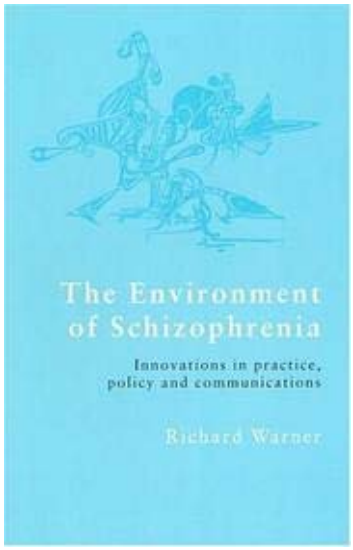
Text and Figures from the Introduction to

The Environment of Schizophrenia



by

Richard Warner

London: Brunner-Routledge, 2000.

What is schizophrenia?

In our own popular culture, there may be more widespread ignorance about schizophrenia than any other common illness. Ask a classroom of American college students — in engineering or English literature — what they know about AIDS or cancer and they will probably have a lot to say. But ask about schizophrenia and the silence will be embarrassing. Although schizophrenia is more common than AIDS/HIV, most people know far less about it. "Isn't it like multiple personality disorder?" people ask. "Is it caused by child abuse?" "Are they mentally retarded?" The answer to all these questions is "No."

What is it about this condition that stifles discussion and learning? AIDS, cancer and schizophrenia are all perceived as contaminating and incurable, but somehow people with schizophrenia are seen as more mysterious, alien and violent. Centuries of fear have promulgated many myths about schizophrenia. What are the facts?

Schizophrenia is a psychosis. That is to say, it is a severe mental disorder in which the person's emotions, thinking, judgment, and grasp of reality are so disturbed that his or her functioning is seriously impaired.

The symptoms of schizophrenia are often divided into "positive" and "negative." Positive symptoms are abnormal experiences and perceptions like delusions, hallucinations, illogical and disorganized thinking and inappropriate behavior. Negative symptoms are the absence of normal thoughts, emotions and behavior; such as blunted emotions, loss of drive, poverty of thought, and social withdrawal.

Diagnostic difficulties

Problems abound in defining schizophrenia. The two most common functional psychoses are schizophrenia and bipolar disorder (also known as manic-depressive illness). The distinction between the two is not easy to make and psychiatrists in different parts of the world at different times have drawn the boundaries in different ways. Bipolar disorder is an episodic disorder in which psychotic symptoms are associated with severe alterations in mood — at times elated, agitated episodes of mania, at other times depression, with physical and mental slowing, despair, guilt and low self-esteem.

On the other hand, the course of schizophrenia, though fluctuating, tends to be more continuous, and the person's display of emotion is likely to be incongruous or lacking in spontaneity. Markedly illogical thinking is common in schizophrenia. Auditory hallucinations may occur in either manic-depressive illness or schizophrenia, but in schizophrenia they are more likely to be commenting on the person's actions or to be conversing one with another. Delusions, also, can occur in both conditions; in schizophrenia they may give the individual the sense that he or she is being controlled by outside forces or that his or her thoughts are being broadcast or interfered with.

Despite common features, different forms of schizophrenia are quite dissimilar. One person, for example, may be paranoid but show good judgment and high functioning in many

areas of life. Another may be bizarre in manner and appearance, preoccupied with delusions of bodily disorder, passive and withdrawn. So marked are the differences, in fact, that many experts believe that, when the causes of schizophrenia are worked out, the illness will prove to be a set of different conditions which lead, *via* a final common pathway of biochemical interactions, to similar consequences.

It is not at all clear what is schizophrenia and what is not. Scandinavian psychiatrists have tended to use a narrow definition of the illness with an emphasis on poor outcome. Russian psychiatrists have adhered to a broad definition with an emphasis on social adjustment. In the United States the diagnostic approach to schizophrenia used to be very broad. With the publication, in 1980, of the third edition of the American Psychiatric Association *Diagnostic and Statistical Manual*, however, American psychiatry switched from one of the broadest concepts of schizophrenia in the world to one of the narrowest.

Why is the diagnosis so susceptible to fashion? The underlying problem is that schizophrenia and manic-depressive illness share many common symptoms. During an acute episode it is often not possible to tell them apart without knowing the prior history of the illness. The records of people with manic-depressive illness, however, should reveal prior episodes of depression and mania with interludes of normal functioning.

Schizophrenia is universal

We should not let confusion about differentiating schizophrenia from other psychoses detract from the fact that schizophrenia is a universal condition and an ancient one. Typical cases may be distinguished in the medical writings of ancient Greece and Rome, and the condition occurs today in every human society. While the content of delusions and hallucinations varies from culture to culture, the form of the illness is similar everywhere. Two World Health Organization studies, applying a standardized diagnostic approach, have identified characteristic cases of schizophrenia in developed and developing world countries from many parts of the globe.

More surprisingly, one of these studies demonstrated that the rate of occurrence of new cases (the incidence) of the condition is similar in every country studied from India to Ireland. However, since both death and recovery rates for people with psychosis are higher in the Third World, the point prevalence of schizophrenia (the number of cases to be found at any time) is lower in the Third World — around 3 per 1,000 of the population compared to 6 per 1,000 in the developed world. The risk of developing the illness at some time in one's life (the lifetime prevalence) is a little higher — around one percent of the population in the developed world.

People recover from schizophrenia

The popular and professional view that schizophrenia has a progressive, downhill course with universally poor outcome is a myth. Over the course of months or years, about 20 to 25 percent of people with schizophrenia recover completely from the illness — all their psychotic

symptoms disappear and they return to their previous level of functioning. Another 20 percent continue to have some symptoms, but they are able to lead satisfying and productive lives.

In the developing countries, recovery rates are even better. The two World Health Organization studies mentioned above have shown that good outcome occurs in about twice as many patients diagnosed with schizophrenia in the developing world as in the developed world. The reason for the better outcome in the Third World is not completely understood, but it may be that many people with mental illness in developing world villages are better accepted, less stigmatized, and more likely to find work in a subsistence agricultural economy.

The course of schizophrenia

Wide variation occurs in the course of schizophrenia. In some cases the onset of illness is gradual, extending over the course of months or years; in others it can begin suddenly, within hours or days. Some people have episodes of illness lasting weeks or months with full remission of symptoms between each episode; others have a fluctuating course in which symptoms are continuous; others again have very little variation in their symptoms of illness over the course of years. The final outcome from the illness in late life can be complete recovery, a mild level of disturbance or continued severe illness.

Figure I.2 is an illustration of the onset, course and outcome of the illness in 228 people with schizophrenia followed into old age by the Swiss psychiatrist, Luc Ciompi. He found that the onset of the illness was either acute (with less than six months from first symptoms to full-blown psychosis) or, conversely, insidious, in roughly equal numbers of cases. Similarly, the course of the condition was episodic or continuous in approximately equal numbers of patients; and the outcome was moderate to severe disability in half the cases and mild disability or full recovery in the other half. Full recovery was observed in more than a quarter of the patients. It is clear that the course of schizophrenia varies a good deal between individuals and that the outcome is often favorable.

It is also true to say that schizophrenia usually becomes less severe as the person with the illness grows older. In addition, the later the illness begins in life, the milder it proves to be. Women usually develop their first symptoms of schizophrenia later than men and the course of their illness tends to be less severe. Onset of schizophrenia before the age of 14 is rare, but when it does begin this early it is associated with a severe course of illness. Onset after the age of 40 is also rare, and is associated with a milder course.

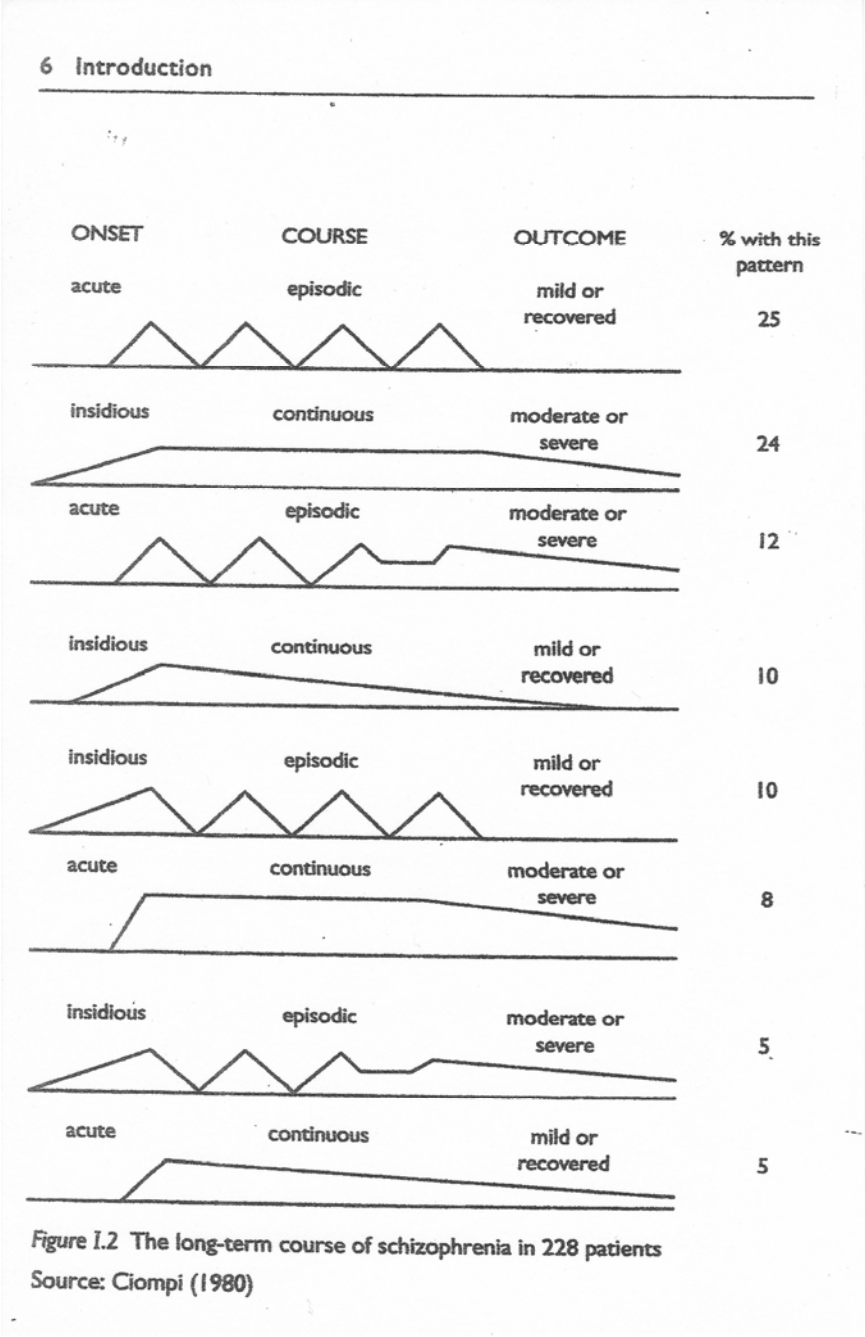


Figure I.2 The long-term course of schizophrenia in 228 patients
Source: Ciompi (1980)

What causes schizophrenia?

There is no single organic defect or infectious agent which causes schizophrenia, but a variety of factors increase the risk of getting the illness — among them, genetics and obstetric complications.

Genetics

Relatives of people with schizophrenia have a greater risk of developing the illness, the risk being progressively higher among those who are more genetically similar to the person with schizophrenia (see Figure I.3). For a nephew or aunt the lifetime risk is about two percent (twice the risk for someone in the general population); for a sibling, parent, or child the risk is about ten percent, and for an identical twin (genetically identical to the person with schizophrenia) the risk is close to 50 percent.

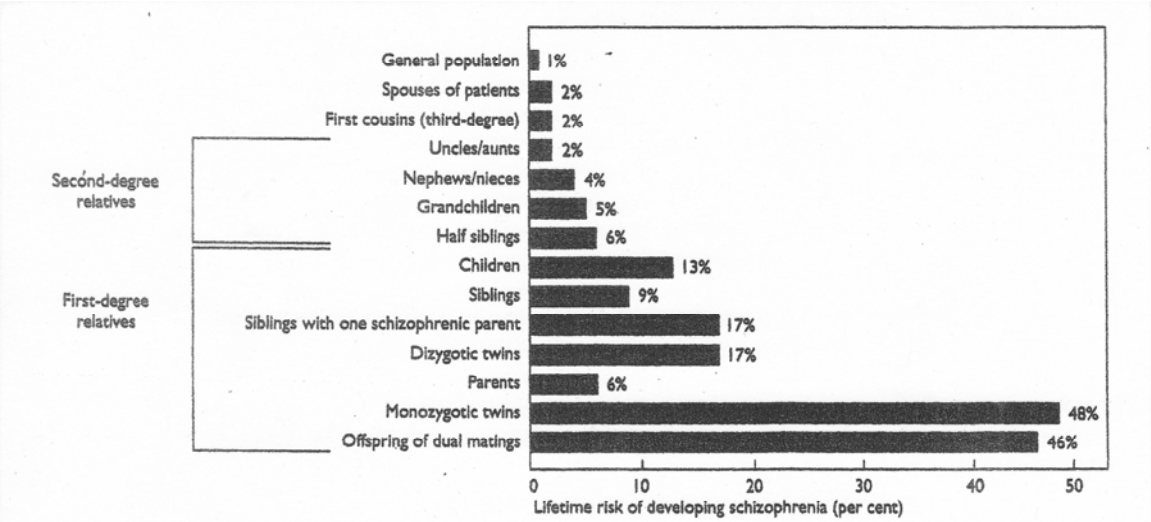


Figure I.3 The average risk of developing schizophrenia for relatives of a person with the illness; compiled from family and twin studies conducted in Europe between 1920 and 1987

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Studies of people adopted in infancy reveal that the increased risk of schizophrenia among the relatives of people with the illness is due to inheritance rather than environment. The children of people with schizophrenia have the same increased prevalence of the illness whether they are raised by their biological parent with schizophrenia or by adoptive parents.

There is evidence implicating several genes in causing schizophrenia, and it is likely that more than one is responsible, either through an interactive effect or by producing different variants of the disorder.

Obstetric complications

Since identical twins only have a 50 percent risk of developing the illness, we know that genetics alone do not explain why someone gets the illness. Other powerful factors have to play a part; one of these is problems of pregnancy and delivery. The risk for people born with obstetric complications, such as prolonged labor, is double the risk for those born with none. A history of obstetric complications has been found in up to 40 percent of patients with schizophrenia, making it a major risk factor.

Viruses

The risk of intrauterine brain damage is increased if a pregnant woman contracts a viral illness. We know that more people with schizophrenia are born in the late winter or spring than at other times of year, and that this birth bulge sometimes increases after epidemics of viral illnesses like influenza, measles and chickenpox. Maternal viral infections, however, probably account for only a small part of the increased risk for schizophrenia.

Poor parenting does not cause schizophrenia

Contrary to the beliefs of professionals prior to the 1970s and to the impression still promoted by the popular media, there is no evidence, even after decades of research, that family or parenting problems cause schizophrenia.

As early as 1948, psychoanalysts proposed that mothers fostered schizophrenia in their offspring through cold and distant parenting. Others blamed parental schisms, and confusing patterns of communication within the family. The double-bind theory, put forward by anthropologist Gregory Bateson, argued that schizophrenia is promoted by contradictory parental messages from which the child is unable to escape. While enjoying broad public recognition, such theories have seldom been adequately tested, and none of the research satisfactorily resolves the question of whether differences found in the families of people with schizophrenia are the *cause* or the *effect* of psychological abnormalities in the disturbed family member.

Millions of family members of people with schizophrenia have suffered needless shame, guilt and stigma because of this widespread misconception.

Drug abuse does not cause schizophrenia

Hallucinogenic drugs like LSD can induce short-lasting episodes of psychosis and the heavy use of marijuana and stimulant drugs like cocaine and amphetamines may precipitate brief, toxic psychoses with features similar to schizophrenia. It is also possible, though by no means certain, that drug abuse can trigger the onset of schizophrenia.

Relatives of a person with schizophrenia sometimes blame hallucinogenic drugs for causing the illness, but they are mistaken. We know this because, in the 1950s and 1960s, LSD was used as an experimental drug in psychiatry in Britain and America. The proportion of these volunteers and patients who developed a long-lasting psychosis like schizophrenia was scarcely greater than in the general population. It is true that a Swedish study found that army conscripts who used marijuana heavily were six times more likely to develop schizophrenia later in life, but this was probably because those people who were destined to develop schizophrenia were more likely to use marijuana as a way to cope with the pre-morbid symptoms of the illness.

The brain in schizophrenia

Physical changes in the brain have been identified in some people with schizophrenia. The analysis of brain tissue after death has revealed a number of structural abnormalities, and new brain-imaging techniques have revealed changes in both the structure and function of the brain during life. Techniques such as magnetic resonance imaging (MRI) reveal changes in the size of different parts of the brain, especially in the temporal lobes. The fluid-filled spaces (the ventricles) in the interior of the temporal lobes are often enlarged and the temporal lobe tissue diminished. The greater the observed changes the greater the severity of the person's thought disorder and his or her auditory hallucinations.

Some imaging techniques, such as positron emission tomography (PET), measure the actual functioning of the brain and provide a similar picture of abnormality. PET scanning reveals hyperactivity in the temporal lobes, particularly in the hippocampus, a part of the temporal lobe concerned with orientation and very short-term memory. Another type of functional imaging, electrophysiological brain recording using EEG tracings, shows that most people with schizophrenia seem to be excessively responsive to repeated environmental stimuli and more limited in their ability to blot out irrelevant information. In line with this finding, those parts of the brain that are supposed to screen out irrelevant stimuli, such as the frontal lobe, show decreased activity on PET scan.

Tying in with this sensory screening difficulty, post-mortem brain tissue examination has revealed problems in a certain type of brain cell — the inhibitory interneuron. These neurons damp down the action of the principal nerve cells, preventing them from responding to too many inputs. Thus, they prevent the brain from being overwhelmed by too much sensory information from the environment. The chemical messengers or neurotransmitters (primarily gamma-amino butyric acid or GABA) released by these interneurons are diminished in the brains of people with schizophrenia, suggesting that there is less inhibition of brain overload.

Abnormality in the functioning of these interneurons appears to produce changes in the brain cells which release the neurotransmitter dopamine. The role of dopamine has long been of interest to schizophrenia researchers, because drugs such as amphetamines that increase dopamine's effects can cause psychoses that resemble schizophrenia, and drugs that block or decrease dopamine's effect are useful for the treatment of psychoses. Dopamine increases the sensitivity of brain cells to stimuli. Ordinarily, this heightened awareness is useful in increasing a person's awareness during times of stress or danger, but, for a person with schizophrenia, the addition the effect of dopamine to an already hyperactive brain state may tip the person into psychosis.

These findings suggest that in schizophrenia there is a deficit in the regulation of brain activity by interneurons, so that the brain over-responds to the many signals in the environment and lacks the ability to screen out unwanted stimuli. This problem is made worse by a decrease in the size of the temporal lobes, which ordinarily process sensory inputs, making it more difficult for the person to respond appropriately to new stimuli.

Why does schizophrenia begin after puberty?

Schizophrenia researchers have long been puzzled about why the illness normally begins in adolescence when important risk factors, such as genetic loading and neonatal brain damage, are present from birth or sooner. Many believe that the answer to this puzzle could tell us a lot about the cause of the illness. We now have some good clues to this mystery.

We know, for example, that normal brain development leads to the loss of 30 to 40 percent of the connections (synapses) between brain cells during the developmental period from early life to adolescence. Brain cells themselves do not diminish in number during this period, only their connectivity. It appears that we may need a high degree of connectivity between brain cells in infancy to enhance our ability to learn language rapidly (toddlers learn as many as twelve new words a day). The loss of neurons during later childhood and adolescence, however, improves our "working memory" and our efficiency to process complex linguistic information. When we are listening to someone talking, for example, and we miss part of a phrase or sentence because someone nearby coughs or sneezes, our working memory allows us to fill in the blank, using a memory store of similar familiar phrases we have heard before.

We now know that, for people with schizophrenia, this normally useful process of synaptic pruning has been carried too far, leaving fewer synapses in the frontal lobes and medial temporal cortex. In consequence, there are deficits in the interaction between these two areas of the brain in schizophrenia which reduce the adequacy of working memory. One intriguing computer modeling exercise suggests that decreasing synaptic connections and eroding working memory in this way not only leads to abnormalities in the ability to recognize meaning when stimuli are ambiguous but also to the development of auditory hallucinations.

It is possible, therefore, that this natural and adaptive process of synaptic elimination in childhood, if carried too far, could lead to the development of schizophrenia. If true, this would help explain why schizophrenia persists among humans despite its obvious functional disadvantages and its association with reduced fertility. The genes for synaptic pruning may help us refine our capacity to comprehend speech and other complex stimuli, but, when complicated by environmental assaults resulting in brain injury, the result could be symptoms of psychosis. As yet, this formulation is speculative, but it allows us to see more clearly how the environment may interact with our innate qualities to increase our predisposition to schizophrenia.

What works?

There is more agreement now about what is important in the treatment of schizophrenia than ever before. In a recent global project designed to combat the stigma of schizophrenia, prominent psychiatrists from around the world agreed on the following principles:

- People with schizophrenia can be treated effectively in a variety of settings. These days the use of hospitals is mainly reserved for those in an acute relapse. Outside of the hospital, a range of alternative treatment settings have been devised which provide supervision and support and are less alienating and coercive than the hospital.
- Family involvement can improve the effectiveness of treatment. A solid body of research has

demonstrated that relapse in schizophrenia is much less frequent when families are provided with support and education about schizophrenia.

- Medications are an important part of treatment but they are only part of the answer. They can reduce or eliminate positive symptoms but they have a negligible effect on negative symptoms. Fortunately, modern, novel antipsychotic medications, introduced in the past few years, can provide benefits while causing less severe side effects than the standard antipsychotic drugs which were introduced in the mid-1950s.
- Treatment should include social rehabilitation. People with schizophrenia usually need help to improve their functioning in the community. This can include training in basic living skills; assistance with a host of day-to-day tasks; and job training, job placement, and work support.
- Work helps people recover from schizophrenia. Productive activity is basic to a person's sense of identity and worth. The availability of work in a subsistence economy may be one the main reasons that outcome from schizophrenia is so much better in Third World villages. Given training and support, most people with schizophrenia can work.
- People with schizophrenia can get worse if treated punitively or confined unnecessarily. Extended hospital stays are rarely necessary if good community treatment is available. Jail or prison are not appropriate places of care. Yet, around the world, large numbers of people with schizophrenia are housed in prison cells, usually charged with minor crimes, largely because of the lack of adequate community treatment.
- People with schizophrenia and their family members should help plan and even deliver treatment. Consumers of mental health services can be successfully employed in treatment programs, and when they help train treatment staff, professional attitudes and patient outcome both improve.
- People's responses towards someone with schizophrenia influence the person's course of illness and quality of life. Negative attitudes can push people with schizophrenia and their families into hiding the illness and drive them away from help. If people with schizophrenia are shunned and feared they cannot be genuine members of their own community. They become isolated and victims of discrimination in employment, accommodation and education.

About the Author

The international mental health care community recognizes Dr. Richard Warner as a leader in schizophrenia treatment and recovery research and development. Dr. Warner is Professor of Psychiatry and Adjunct Professor of Anthropology at the University of Colorado.

Dr. Warner served as the Medical Director of the Mental Health Center of Boulder County, the public mental health facility for a population of nearly 300,000 people in the Front Range of Colorado, for 29 years until September, 2005. While at the Mental Health Center of Boulder County, Dr. Warner helped to develop a comprehensive community support system for people with serious mental illness that has earned an international reputation. The system includes a residential treatment program for people with serious mental illness (originally called Cedar House later renamed Warner House), at which Dr. Warner was the Chief Psychiatrist.

The University of Birmingham recognized Dr. Warner for his global contributions mental health care and research by inviting him to participate in a series of lectures hosted by the Centre of Excellence in Interdisciplinary Mental Health (CEIMH). A recording and transcript of the conversation with Dr. Warner is available for viewing and download. Visit http://www.ceimh.bham.ac.uk/tv/dickwarnerpreview.shtml.

At the Mental Health Center of Boulder County, Dr. Warner also helped design and develop:

- A variety of community-based alternatives to the hospital for the treatment of people with acute psychiatric problems.
- An innovative jail-diversion program for people with mental illness.
- Programs to employ people with mental illness, including a specialist pharmacy for people with mental illness that employed people with mental illness as pharmacy technicians.
- Regular public education on mental illness.

Dr. Warner is the author of a number of books including:

- *Recovery from Schizophrenia* (third edition: Brunner-Routledge, 2004),
- *The Environment of Schizophrenia* (Brunner-Routledge, 2000), and
- *Alternatives to the Hospital for Acute Psychiatric Treatment* (American Psychiatric Press, 1995).

With Julian Leff, he co-authored *Social Inclusion of People with Mental Illness*, which was published by Cambridge University Press in 2006. Dr. Warner's books have been translated into seven languages. Much of his research has focused on social factors that impact the course of mental illness.

Beginning in 1995, Dr. Warner helped direct *Open the Doors*, a global campaign of the World Psychiatric Association to combat the stigma of schizophrenia. He was closely involved in two of the anti-stigma programs, launched by Open the Doors – in Calgary, Alberta, and in Boulder, Colorado.

Dr. Warner lectures and consults in several countries around the world on a regular basis. He has visiting faculty appointments at two universities in Great Britain and is on the editorial boards of professional journals in four countries.

Colorado Recovery is located in Boulder, Colorado. You can find out more about schizophrenia treatment options at Colorado Recovery by visiting www.coloradorecovery.com or calling 303-440-5140.