Bias and Schizophrenia

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The concept of schizophrenia is an icon of contemporary psychiatry: most clinicians and laypeople have been led to believe that it is an indisputably real mental illness and that solid scientific research has led to a body of well-established knowledge about schizophrenia, what it is, what causes it, and how best to treat it. Most clinicians have been taught some version of the following "received view":

1) Schizophrenia is a brain disease. The specifics of its pathology and etiology are not fully known, although:

   -Schizophrenia has stable prevalence rates across cultures and over time (e.g., approximately 1% prevalence)
   -It is well-established that schizophrenia has a genetic component in its etiology
   -Schizophrenia is associated with a number of environmental stressors that might play a role in its etiology (e.g., pre-natal exposure to famine, viral infection, and stress; birth trauma)
   -There are a number of compelling findings and promising leads concerning the pathophysiology of schizophrenia (e.g., dopaminergic dysregulation, ventriculomegaly, hypofrontality, hypertemporality, a variety of neurocognitive deficits)

2) Schizophrenia has a characteristic, identifiable clinical picture and can be reliably diagnosed using criteria found in the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (American Psychiatric Association 1994).

3) Schizophrenia has harmful psychological and social consequences, and it constitutes a serious public health problem

4) Schizophrenia is **primarily** treatable with psychotropic drugs, and, there have been significant improvements in such treatment in the past decade (e.g., the atypical anti-psychotics.) Schizophrenia is managed (palliation, support, relapse prevention, rehabilitation) with a combination of the primary pharmacological treatment and appropriate ancillary techniques (e.g., individual psychotherapy, psychosocial interventions)

5) Schizophrenia is unjustifiably stigmatized, and the stigma can be reduced through teaching that schizophrenia is a disease

There is little doubt that millions of people suffer from severe mental illness and experience a wide range of problems including: cognitive impairments, hallucinations and delusions, negative emotions such as fear, anxiety, and depression, behavioral and social skills deficits, dysfunctional social identities and roles, demoralization, poverty, inadequate housing, no friends, nothing to do. And it is well-established that there are effective ways of helping people with the various problems falling under the label "severe mental illness". The subject of this paper is whether the concept of schizophrenia and the associated received view have anything useful to add to clinical practice concerned with severe mental illness. They do not.[i] The accepted beliefs about schizophrenia lack scientific credibility because they are not supported by high-quality research; and they lead to a simplistic view of severe mental illness and to harmful distortions of clinical practice.

Part 1: The Received View of Schizophrenia Is a Stereotype.

The full case for the claim that the received view of schizophrenia lacks scientific credibility involves detailed examination of the research record (Boyle, 1990; Bentall, 1990; Heinrichs, 2001) along with a close critical scrutiny of the research program associated with the received view including its standards of evidence and potential for progress (Poland, in press), the intactness and integrity of the scientific community engaged in this research (Poland and Spaulding, forthcoming), and the practical utility of the received view even if its claims are not supported. But even short of such a comprehensive assessment, it is informative to consider three widely acknowledged problems with what most clinicians and laypeople believe to be true about schizophrenia.

First, the concept of schizophrenia has never actually been shown to have either **construct validity or predictive validity**. That is, there has never been sufficient scientific evidence that the putative signs and symptoms of schizophrenia are inter-correlated and hence constitute a genuine syndrome; the fact that the *DSM* listing for schizophrenia stipulates certain features as criteria does not prove that those features constitute a syndrome. This raises serious doubts about the scientific credibility of all instances of schizophrenia research.[ii]

Further, the research record concerning schizophrenia is replete with findings that are methodologically flawed, negative, unreplicated, inconsistent, weak, nonspecific or uninterpretable.[iii] Such findings do not provide support for scientific hypotheses about schizophrenia, although they do comprise massive amounts of data that lead some to talk as if various hypotheses have been tested and supported; but it is doubtful that any hypothesis about schizophrenia has ever been **rigorously** tested and supported. Thus, in addition to problems of construct validity, the concept of schizophrenia does not have any well-established predictive validity either. Although some would consider it heretical to say so, there has been no substantial improvement in scientific knowledge about schizophrenia over the past 100 years.[iv]

A second widely acknowledged problem with clinical and research use of the diagnostic category of schizophrenia concerns its well-documented **heterogeneity** with respect to criterial features, associated features, biological and psychological processes, and contextual features and processes (Heinrichs, 1993, 2001). Thus, individuals diagnosed as "schizophrenic" are likely to differ from each other even with respect to the clinical features that provide the basis for the diagnosis (i.e., the DSM criteria), as well as

with respect to biological, psychological, behavioral, and social **processes** that operate both internally and externally. Such "process heterogeneity" means that the clinical dynamics (course, outcome, response to intervention) of the condition also vary widely, as clinicians and researchers have repeatedly discovered (Poland, et al, 1994). Although this massive heterogeneity is widely acknowledged there is substantial disagreement about its significance. Those who are firmly committed to the existence of schizophrenia suggest that there is heterogeneity because schizophrenia is a disease (or multiple diseases) that is embedded in widely varying biological, psychological, or social contexts with which it interacts. They believe that, with time and research progress, such heterogeneity will become better understood and managed. Those who have no commitment to the belief that "schizophrenia" picks out a well-defined disease[v] suggest that the heterogeneity is part of the evidence that the category is scientifically and clinically meaningless, that schizophrenia does not, in fact, exist.[vi]

At the very least, it should not be **assumed** that one or the other of the interpretations of diagnostic heterogeneity is correct. This issue should be resolved on scientific grounds, and prior to such resolution, the scientific credibility of the diagnostic category is, at least, in doubt. Given its lack of established validity, that doubt is a serious one, since all that we know for sure is that many individuals have been grouped together under the label "schizophrenia," that these individuals exhibit massive heterogeneity at all levels of functioning, and that there is no current understanding regarding what (if anything) unites them.

Finally, this doubt regarding scientific credibility is reinforced by yet a third widely acknowledged problem concerning schizophrenia, the problem of **phenotypic**

definition (i.e., what trait is "schizophrenia" supposed to pick out?), which has emerged in recent years with increasing clarity in the context of genetic research. During the past two decades, spurred on by claims that the research record has clearly and strongly established that there is a genetic component in the etiology of schizophrenia (cf., Gottesman, 1991), significant research efforts have been directed at discovering the genetic basis of schizophrenia via microgenetic research methodologies (e.g., gene linkage analysis). Such research has generally been acknowledged to have failed so far in identifying specific genes or constellations of genes that are either a locus of genetic defect or markers for such loci. All putative findings (often announced to the public with much fanfare) have been discredited on various grounds, and at present there do not seem to be any "promising leads" in this area of schizophrenia research, although enthusiastic researchers often claim that there are.

The stock explanation of this research failure is (a) that the genetic basis for schizophrenia is quite complex (many genes, interactions with the environment) and (b) that there is a lack of a well-defined phenotype to target in the micro-genetic research. The first component of the explanation is a rehearsal of what are essentially *ad hoc* genetic hypotheses arrived at by the failure of traditional genetic research programs (family, adoption, twin studies) to reveal evidence for a single-gene hypothesis requiring no substantial environmental contribution. Although they would not frame it as I just have, researchers in this area acknowledge that the polygenic, multifactorial genetic hypothesis for schizophrenia is such that it is not known how many genes are involved, what their contributions are, what the contribution of the environment is, what developmental processes are implicated, etc. This is not much of a scientific hypothesis,

and it is one that simply begs the question of whether there is such a thing as "true schizophrenia".[vii]

The second component of the explanation of the failure of microgenetic research is a flat-out acknowledgment that the concept of schizophrenia is not well suited for research of this sort: i.e., it is too ill-defined, involving vague, evaluative, polythetic criteria[viii] and exhibiting massive categorical heterogeneity and no demonstrated construct validity. What research of this sort requires is well-defined phenotypes that can be accurately measured and that exhibit sufficient categorical homogeneity. Until such phenotypes are identified, micro-genetic research is stymied.[ix] What seems not to be adequately appreciated by researchers in this area or by consumers of such research is that the existence of a poorly defined phenotype also compromises the original family, twin, and adoption studies supporting the empirical presupposition of the micro-genetic research (viz., that there is a well-established genetic component in the etiology of schizophrenia.) What exactly does it mean to say that there is a well-established genetic component in the etiology of schizophrenia if "schizophrenia" is a poorly defined term without any clear referent? In all cases (e.g., family, twin, and adoption studies, gene linkage studies), empirical findings are essentially uninterpretable if the critical variable in the research is poorly defined. At most, empirically identified correlations, even if statistically significant, do little more than establish a correlation in a sample between some variable of interest (e.g., patterns of familial transmission) and **the label**, "schizophrenia". Under appropriate methodological conditions, such a finding might warrant an inference to a correlation between the variable and the label in a broader population. But, beyond that the research cannot and should not be further interpreted.

Thus, in the light of these three problems (validity, heterogeneity, definition) it would appear that the received view is nothing more than a set of unsupported (if meaningful at all) beliefs about a putative disease called "schizophrenia" and about the people supposedly afflicted with this "disease." Because of this, when clinicians classify individuals as having schizophrenia there is a substantial **loss of information** with no compensatory gains in either understanding of a person's condition or predictive power.[x] As we shall see below, the schizophrenia label actually obscures the complexity that must be managed if clinical goals are to be pursued effectively. The received view is a stereotype that provides a simplistic understanding of the people who are the subjects of clinical practice, and that introduces a substantial risk of error and harmful treatment.

Part 2: The Schizophrenia Stereotype Leads to Harmful Biases

In the case of severe mental illness, bias can result from any component of the received view of schizophrenia, and it can arise in at least five general areas of clinical practice: processing information, making inferences, clinical understanding, intervention, and clinical identities, roles, and relationships. Together, such biases undermine clinical practice and harm both the clinician and (especially) the people who seek help for severe mental illness.

Information Processing

Information processing includes, among other things, observation, attention, information search, memory, and information recording; such processes determine what information is available for clinical reasoning, judgment, and decision making, and as a consequence what information plays a strong role in shaping clinicians' feelings,

attitudes, and motivations. When information processing is influenced by the schizophrenia stereotype, the following bias is promoted:

> **Bias 1**: The tendency to observe, attend to, collect, record, remember, and highlight primarily a narrow range of information concerning pathological, clinically identifiable features (e.g., hallucinations, delusions, bizarre behavior, disorganized behavior, "negative symptoms"[xi])

In diagnostic practice guided by the schizophrenia stereotype, the kinds of information deemed to be of most clinical interest tend to be the pathological clinical features listed in the *DSM*. This increases the salience of such features, the categorizing of ambiguous events as instances of such features, the effort expended in systematically rooting them out, and the recording of them in clinical records. Thus, bias 1 leads to the **creation of a body of clinical information that is heavily weighted with respect to a narrow and impoverished range of features** that are readily identifiable and negatively valued by the clinician and are the primary currency in psychiatric discourse about schizophrenia. They are sought for in dyadic clinical diagnostic interviews, highlighted in clinical records, and considered sufficient for diagnosis.

The focus on these features is maintained at the expense of a much wider range of features not included in *DSM* criteria and not deemed of **primary diagnostic significance**. Thus, many types of information either are not attended to or collected or are relegated to a secondary or irrelevant status for the purposes of a diagnostic assessment: e.g., information regarding the person's functional capacities along a wide range of biological, psychological, behavioral, and social dimensions, or regarding the person's specific functioning in a wide range of personal and social contexts, or regarding the person's view of their life history, projects, goals, plans, outlook, and positive relationships. Those in the grip of the received view of schizophrenia tend to minimize

the importance of such information when it comes to figuring out **what is wrong with the person**. Either such information is not collected at all, or, if it is collected, its primary significance concerns figuring out what is an appropriate DSM diagnosis. This applies to the clinician's taking of the person's medical and social histories, as well as use of psychological tests and skills assessments. For example, such ancillary information is often used to corroborate a clinical diagnosis, using such investigative questions as: What is the person's diagnostic history?, what is the person's family history with respect to mental illness?, was the MMPI profile consistent with a diagnosis of schizophrenia?, was there a recent decline in functioning?, is there clinically significant impairment in functioning? Thus, even when information other than about the features in the *DSM* criteria for "schizophrenia" is collected, the operation of the stereotype tends to lead to the minimizing of its role in diagnosis and to locating it in a pathology-oriented framework.

Why are these consequences of bias 1 harmful? The building of a narrow, pathology-oriented data base poorly equips the clinician to understand what is wrong and what sorts of causal processes are in play in the person's world or the clinical setting. In addition, an impoverished, pathology-oriented body of information fails to provide the basis for an adequate understanding of the person, their life, their goals, and their values. The sense that there is something wrong with the person dominates, rather than being one part of, clinical activity. As a result, the individual whose life is at the center of clinical practice loses status and is disempowered, giving cause for alarm because the agency of the person is one of the most important factors in successful clinical practice.

Inferential Practices

The schizophrenia stereotype leads to clinical inferential practices that exhibit the following bias:

> **Bias 2**: The tendency to infer the existence of a core disease process that explains the presence of, and relationships among, clinical features, and to interpret events and features of the person as the manifestations of an individual biological disease condition

Thus, whenever information deemed relevant to diagnosis is evaluated, the clinician in the grip of the schizophrenia stereotype is primed to see groups of clinical features and events as inter-correlated and all being manifestations of a single, underlying disease; in addition, essentially ambiguous features and events (e.g., a report of an hallucination, a delusional statement, a bizarre act) are likely to be interpreted as manifestations of a brain disease.[xii]

Although some defenders of the schizophrenia stereotype acknowledge that schizophrenia might be, not a unitary disease, but rather a spectrum disease that varies along a number of dimensions or "multiple diseases" (the term "schizophrenia" is applied to different disease processes in different individuals), in individual cases it is invariably assumed that some disease process causes the clinical manifestations. However, there is no scientific evidence of what that disease process might be or of how the disease might be causally related to its alleged clinical manifestations. Clinicians who exhibit Bias 2 tend to ignore or minimize the importance of causal hypotheses not involving a core brain disease: direct environmental impact or environment-individual feedback loops; complex biological, psychological, and social interactions; and "normal" psychological processes involving choice, social learning, and attributional processes. Since individual features and events that gain clinical attention quite possibly result from such alternative sorts of

causal process, a rigid, stereotypic, disease hypothesis tends to obscure the real causes of a person's problems.

Bias 2 also leads clinicians to ignore the distinct possibility that the clinical features they see are **independent** of each other (e.g., a person's hallucinations are related to a biochemical dysregulation, while their delusional speech reflects a complex social learning history), or that they are **inter-related in some other way** (e.g., escalating arousal that makes it hard for a person who already has social skills deficits to manage a distressing social conflict), both of which are regularly discovered to exist.[xiii]

The common assumption among clinicians and laypeople that hallucinations and delusions form a meaningful pair and that they are the essential features of schizophrenia is an instance of the profound impact that the non-scientific clinical psychiatric tradition has exerted. But not even the *DSM* requires that hallucinations and delusions be present in "schizophrenia," and it certainly doesn't require that they be present together. No careful scientific investigations have established that delusions and hallucinations are empirically correlated with each other, and they are well known not to be specific to a diagnosis of schizophrenia. Thus, the belief that such features are inter-correlated and the consequence of the same core **brain disease** in "schizophrenia" can only reflect deeply-held ideological commitments. Why would one **assume** that hallucinations and delusions form a meaningful cluster just because they happen to co-occur in some cases?

Clinical Understanding

Clinical understanding affected by the schizophrenia stereotype leads to the following bias:

> **Bias 3**: The tendency to view people diagnosed with schizophrenia in terms of a disease model, according to which a core disease process drives the perception,

> thought, feeling, and behavior of the person; and, hence, to view the person as the victim of a brain disease over which he or she has no control.

As just discussed, biased inferential practices under the influence of the schizophrenia stereotype tend to promote **a simplistic causal understanding** of the condition of the person as involving a core disease process and the pathogenic cascades to which it leads: all pathological features of the person will tend to be understood as downstream causal consequences of the core disease process. Within such a framework, the person's perception, thought, feeling, and behavior are considered to be driven by an internally-located, pathological process,[xiv] and thus, they tend to be viewed as the psychologically meaningless causal fallout of a diseased brain. The alternative causal hypotheses mentioned above (viz. independence of problems, environmental causes, complex interactional feedback loops, normal psychological processes) tend to be ignored or minimized; and, a pathology-oriented view of the person as the passive victim of a brain disease tends to predominate.[xv] In this way, the schizophrenia stereotype compromises clinical practice by misleading clinicians about the character and complexity of the circumstances in which individuals are embedded and of the problems with which they are attempting to cope.

Bias 3 also tends to promote **a simplistic understanding of the people** (not just the causal nexus in which they are embedded) who suffer from severe mental illness. A view of the person as the passive victim of a brain disease leads to a substantial loss of understanding of both the **perspective** of the person and their **agency**. If a clinician tends to regard the person's perception, thought, feeling, and behavior as essentially the "psychologically meaningless causal fallout of a diseased brain," then both the person's perspective and their actions will be dismissed as manifestations of the core brain disease

process, things to be eradicated by treatment rather than to be grasped as essential components of clinical understanding. Such a dismissal will lead the clinician to lose sight of such things as:

> -the ways in which the person apprehends what is happening in a clinical setting
>
> -how the person reacts to being classified and treated in certain ways by clinicians and by others
>
> -the social roles that the person occupies, or the ways in which a person might resist pressures to occupy such roles (e.g., the role of "mental patient")[xvi]
>
> -the person's outlook on their life: their values, goals, aspirations, plans, prospects, historical understanding, and understanding of their current circumstances
>
> -the operation of the person's cognitive architecture and of normal psychological and social processes in the production of behavior
>
> -the ways in which a person's actions may well be quite legitimate in the light of their circumstances, their limitations, and the way they are treated

Without such understanding, much of what the person does will be unintelligible to the clinician, especially given the limitations of looking at only a narrow range of factors and assuming that a core brain disease causes the trouble. Without a genuine appreciation of the person's predicament from their own point of view, effective and respectful clinical relationships are difficult to establish, and that creates further impediments to the kind of understanding that should underlie diagnosis.

Intervention Practices[xvii]

Good clinical practice leaves no room for an impoverished or pathology-oriented data base, skewed inferences, or simplistic understanding. Unfortunately, these biases, in conjunction with various components of the schizophrenia stereotype, lead to a fourth

bias in clinical practice, one that concerns the design and implementation of clinical interventions:

> **Bias 4**: The tendency to target the putative brain disease and its manifestations as the **primary** object of intervention and to monitor symptoms as the principle measure of treatment success or failure.

Within the framework induced by the schizophrenia stereotype, the brain disease status of the person's condition occupies center stage, subordinating all intervention to its control and management. For these purposes, psychotropic drug therapy is the first and foremost form of intervention, and symptom monitoring is the first and foremost form of assessment regarding treatment success or failure. As a consequence of this bias, the wide variety of possible alternative causal processes alluded to earlier will tend to go unidentified and unaddressed. Bias 4 is dangerous because it tends to short-circuit serious clinical thought regarding intervention, leading to rote and ill-conceived intervention plans and leaving the clinician without adequate resources for understanding why a chosen intervention fails to be effective (e.g., non-response to drug therapies).

Clinical Roles, Identities, and Relationships

The last area in which the schizophrenia stereotype exerts a biasing influence concerns the social infrastructure of clinical processes and practices (the ways in which clinical identities, roles, and relationships are shaped):

> **Bias 5**: The tendency to create a social infrastructure for clinical activity in which the person's **identity** is that of a victim of disease over which he or she has no control, the person's **role** is that of a patient whose primary responsibility is to comply with treatments prescribed by the clinician, and the person's **relationships** involve occupying inferior status and authority relative to others.

Clinicians under the influence of the schizophrenia stereotype will tend to interact with persons diagnosed with schizophrenia as if they were passive victims of a disease process

who need to be treated by a physician using essentially biomedical intervention strategies and techniques. As a critical part of this interaction, such clinicians will tend strongly to "educate" the persons in their care to embrace the same view of themselves: persons diagnosed with schizophrenia are taught to believe that they are the passive victims of a brain disease called "schizophrenia". In this way, persons diagnosed with "schizophrenia" are led to internalize a stigmatizing social stereotype ("schizophrenia") and to embrace self-attributions of a sort (i.e., negative, internal, stable, global) known to have harmful personal consequences.

Clinicians under the influence of the schizophrenia stereotype will also tend to educate everyone else, including the family and friends of the person and other members of a hospital staff, along the same lines.[xviii] This creation of a shared understanding among all principal parties leads to a rigid structuring of the person's social world, in which their first- and third-person **identity** is that of the passive victim of a brain disease, in whom pathological features and behavior occupy a dominant focus of attention. As a consequence, the person's status, roles, and relationships in this social world are powerfully shaped and negatively influenced. Specifically, this form of patient education tends, through implicit and explicit learning and powerful social processes, to reinforce dysfunctional mental patient identities and roles, rather than creating a context in which a person can pursue meaningful alternatives. Further, the impact of such education includes: the demeaning of the person, the promotion of a passive orientation of the person toward their problems, the disempowerment of the person in their life, the undermining of the person's engagement in their own treatment, the creation of flawed

clinical relationships, and consequently the promotion of harmful and unproductive clinical and social processes.

Part 3: Conclusion

The received view of schizophrenia, then, is a stereotype that leads to a number of biases that influence the process of diagnosis and all other critical dimensions of clinical practice, undermine them, and promote a variety of harms. This stereotype and the biases it promotes have their roots deep within the culture: the broad socio-economic infrastructure in which mental health care is embedded, the reimbursement practices specifically regarding the financing of mental health care, the current zeitgeist within mental health practice, and the training and socio-economic reward structure of the clinical professions (Poland & Caplan, in press). What is required is a serious transformation of research and clinical practice, something that will require the development and dissemination of viable alternative forms of practice, a radical undoing of the deep entrenchment of current forms of practice, and a responsible management of the processes of change in order to safeguard the interests and well-being of those who are dependent upon clinical practices and social policies concerned with mental health.

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NOTES

[i] See Spaulding, Sullivan, and Poland 2003 for a framework in which the problems of severe mental illness are comprehensively identified and addressed, without reliance upon psychiatric diagnostic labels such as "schizophrenia".

[ii] See Boyle 1990 for important discussion of this line of critical discussion. See also Poland in press.

[iii] See Heinrichs 2001 for a review of a wide range of research hypotheses concerning schizophrenia.

[iv] Those in the grip of the received view of schizophrenia see the significance of this fact in terms of the immaturity of current science and the complexity of the disease: with more time and resources research will produce an understanding of the brain disease called "schizophrenia". However, the alternative hypotheses, viz., that there is no such thing as schizophrenia and that the concept is scientifically meaningless, seem better confirmed by the research record to date than are the received view and this optimistic outlook for the associated research program.

[v] This is not to say that there are no brain diseases at all; it is to say that a commitment to the idea that "schizophrenia" picks out a brain disease is currently groundless. Certainly it is possible that some individuals who happen to fall into a category defined by DSM-IV criteria for schizophrenia have, as part of their condition some brain disease or other. But, this in no way vindicates the category as a brain disease; and, in any event, it should be kept in mind that not just any condition that leads to problems is a disease.

[vi] The claims that the category of schizophrenia is scientifically and clinically meaningless and that schizophrenia does not exist inevitably strike those who are firmly committed to the concept as obviously false or at least overstated, since they believe that the category has proven useful in clinical and research practice and that people with serious problems meeting the diagnostic criteria for schizophrenia exist. How could the category be meaningless? How could the condition not exist? But being puzzled in this way is largely a reflection of an a priori and empirically unsubstantiated commitment to the hypothesis that schizophrenia exists.

[vii] It bears re-emphasizing at this point that critics are not denying that there is such a thing as severe and disabling mental illness; what is at issue is whether "schizophrenia" adds or detracts from scientific (and clinical) practice concerned with SDMI. In the case of the genetics of "schizophrenia", the claim is that the label is a severe impediment to research.

[viii] Polythetic criteria are those that involve multiple sets of sufficient conditions for category inclusion. Such criteria are legitimately employed in cases where a given condition expresses itself in a variety of ways. The problem with employing polythetc criteria in the case of schizophrenia is that so far as we know there is nothing but the criteria: there is no known underlying condition that is variously expressed. Put another way, to assume the legitimacy of polythetic criteria is simply to reiterate the assumption that "schizophrenia" picks out a disease condition; but the evidence for this assumption does not exist.

[ix] Which is not to say that it does not proceed full throttle. Researchers in this area are scrambling to identify any trait that is sufficiently well defined and measurable and that exhibits even some empirical association with the (ill-defined) diagnostic category of schizophrenia. Examples of such traits are: smooth pursuit eye-tracking deficits; and, P300 and P50 evoked potential abnormalities associated with sensory processing of sounds (novelty, gating).

[x] For example, if a person reports hallucinations that are distressing and disruptive, a clinician might consider prescribing an anti-psychotic drug known to be helpful in managing hallucinations. But, a diagnosis of schizophrenia is not required for predicting that the drug might be effective and it adds nothing to understanding what is going on in such a case.

[xi] The so-called "negative symptoms" of schizophrenia include: flat affect, lack of motivation, poverty of thought, and poverty of speech.

[xii] The report of an hallucination or the making of a delusional statement, for example, are essentially ambiguous because: although hallucinations are real phenomena, not every report of an hallucination is an accurate report; and, although delusional statements are often made, they can have very different sorts of clinical significance ranging from putative pathophysiological dysregulations to complex social learning histories and outright manipulations.

[xiii] See Spaulding, Sullivan, and Poland 2003 for a clinical approach to severe mental illness in which just such fine-grained causal analysis is pursued.

[xiv] A particularly important consequence of the bias involved here is a tendency to "decontextualize" the person's problems. The schizophrenia stereotype implies that a core disease process exists within the individual and that a) it is perhaps partially caused by factors in an environmental context, b) it can have consequences in an environmental context, but c) neither causal antecedents nor causal consequences of the disease nor any other features of the context are parts of the disease process itself. That is, the context can be completely detached from an understanding of what is wrong with the person.

[xv] See Spaulding, Sullivan, and Poland 2003 for discussion of why a decontextualized view of severe mental illness leads to serious misunderstanding of the nature of a person's problems and of why according causal privilege to core disease processes within an individual's brain is quite misguided (i.e., causation can move in all directions, it can involve features and processes at **any** level of causal analysis, and it can involve "normal" as well as pathological processes.)

[xvi] The fact that traditional psychiatric practices have tended to reinforce dysfunctional roles, identities, and behavior through a focus on pathology, a lack of personal respect, and treatment that reinforces disability, makes such resistance quite poignant.

[*xvii*] There are two further biases (concerning intervention practices and clinical identities, roles and relationships) that the schizophrenia stereotype tends to promote, and which I shall mention but not discuss in detail. Each is intimately bound up with diagnostic practice and interacts with the other biases introduced by the stereotype.

[xviii] From the point of view of the clinician, building appropriate relationships based upon a shared understanding of the person is critical to implementing effective clinical intervention plans and to supporting the patient in their efforts to manage their disease. But, of course, such a point of view makes a number of questionable assumptions.