

CHAPTER I

“Welcome to the Machine”

My research interest in schizotypy and schizotypic psychopathology crystallized in a critical clinical moment during my clinical training at The New York Hospital–Cornell Medical Center/Westchester Division. I had the good fortune to be there when trainees (both PhDs and MDs) were still taught to “talk to their patients”,¹ that is, to try to understand the experience of a patient and to attend to his or her intrapsychic and interpersonal processes (in addition, of course, to learning all about modern classification, diagnosis, and psychopharmacology). There, we were asked to conduct psychiatric interviews with patients in a weekly case conference. The patients for the conference were selected from the case panels of one’s peers, and the goal of the exercise was to gain a diagnostic impression and understanding of the patient by interview. One’s interviewing style was, of course, subject to considerable scrutiny and critique by both one’s supervisors and peers. One day, I was to be the interviewer, and I met the patient selected for interview outside the conference room. He seemed rather emotionally flattened (but not depressed), dressed somewhat oddly (with an oversized jacket on

¹By the late 1980s the impact of a managed care approach to health care in the United States was being reflected in the reorganization of psychiatric and clinical psychology training programs. At the time, most emphasis was placed on selecting the right medication for a patient and lesser emphasis was placed on the development of finely tuned empathic psychotherapeutic skills. Although this probably seems rather odd, given what one often thinks of when considering what many clinical psychologists and psychiatrists are *supposed to do*, it was largely true nonetheless. A result has been the emergence of a new cohort of clinicians who actually have relatively weak clinical skills.

a warm day), and he was wearing dark sunglasses (indoors). I introduced myself, shook his hand, and began to describe what would happen in the next 45 minutes or so. He interrupted me to ask, "Are you taking me into a star chamber? Is this the machine? Is this 'Welcome to the Machine?'" I reassured him that the conference room was neither, nor was it the *Camera stellata* from English history. I asked him what he had in mind. He told me that he frequently saw the shape of things change before his eyes and that he often felt that he saw colorful objects sail through his field of vision. "Do you really see these things?" I asked. He responded with, "Sort of yes, sort of no; I don't really believe anything is there and I know things don't really change their shape." "Are you super anxious when this sort of thing happens?" I followed. "Nope, not really, not at all," he said. "Are you really anxious now?" I asked, expecting him to say something in the affirmative (most patients do get anxious before a case conference experience). He responded without any real emotion, "No, I am not particularly anxious, but, can you tell me, is this a star chamber? Is this the machine?" "No, it is not," I replied, though I was perplexed by what he had in mind. I was fascinated by what this young man described as perceptual aberrations. We entered the conference room. He surveyed the trainees seated about the room and asked if he could leave. I responded with an old Harry Stack Sullivan interviewing technique, namely, we sat with our backs to the group in chairs set at an angle to one another. He immediately seemed more at ease. In the interview that followed, it became very clear to me that this fellow did not like being around people (he felt odd, out of place, "different"). He found social contact to be a stressful and aversive experience. His lack of connections to other people in the world was offset by an intense immersion in the inner world of fantasy and unusual perceptual experiences. What was especially clinically interesting was that he was clearly *not* psychotic (i.e., he was *not* suffering from schizophrenia or some other psychotic illness). Quite simply, so began my interest in the schizotype, the experience of the schizotype, schizotypy as a theoretical construct, perceptual aberrations, and the potential connections between schizotypy and both schizotypic psychopathology and schizophrenia.

Getting Oriented

The focus of this book is lessons I learned in the experimental psychopathology laboratory while doing empirical research on schizotypy and schizo-

typic psychopathology. The place to start is phenomenology and description.² I have always found it useful to review several cases that convey the essence of what is meant by schizotypic psychopathology. In our observation of these cases, we consider both the *signs* and *symptoms* of schizotypic psychopathology in the form of vignettes. For our purposes, a *sign* is some behavior, feature, or other characteristic that we can observe with our own eyes, whereas a *symptom* refers to a thought, feeling, or other aspect of psychological life that an individual reports to us (sometimes in response to a question or via spontaneous expression). Let us consider the following individuals. None of these individuals has schizophrenia, nor could all of them be diagnosed with a DSM-IV schizophrenia-related personality disorder, yet they are all schizotypes.

- **Case 1:** Dennis is a 24-year-old single male graduate student who is studying physics at a research university. He leads a socially isolated life and has but one “friend,” with whom he has esoteric “talklets.” Occasionally, he speaks briefly to his peers in the graduate program; they refer to him among themselves as “the loner.” The word *talklet* is one that he uses, and he assumes that a listener will understand its meaning. For him, a talklet refers to a relatively brief conversation with another person. Most of these conversations are short in duration, often rather one-sided, with Dennis simply “speaking *at* someone.” Dennis is rather unaware that others experience his “talklets” in this manner. He claims to feel no strong emotions, feeling neither joy nor sadness ever. The only emotion he seems to feel is anxiety, typically when he is in the presence of other people. He is not so much concerned that he will do something foolish or embarrass himself in front of other people; rather, he simply finds social encounters to be aversive, unpleasant, and unsettling. The anxiety he feels is ever present in all his social interactions, except with his elderly parents. His speech can be difficult to understand due to odd word usage. For example, he speaks of “technicalizing” with his friends, assuming, again, that a listener will know what this word means. According to Dennis, *technicalizing* means talking about anything that “implies a mathematical basis.” He frequently thinks neutral events have “special relevance” for him, for example, he thinks (but does not believe) that shopkeepers set up their window displays with him in mind. Dennis often seems to misperceive aspects of his body, such as think-

²Many psychiatric and clinical psychology trainees jump straight into the intricacies and puzzles of an individual’s life in an effort to understand a patient. Moral: Start simply; start with phenomenology.

ing that his hands momentarily seem misshapen or larger (although, again, he does not believe anything has really happened to his hand).

- **Case 2:** Stephen is a 51-year-old, single male who works for the U.S. Postal Service, typically during the midnight shift. He rarely speaks to his coworkers beyond relatively superficial greetings. Instead, he focuses on “sorting the mail in his cage,” as he terms it, and he normally hums musical tunes quietly while he works. Stephen usually sleeps on the couch in his work clothes after returning home from his job, often leaving the lights on in his apartment. He uses the couch even though he has a comfortable bed. He lives alone, shares his apartment with a small dog, and rarely ventures out except to do errands or go to work. Stephen never speaks to his neighbors, frequently passing them in the hallway without making any kind of eye contact or showing any sort of recognition that another human being is nearby. His face is essentially expressionless most of the time. In conversations at work, his statements are brief, consisting of few words, and at times his expressions are difficult to understand. For example, he said the following to his supervisor when describing how hard it was to open some boxes: “The outer exterior of the box seemed to be expressed outward, which hardens the work with such cardboard structures.” He describes seeing trails of yellow, red, and blue light following behind stars in the evening sky, and he feels these colors have special significance for him, namely suggesting that his inner nature is “astral.” Just what *astral* means is not particularly clear; he is distinctly not New Age in outlook. Stephen is reluctant to go into banks because he feels he might be observed closely, and he is worried that bank tellers might try to “cheat me a little.” When some U.S. currency changed its design, Stephen reported that he didn’t like using the newly formatted currency. In fact, after receiving some “new” 20-dollar bills from the ATM, he entered the bank, uneasily, to request that the bank teller exchange his money, changing the “new 20s” for “old 20s.” When asked why he wanted to do this, Stephen said to the teller, “I’m not sure about the new money.” He often seems awkward (e.g., holding his arms in odd postures) and nervous, frequently rocking back and forth from one foot to another.

- **Case 3:** Alice is a 33-year-old single woman with a BA in English, and, despite her educational attainment, she works at a low-level clerical position at a local bed-and-breakfast inn. Throughout the day she spends a great deal of time daydreaming, typically envisioning herself as a magazine writer, and she rarely speaks to others unless spoken to first. She reports

having a consistently “uneasy” feeling when around others and on occasion says, “interacting with others is painful for me, it is associated with the same pain you feel when your knuckles hit and run across a carrot grater.” She has experienced herself as “different from others” for as long as she can remember and sometimes feels as though she gets “all mixed up or confused” when doing mundane things, such as shopping or taking a walk. Her parents report that she was somewhat awkward and clumsy as a child and had trouble manipulating small objects such as puzzle pieces or small toys. She often feels that numbers, symbols, and certain images are imbued with a magical power of sorts, and she will alter her behavior depending on the numerals appearing in the date. When walking down the street, she is especially attentive to the expressions on the faces of those who pass her. To her, a smile on the face of a stranger is often taken to mean that the stranger knows something about her (usually something undesirable). She has never dated and reports having no sensual feelings or sexual desires. Her relatives tell her that her speech is hard to follow. She often stays up late into the night reading philosophy and religious texts.

- **Case 4:** Claire, a 27-year-old married woman, works as a code writer for a large software company in a Northern California city. Claire tends to dress in an unusual manner, often wearing clothing that often seems too heavy for the warm climate in which she lives. Throughout childhood she had only one friend, whom she continues to talk to on the phone on a weekly basis. She has no other close friends to speak of beyond her husband. In college, she pursued a double major in German literature and computer science. She met the man she would later marry in a college computer science class. He told her that he was drawn to her because she was “quirky” and “eccentric.” Claire has described an “unusual ability to sense what will happen in the world,” something akin to a “sixth sense,” and she maintains that it goes beyond simply intuition. She also feels that she can influence events with her mind; for example, she thinks that she can make a red light turn green (though she denies that she really “believes” she can do so). She collects small figurines and amulets that she feels help her to “find her way through the world.” Claire’s coworkers do not know her very well, but they find her “pleasant enough, although sort of flaky.” When speaking to most people she appears ill at ease and seems relieved when the conversation ends. On occasion her grimaces or giggles in response to some aspect of a conversation are regarded by other people in the conversation as odd or “weird.” Her face, otherwise, displays little in the way of emotion.

A First Pass at Terminology and Organizing Our Concepts

Clearly, each of these people seems very interesting in certain ways. In some instances they reveal unusual beliefs or behaviors. Some descriptors tend to be relevant to virtually all of them, such as *odd* or *eccentric*. The emotional characteristics of these individuals seem a bit “off” as well; the central theme in the phenomenological picture is that they show minimal *displays* of emotion but have a fair amount of anxiety floating about in their *inner* experience. It is also interesting to observe that some of the behaviors, beliefs, and experiences seem quite distinct from normative psychological functioning (e.g., magical thinking, i.e., belief in forms of causality that are clearly at odds with conventionally accepted forms of causation or perceptual aberrations, disturbances in the perception of the shape or configuration things), whereas others might be construed as more extreme versions of commonly occurring dimensions of experience (e.g., sociality, or degree of engagement with the social world). This is a fascinating issue that raises deep conceptual issues and calls for complex research approaches; we return to this issue more fully later on. For the purposes of our discussion, this person whom we seek to understand will be known to us as a *schizotype*, revealing what we term *schizotypic* psychopathology. None of these individuals has clinical schizophrenia. Some, but not all, of these individuals might be diagnosed with schizotypal personality disorder in the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed, text rev.; DSM-IV-TR; American Psychiatric Association, 2000). Schizotype, schizotypy, and schizotypic psychopathology, as concepts, are developed throughout this book. Not all schizotypes present clinically in a similar fashion; in short, they do not necessarily share the same features. Moreover, the features that do exist *within* this class of psychopathology vary in intensity, as well as in their impact on social or occupational functioning.

The cases described here could be considered to contain dilute components that are suggestive of schizophrenia, the profound psychotic illness characterized by massive disorganization of thought, perception, behavior, and emotion, as well as gross impairments in occupational and social functioning. *However, none of those patients described is psychotic.* Individuals with schizophrenia display hallucinations, delusions, thought disorder, lack of social relations, diminished motivation to function, and, at times, highly bizarre behavior. A primary assumption in this discussion is that schizotypy is an underlying (invisible to the naked eye) liability for schizophrenia. By implication, all people with schizophrenia harbor schizotypy. However, one may harbor schizotypy that reveals itself at the cognitive, interpersonal, and

emotional levels in ways other than full-blown psychosis, and this is precisely the sort of manifestation depicted in the four preceding cases.

The Breadth of the Schizophrenia Phenotype: Implications for Defining the Nonpsychotic Schizotyp

The definition of schizophrenia proper has had a complex history (Lenzenweger, 1999b). When first described by Kraepelin (1919/1971) and Bleuler (1911/1950), the working breadth of the disease phenotype was rather narrow, which meant that a distinctive and severe set of features constituted the illness and the diagnosis was not given frequently. This traditional, narrow definition seemed to stay largely in place among European psychiatrists and psychologists. From the 1920s through the 1960s, however, the definition of schizophrenia in the United States was broadened excessively. Indeed, during my training days at the New York State Psychiatric Institute, some long-term observers of the definition of schizophrenia would often quip that, “in the 1950s, everything that walked west on 168th in Manhattan had a schizophrenic core” (one walked west on 168th Street to go to the Psychiatric Institute from the New York City subway).

The 1950s represented the period in which, arguably, the breadth of the definition of schizophrenia was really at its greatest. During that time, “everyone could develop schizophrenia,” in the minds of some clinicians. It was the heyday of the “psychotic core” concept, which meant that if one pushed hard enough (with stress) and dug deeply enough (clinically), one would find psychosis *in every person*. According to this view, we all possessed the capacity to develop schizophrenia (not merely a psychotic phenomenology that most of us would show with enough sleep deprivation or with a dose of LSD, but rather true blue schizophrenia). This vision of schizophrenia owed much to the overzealous overextension of the psychodynamic model to schizophrenia. Interestingly, this development would likely have been to the chagrin of both Sigmund Freud, the Viennese neurologist who founded psychoanalysis and who thought the origins of schizophrenia would be revealed as genetic, and Carl G. Jung, the Swiss psychiatrist and psychoanalyst who founded analytical psychology and who early in his career studied word associations in schizophrenia, and who thought a “toxin X” would eventually be revealed as the cause of schizophrenia. During the late 1970s and early 1980s, the breadth of the schizophrenia concept in the United States returned to a narrower, more “European-like” width because of (1) concerns about diagnostic reliability, (2) excessive variation in the rates of the schizophrenia diagnosis in the United States as compared with

the United Kingdom, and (3) reemergence of interest in clinical diagnosis as reflected in explicitly defined criteria and the development of *structured interviews* (see Lenzenweger, 1999b; Gottesman, 1991).

Interestingly, as the definition of schizophrenia returned to a breadth that seemed more consistent with that of the original observers (Kraepelin, Bleuler) during the past 35 years or so, there has been an *enhanced* level of interest in those individuals who bear some dilute phenomenological resemblance to schizophrenia and who might harbor a schizophrenia liability—namely, the schizotype. This modern increased interest in the schizotype has been guided by both rigorous theoretical models and careful empirical research. The interest is fueled by the reality that *schizotypes do not have clinical schizophrenia yet harbor the liability for the illness*. The connections between schizotypy on the one hand and schizotypic psychopathology and schizophrenia on the other have been established by years of research in the laboratory, including mine, and the logic and methods of such work are emphasized here.

What Are the Clinical Features of the Schizotype?

One could sift through the four cases described previously and discern many of the cardinal features of the schizotype. The model schizotype is someone who has relatively impoverished interpersonal relations, often having few friends aside from family members. The manner in which the schizotype speaks and uses language can be rather unusual. Words may be used in an unconventional manner that other people do not readily understand or find confusing. The schizotype may have momentary oddities in perception, for example, seeing objects or body parts briefly change their shape or size, and may think relatively neutral events have special significance for him or her (e.g., thinking that a professor directs a lecture specifically to him or her or thinking that others frequently comment on his or her behavior or appearance). He or she may think that he or she can make things happen, as if by a magical ability, such as “causing” a person to call on the phone just by thinking about that person. Emotions and affect expression are rather atypical in the schizotype as well. A schizotype may display little, if any, affect on the face, often appearing rather blunted or expressionless. He or she may claim to feel little to no emotion. What is particularly interesting is that many schizotypes report feeling highly anxious around people, so much so that many carve out lives that minimize contact with other people. Suspiciousness is also a characteristic of many schizotypes. This suspiciousness

can take many forms, such as frequent concerns about being cheated or conspired against, but it falls short of the psychotic level of delusional beliefs (they do not *believe* that they are being cheated or persecuted). Many master clinicians and researchers have suggested other important schizotypic signs and/or symptoms (see Chapter 5).

How Does “Schizotypy” as a Construct Connect to Schizophrenia?

The signs and symptoms of schizophrenia reflect a psychotic illness in an individual, and understanding the nature of those features is essential for understanding schizotypy and schizotypic psychopathology. In this context, the intended meaning of the term *psychotic* is critical. It should be understood to have two primary meanings, implying a mental state reflective of illness that (1) is very severe (*quantitative* meaning) and (2) evidences a *break with reality constraints* in the realm of perception (hallucinations), thought organization (formal thought disorder), and beliefs (delusions) (*qualitative* meaning). As noted previously, the early observers of schizophrenia, such as Emil Kraepelin (1919/1971) and Eugen Bleuler (1911/1950), provided the classic descriptions of the illness. Any serious student of schizophrenia should take the time to read these masterworks. However, both of these observers also took careful notice of what we would now term *schizotypic psychopathology*. How did Kraepelin and Bleuler learn so much about the phenomenology of schizophrenia and schizotypic psychopathology? Although it may seem implausible today, given that most psychiatric patients currently spend less than 3 days in the hospital, the patients in those days lived their lives out, for the most part, in psychiatric hospitals. There, efforts were made to provide humane care as well as refuge for the deeply psychotic person, who typically suffered from schizophrenia or related illnesses, but most never returned home. As a consequence, Kraepelin, Bleuler, and other attending clinicians came to know not only the patients in great detail but also their biological family members. The family members visited their relatives at the hospitals and in doing so they themselves could be observed. It is through these observations, in part, that some of the earliest insights into schizotypic psychopathology were gained. Consider Kraepelin’s and Bleuler’s observations made after years of seeing the biological relatives of people affected with schizophrenia (Box 1.1). It should be clear from these observations that both Kraepelin and Bleuler thought there was some connection between the clinical illness schizophrenia, or really the underlying

BOX 1.1. Early Observations from Masters of Phenomenology

In the families attacked there comes under observation with relative frequency besides dementia praecox a series of other anomalies, especially manic–depressive insanity and *eccentric personalities* [emphasis added]. . . . The latter are probably for the most part to be regarded as “latent schizophrenias” and therefore essentially the same as the principal malady.

—KRAEPELIN (1919/1971)

There is also a latent schizophrenia, and I am convinced that this is the *most frequent form*, although admittedly these people hardly ever come for treatment. . . . In this form we see *in nuce* all the symptoms and all combinations of symptoms which are present in the manifest types of the disease.

—BLEULER (1911/1950)

liability for the illness, and these unusual features observed in the relatives of schizophrenia patients. Kraepelin was struck by the high rate of eccentric personalities among the biological relatives of his schizophrenia patients. Bleuler thought there was a form of the illness characterized by dilute forms of symptomatology suggestive of schizophrenia proper. The implication of these seminal conjectures was really quite profound for the history of schizophrenia research—namely, that schizophrenia could manifest itself in an alternative form; it need not always appear as the well-known clinical phenotype. These *clinical observations* laid the foundation stones for the schizotypic psychopathology and schizophrenia connection. However, at the time, the early 1900s, this notion represented a hypothesis.

What Is the Impact of Schizophrenia on Society and Why Do We Study It?

Schizophrenia is a profound mental disorder, perhaps the most severe form of psychopathology known to humankind.³ It affects roughly 1 in every 100 individuals and appears across all cultures, countries, and continents. The illness is *not* a “myth,” *not* “a sane reaction to an insane world,” *nor* the result

³Interestingly, the case has been made that schizophrenia is a relatively modern illness and has not been among human beings for nearly as long as other illnesses. Gottesman (1991) argues this perspective, and Evans, McGrath, and Milns (2003), in a review of Greek and Roman literature, find no compelling evidence of the illness in classical times.

of “labeling” or a “double bind,” all ideas now considered defunct and recognized as devoid of intellectual merit (although popular during the 1960s and the heyday of the so-called antipsychiatry movement). Rather, schizophrenia is now widely considered to be a brain-based disorder that involves a substantial genetic component, dysfunctional neurobiology, and as yet unspecified environmental inputs (e.g., birth complications, in utero exposure to maternal influenza, noisome work conditions) that come together to generate the illness. Schizophrenia strikes early in life (late teens, early 20s), can be severely disabling across the life span, and results in rather tremendous economic costs such as those related to direct care, aftercare and support (and rehabilitation), and forgone earnings (due to being lost to the workforce). It is important to emphasize that, with the newer medications available for the treatment of the illness, the typical person so affected does not spend his or her entire life in a psychiatric hospital (as in the days of Kraepelin and Bleuler). Today, many people with schizophrenia pursue fuller lives, with the help of medication and aggressive psychosocial treatment. This is true of contemporary schizophrenia sufferers when compared with those so affected but 20 years ago. However, even today, many schizophrenia patients do not return to the level of psychological and social functioning they had before the illness struck, and they are faced with the challenges of a chronic illness, which reappears from time to time, in their daily living.

Clearly, schizophrenia is a major form of psychopathology that is massively disabling; thus we want to try to understand its causes and development, with an eye toward (eventually) prevention. We are motivated in this goal by many factors, not the least of which concerns the cost of this illness to the individual and society. The monetary cost of schizophrenia has been the focus of some careful economic modeling, and the numbers generated by these exercises are staggering. According to estimations (Wu et al., 2005) done using 2002 dollars and prevalence data from the well-known National Comorbidity Survey—Replication, a large-scale, national epidemiological study of psychiatric disorders, the overall cost was estimated at \$62.7 billion. This figure can be further broken down into direct health care cost (\$22.7 billion), direct nonhealth excess costs (e.g., living costs; \$7.6 billion), and total indirect excess costs (e.g., unemployment; \$32.4 billion; Wu et al., 2005). These figures reflect the total economic impact of schizophrenia on society (i.e., the cost posed by the illness in people with the illness vs. the cost had they never been affected by schizophrenia). In England, based on 2004–2005 estimations, the total societal cost of schizophrenia was £6.7 billion (or \$22.1 billion; Mangalore & Knapp, 2007). Costs in the United

States and in England are somewhat difficult to compare because of massive differences in health care costs resulting from large differences in care and support systems. Finally, Knapp, Mangalore, and Simon (2004) estimated the worldwide societal cost of schizophrenia, and, although there is considerable variation across countries, the economic impact of schizophrenia is really quite astounding. Knapp et al. (2004) estimate that between 1.5 and 3% of total national health care expenditures are due to schizophrenia and that “sizeable portions of total inpatient budgets are accounted for by people with schizophrenia” (p. 290). The monetary estimates, of course, do not even gauge the “pain cost” of schizophrenia—by this we mean the psychological pain and anxiety suffered by patients themselves, and by their partners, children, parents, and so on. For example, how can one estimate the cost of the psychological fear and anxiety felt by parents when they receive a 4:00 A.M. phone call from the police indicating that their son was picked up while walking down the interstate highway in the nude claiming that the Central Intelligence Agency was inserting microchips under his fingernails? Or the despair felt by a young woman who had been a talented mathematics major in college when, on emergence of the illness, she could no longer even subtract 3s in a sequence beginning at 100 due to cognitive disorganization from her schizophrenia?

One, of course, could argue that the study of schizophrenia is interesting in its own right given the profound deviations in normal human psychological functioning seen in the illness. It is a fascinating and perplexing problem beckoning to be understood. Study of such deviation provides useful information on the nature of normative functioning whereby illuminating the pathological informs the normal (a fundamental tenet of developmental psychopathology; Cicchetti & Cohen, 2006). The challenge to understand the causes of schizophrenia receives most of its urgency and sense of purpose, however, from the pain and cost components associated with it.

What Leverage Does Study of Schizotypy and the Schizotype Offer for Uncovering the Causes of Schizophrenia?

Schizophrenia has long frustrated generations of research workers (e.g., it has even been thought of as “graveyard” for psychopathology geneticists, notwithstanding some gains that have been made in recent years), and its specific etiology (Meehl, 1972b, 1977) remains elusive. How best to gain

leverage on this disorder? I argue that this is done best through the study of the schizotype and full incorporation of a schizotypy model in schizophrenia research.

The primary thesis of this book, therefore, concerns a conceptual and empirical approach that is intended to provide leverage in our understanding of the fundamental nature of schizophrenia. Moreover, it is argued that a coherent model that stresses several components at different levels of analysis will optimally provide that leverage. The primary working assumption of this argument (as well as the program of research on which it is built) is that *schizophrenia is a manifestation of an underlying construct (more about constructs to follow) known as schizotypy and schizotypic psychopathology is to be thought of as an alternative manifestation of schizotypy and, by implication, a variant of schizophrenia liability.* The primary utility of schizotypic psychopathology as a unit of analysis (in relation to schizophrenia) is that it potentially represents a clearer window on the underlying liability for schizophrenia per se. How can this be so? The reason for this assertion can be best found in an analogy. Imagine that a house mysteriously burns to the ground, and the fire investigators need to determine what happened. How did this fire start? It is all rather difficult to probe through the ashes and burnt debris to find a valid cause for the fire. Imagine further that the fire actually began in the breaker box that contains the crossroads of the major electrical circuitry for the house. The fire investigator might be able to find the breaker box and the remnants of charred breakers, melted and twisted plastic components, burnt wiring, and so on. However, in this mess of twisted and charred debris, it would be particularly hard to figure out just which specific wire or poor connection was the cause of the sparking that ignited the fire. One could guess, or limit potential explanations, based on what is known about typical breaker boxes; however, the precise unfolding of events cannot be known with certainty *after the fact*. Imagine further that we had been electrical inspectors and had been able to examine and record the condition and status of the various wiring and connections in the breaker box prior to the fire. If we had been in this position as inspectors, then we could probably say with greater confidence that the two wires without insulation and nearly touching one another could represent a genuine fire hazard. If we had known what was wrong *ahead of time* (prior to the onset of the fire), then, perhaps, we (1) could understand more fully what gave rise to the fire and (2) could have engaged in preventive intervention (i.e., we could have fixed the wires that lacked insulation).

By extension to schizophrenia, imagine that the clinical illness of schizophrenia represents the fire that has already begun. As time goes by,

particularly during, say, the first 5 years of the illness, the brain begins to manifest illness-related changes. Thus, in a very real sense, the study of the schizophrenia-affected brain represents an endeavor that is, *by definition*, clouded by the illness per se and all its various impacts. To discover the precise site(s) (or nature) of the etiological dysfunction responsible for schizophrenia after the illness has expressed itself is not unlike trying to discover the nature of the wiring problems in the breaker box that caused our hypothetical house fire. This is so because the transition to a psychotic state, along with various associated processes (e.g., medication, institutionalization, deterioration in functioning, stigma associated with a major mental disorder, impact of comorbid conditions, e.g., substance abuse) inevitably color the neurobiological, neurological, cognitive, personality, and social functioning of an individual. Depending on one's point of view, the clinical illness can merely be seen as "clouding" the picture, and therefore it hampers the search for important clues as to the nature of schizophrenia (though this problem can be overcome with sufficiently clever research designs), whereas another point of view would hold that the causal picture (etiology and fundamental pathologies) becomes opaque with the onset of clinical illness.

How, then, should the psychopathologist proceed? The answer to this question, more or less, depends on how one defines the beginning of the illness, as well as how one defines the boundaries of the illness (i.e., Does schizophrenia always look like schizophrenia?). *The present discussion is based, therefore, on a second critical assumption, namely that schizophrenia begins long before the emergence of the well-known clinical symptoms of the disorder.* Alternative manifestations of liability and early developing pathology are not necessarily easily accepted or easily defined assumptions. For example, simply defining the beginning of the illness can be challenging. One could conceivably take the position that the illness began long before the manifestation of the clinical signs and symptoms of schizophrenia. For example, one could restrict oneself to emerging preschizophrenia, or *prodromal*, symptoms and behaviors that appear during the buildup to clinical schizophrenia. One would maintain that the "fire" began then. Alternatively, one could hypothesize that the illness begins earlier in childhood and, therefore, examine dysfunctions that are known to be associated with later schizophrenia, such as motoric abnormalities, in the late teens or early 20s. It may actually be the case that schizophrenia or, perhaps more accurately, the pathological processes that reveal themselves in clinical schizophrenia, begin *at biological conception*. The blueprint for the illness might be laid down nearly immediately and slowly begin to reveal itself through

subtle deviations in neural development, disruption in neurobiological systems, abnormalities in behavioral development, and, later, impaired psychological functioning.

The assumption that schizophrenia liability can reveal itself in *alternate forms* represents a theoretical bridge that both expands what one typically thinks of as the phenotypic boundaries for schizophrenia and, importantly, provides powerful conceptual and statistical tools for illuminating the nature of schizophrenia (Meehl, 1962; Lenzenweger, 1998; Kendler, Neale, & Walsh, 1995). It should be evident that one of these *alternate expressions* of schizophrenia liability is schizotypic psychopathology. It should be understood, therefore, that schizotypic psychopathology is *not* merely an *analog* of schizophrenia; rather, it represents a valid, albeit nonpsychotic, expression of the same liability that underpins schizophrenia. An *analog* in psychopathology research approaches means utilizing an artificially created—typically in the laboratory—deviation in psychological state or functioning that shares presumably some aspects with a genuine form of psychopathology. For example, analog depression represents a transient emotional state induced in a laboratory for the purposes of trying to understand some aspect of clinical (major) depression.⁴ *In short, schizotypic psychopathology is not an analog for schizophrenia; rather, it is a valid alternate expression of schizophrenia liability. It is the real thing.*

A Thumbnail Sketch of the Benefits of the Schizotypy Model Approach

In summary, what are the benefits of the schizotypy model approach in our search for the causes of schizophrenia? First and foremost, the study of schizotypic psychopathology provides a “cleaner” window on underlying schizophrenia liability. By cleaner I mean an opportunity to study in the laboratory genetically influenced, neurobiologically based processes (neurocognitive, affective, personality) that are uncontaminated by “third variable” confounds, such as medication, deterioration, and institutional-

⁴Creating analog depression might involve something like making normal undergraduate students believe that they have done poorly on some sort of psychological task or test and then studying a specific aspect of their cognitive functioning, such as attributional style. One might hypothesize that one could induce a pattern of thinking characterized by seeing the causes for failure as internal to the self, stable over time, and global in its impact, thus bringing about “depression” in the students. The assumption of an analog research approach is that it provides leverage on genuine depression. But to what extent does such analog depression really mimic clinical depression?

ization. Schizotypic psychopathology represents the breaker box before the fire. Second, the schizotypy model approach to schizophrenia also provides a rich opportunity to discover *endophenotypes* for schizophrenia liability. Endophenotypes (Gottesman & Gould, 2003; Shields & Gottesman, 1973) represent genetically influenced manifestations of the underlying liability for an illness that are invisible to the unassisted or “naked” eye (Chapter 7). Third, incorporation of valid schizotypy indicators (e.g., schizotypic psychopathology) into genomic investigations directed at etiology and development of schizophrenia will enhance the power of such studies (Lenzenweger, 1994; Brzustowicz, 2007; Brzustowicz & Bassett, 2008). Fourth, via longitudinal investigations, the study of schizotypic psychopathology can elucidate *epigenetic* factors that might relate to the differences in outcome of schizotypes (i.e., stable schizotypal personality disorder vs. conversion to schizophrenia).

A Word about “Experimental Psychopathology”

Finally, it is important to define what is meant by *experimental psychopathology*. Many students in psychological science are familiar with the more traditional subdivisions of the field, such as experimental, cognitive, developmental, clinical, social, industrial/organizational, and so on. Psychological science is beginning to mature, and, as a result, the complexity of the problems that the contemporary discipline focuses on has required the field to realize that many traditionally compartmentalized approaches to human behavior are insufficient for tackling the problems of greatest interest. For example, personality psychology has begun to move away from strictly questionnaire research to incorporate neurobehavioral systems models, affective and emotional science perspectives, and genetic vantage points. The scientific study of psychopathology has also matured and grown beyond the simple psychological testing and descriptive approaches of clinical psychology. For example, one cannot really ponder a complex topic such as schizophrenia without bringing in elements of experimental psychology, psychometrics, behavioral genetics, cognitive science, and all manner of neuroscience. Experimental psychopathology emerged over the past 30 years as a powerful approach to the study of psychopathology.

The classic definition of experimental psychopathology centers on the use of the experimental methods and the rigors of the experimental psychology laboratory in the study of psychopathology. This definition and the resulting research subdiscipline owes much to Brendan A. Maher’s semi-

BOX 1.2. On the Nature of Experimental Psychopathology: Lenzenweger's View

Experimental psychopathology is the psychological science discipline that uses the methods of the experimental psychology laboratory in conjunction with quantitative analytic approaches to gain leverage on etiology and pathogenesis of psychopathology, within a brain-based (genomic, endophenotype, neurobiological) diathesis–stressor matrix.

nal volume titled *Principles of Psychopathology: An Experimental Approach* (Maher, 1966). Maher's vision for an effective approach to the study of psychopathology has yielded considerable fruit and continues to grow and develop (see Lenzenweger & Hooley, 2003). Incorporating the neuroscience perspective and embracing the technology of neuroimaging, as well as modern genomics, modern experimental psychopathology continues to provide an essential vantage point for seeking to better understand the nature of psychopathology. I would, therefore, offer my own definition of contemporary experimental psychopathology⁵ (Box 1.2). We unpack this definition, explicitly and implicitly, as we progress through this book. However, at this juncture, we must take a necessary conceptual and methodological detour. This detour is intended to convey some basic notions regarding an effective way to think about doing experimental psychopathology research. The issues covered in Chapters 2, 3, and 4 will be the “balls that one will need to keep in the air” when attempting to unravel complex problems such as schizophrenia and schizotypic psychopathology.

⁵Although most experimental psychopathologists have training as psychologists in the methods of the experimental psychology laboratory, it is important to note that the “experimental psychopathology” perspective is really just that, a perspective. It represents an approach or vantage point. It is not a professional guild; there are no membership cards. Rather, it represents an approach that is founded on shared values embodied in the merits of laboratory-based science. Whereas many experimental psychopathologists began their careers with formal training in clinical psychology, there are many experimental psychopathologists who do not hold that credential. Many psychiatrists (MDs) who have come to laboratory research on mental illness are considered experimental psychopathologists.