violent rape. The adverse feelings of shame, guilt, and judgment; the social isolation; and the subsequent ingestions of a hallucinogenic drug made in a desperate attempt to escape the excruciating pain and suffering may exacerbate the psychotic symptoms, thus further compounding the issue. Despite its somewhat crude and unrefined state, this model is promising and explains many recent research findings pertaining to schizophrenia (Kingdon & Turkington, 2005).

Then there is the (over)emphasis and uninhibited use of pharmaceuticals in treatment, an approach that unleashes a different assemblage of issues. What could the prodigious use of neuroleptics in psychiatry intimate other than the 19th-century absolution that the etiology of mental illness is exclusively biological in nature? Conceptually antiquated and unsound, this paradigm reckons schizophrenia to be a distinct entity marked by clinical and biological consistency across the species—something akin to a malignant brain tumor or a congenital

abnormality like sickle-cell anemia. If this were the case, then a coherent, consensual formulation of schizophrenia based on a unified pathophysiological model ought to have emerged by now. But it has not. Riddled by methodological inconsistencies and failures to replicate physiological and anatomical findings (Williamson, 2006), a century of grail quests for common neurobiological factors and a unified pathophysiological theory has come up empty-handed.

Moreover, neuropsychological findings and longitudinal neuroimaging are not in concordance with a postulate of schizophrenia as a dementing or neurodegenerative brain disease; there is no evidence that progressive cognitive decline continues to occur after psychotic decompensation, or that decrements in the ventricular and cortical brain volumes of individuals with a diagnosis of schizophrenia are precipitated by an insidious underlying pathophysiology (Zipursky, Reilly, & Murray, 2013). If a unified pathophysiological model cannot be identified, then the functional changes observed may, *a fortiori*, be explicable in the context of substance abuse, polypharmacy, and other ancillary factors. Despite the manifestation of deficits and developmental abnormalities observed, persons with this debilitating disorder can achieve substantial rehabilitation of function in cognitive, interpersonal, and vocational domains (Zipursky et al., 2013).

Nonetheless, putative neurobiological research in the guise of postmortem neuroimaging investigations of the brains of individuals diagnosed with schizophrenia has unveiled commonalities, especially in the frontal, medial, and anterior cingulate regions (Jung, Jang, Byun, An, & Kwon, 2010). The postmortem brain tissue revealed reduced densities in the input layers of the prefrontal cortex, an area permeated with pyramidal cells. There was also consistent demonstration of ventricular enlargement along with temporalimbic abnormalities where reduced hippocampal volume is accompanied, somewhat paradoxically, by neuronal hyperactivity in the hippocampal and para-hippocampal areas (Blom & Sommer, 2010).

Typically, these structural abnormalities would coexist with corresponding functional abnormalities. According to a fine-scaled analysis of 12 functional neuroimaging studies (Glahn et al., 2005), task-related activation involving working memory was substantially altered in individuals with schizophrenia: Compared with a control group, they displayed abnormal hypoactivity and hyperactivity in multiple brain regions. The stipulated physiological and anatomical discrepancies all imply neurocognitive alterations to self-related processing; to loss of inhibitory, top-down control over maladaptive

cognitive and emotional behaviors; and to perseveration or deficiency when it comes to volitional switching between thinking modes (Kyaga, 2015). This idiosyncratic perceptual style is accompanied by salient impairment across all cognitive domains (attention, verbal and working memory, semantic language, and processing speed), as evidenced by neurocognitive studies juxtaposing the standardized performance rate of clinical and nonclinical samples, on various aptitude tests (Reichenberg & Harvey, 2007). As eruditely echoed by Heinrichs (2005), the substantial gap between patient and control groups on neurocognitive aptitude is not reflected in the neurobiological correlates, meaning that discrete psychosocial stressors imposed by the social environment are a powerful determinant in selecting which genetically vulnerable individuals will lapse into schizophrenic psychosis. The brain is a socially mediated organ, and the larger discrepancies found in neurocognitive domains reflect that truth.

Despite the phenomenal heterogeneity seen across persons with schizophrenia, the clinical bias is to subdivide according to symptom dimensions: positive symptoms, negative symptoms, and cognitive deficits and disorganization (Andreasen, Arndt, Alliger, Miller, & Flaum, 1995; O' Leary et al., 2000). Positive symptoms, the core diagnostic marker used by contemporary psychopathology, refer to perceptual excesses like hallucinations and delusions. Hallucinations primarily occur in the auditory modality as voices that are qualitatively distinct from thoughts or inner speech (Blom & Sommer, 2010); however, they can also be visual and tactile. These may be primary or secondary to negative symptoms, otherwise operationalized as perceptual absences. Delusions beleaguering patients are often characterized by thought broadcasting; thought insertion, withdrawal, and transparency; grandiosity; and persecution (Kingdon & Turkington, 2005). The continuum of negative symptoms encompasses restricted or blunted affect, alogia, avolition, anhedonia, and extreme social withdrawal—all of which may precede or succeed medication side effects and depression. The degeneration of executive functioning indicated by disorganized speech, nonlinear and disordered thought patterns, short-term memory deficits, and bizarre behavior comprises the third dimension and may further compound existing feelings of dejection and isolation. There is much interest surrounding the differentiation, classification, and operationalization of symptoms and symptom clusters so that persons suffering from schizophrenia may be further divided into subgroups; however, these have not really been of any notable benefit or value when it comes to predictive or etiological

validity (Helmes & Landmark, 2003).

Of the configuration of symptoms described, most would concur that the auditory hallucination phenomenon is by far the most stigmatizing, demoralizing, and distressing feature of the condition (McCarthy-Jones, 2012). Are voices pervasive? After perusing the exhausting, voluminous literature that examines prevalence rates for auditory verbal hallucinations in this clinical population, McCarthy-Jones (2012), of the Macquarie University Centre for Cognitive Science in Sydney, Australia, estimated a weighted average prevalence rate of 70%. Despite the idiosyncratic nature of subjective phenomenal experience, studies on auditory verbal hallucinations have identified ubiquitous qualities, themes, and contents that have remained stable over time (McCarthy-Jones, 2012). Typically voices heard by patients with schizophrenia are malicious and hostile, diabolical even (e.g., "You're fat, ugly, and useless"; McCarthy-Jones, 2012). Mutable in diction with a simple, limited vocabulary, voices express themselves as either single-word expletives ("shit, damn," etc.) or flagrant running critiques that undermine, threaten, ridicule, and beleaguer the patient (e.g., "you are stupid," "we'll come for you if you don't"). They are couched in somatic concerns (e.g., "get the milk") and complaints and issue outrageous commands (e.g., "burn yourself," "kill your husband and daughter"). Pronoun use indicates that many voices are appraised as discrete and separate identities (e.g., "He's in bed," "We're going to kill you") though corroborating evidence to support this contention never comes to light. Most negative voices, if not all, have been a primary reason as to why sufferers question their own sanity and decompensate. As one female voice-hearer has articulated with fervor, "You can't have twenty people screaming at you constantly without going to pieces in a little while" (Van Dusen, 1972, p. 148).

A return to late 19th-century Germany is essential for a clearer understanding of how the concept of schizophrenia evolved, a time when the inaugural medical field of biological psychiatry was about to undergo a rapid metamorphosis. Sometime in 1883, the German psychiatrist Emil Kraepelin (1856-1926) published the first edition of his book, a work entitled *The Compendium of Psychiatry* (Bentall, 2003). In the second edition he fleshed out some original ideas on the classification of psychiatric disorders, and it is these that have had a far-reaching, revolutionary impact on the field. Even though he studied under the tutelage of the celebrated philosopher Wilhelm Wundt, Kraepelin's approach to mental illness was quintessentially biomedical. Some core assumptions propounded were that mental illnesses are