Emotional Impact on Logic Deficits May Underlie Psychotic Delusions in Schizophrenia

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Abstract

Psychotic delusions, defined as false immutable culturally discordant beliefs, constitute an endemic symptom among patients with schizophrenia. We examined whether the deficits in reasoning responsible for the formation and maintenance of delusions are a product of inappropriate discrimination between relevant and irrelevant data or rather the product of inappropriate processing from data that is appropriately relevant. We used a Logical Reasoning Task that presents items that test inferences and the choice of relevant information within two separate sections, one of which uses language designed to be affect-neutral, the other of which uses language with violent imagery that is designed to be affect-laden. Our preliminary results indicate that all patient groups show significant deficits on the types of reasoning that we measured, relative to healthy controls. While non-delusional patients also showed deficits in reasoning, delusional patients were unique in that their performance on reasoning tasks was most similar to that of healthy individuals *as long as the context was affect-neutral*. In the affect-laden contexts, however, delusional patients' reasoning significantly declined, while healthy individuals' performance was only mildly affected. We suggest that delusional patients may suffer from a vulnerability to arousal which causes them to commit types of reasoning errors similar in kind to those made by healthy controls under much more severe forms of stress.

Logic Deficits and Psychotic Delusions

A 36-yr-old man is arrested upon trying to enter the United Nations, arguing that he is Ambassador to Alpha Centauri; a 48-yr-old homeless woman believes that she is the wife of Thomas Aquinas; a 22-yr-old college student gradually becomes convinced that the CIA, FBI, and New York City Police Department have been following him since birth. All three of these individuals suffer from "psychotic delusions," the adoption of false, often bizarre, beliefs that are held in spite of ample evidence that contradicts the belief or greatly diminishes its likelihood. Psychotic delusions are characteristic of a number of mental and neurological illnesses, but are most common in schizophrenia and the manic phase of bipolar disorder. Delusional ideation produces much of the social alienation, lack of treatment compliance, and poor functioning associated with these diseases. Even so, the neurobiological and cognitive mechanisms responsible for the formation and maintenance of psychotic delusions are one of the least understood aspects of these illnesses.

In the absence of sensory distortion, it seems reasonable to assume that delusional patients have access to the same information about the world as everyone else. Yet if this is the case, we are left with two large puzzles. The first is why patients, who presumably start from the same premises as healthy individuals, manage to reach such radically different conclusions. The second is why patients' false conclusions revolve so consistently around certain themes, such as paranoia and grandiosity. Our study was designed to begin to address both questions.

Since logic was first developed to formalize rationality, it makes sense that it would serve as a useful tool in modeling aberrations of reason. If individuals infer conclusions from a set of premises by applying a pre-established category of rules of reasoning, then false conclusions may be arrived at by either starting from false premises or by invalid inferences. The vast majority of literature on schizophrenia and logic address the second of these possibilities, beginning with von Domarus' idea

that patients with schizophrenia consistently use a specific fallacious inference (Von Domarus, 1944). More modern studies have tested patients' abilities to use standard logical inferences (Ho, 1974; Kemp et al, 1997; Watson and Wold, 1981). Related to these are studies linking poor performance on the Wisconsin Card Sorting Task (a rule-generating task) with schizophrenia (Pantelis et al, 1999), and the correlation between delusion thought and a peculiar style of reasoning in which patients "jump to conclusions" (Huq, Garety, Hemsley, 1988). Results from these studies have been inconclusive. Though valuable as preliminary data, Mujica-Parodi, Malaspina, and Sackeim (2000) have argued that these studies are flawed because of the heterogeneity of patient samples, floor effects, and the conflating of different types of logic as if they reflected the same type of reasoning.

Surprisingly, little attention has been paid to the other possibility by which false conclusions may be reached: inappropriate choice of premises. This absence is all the more striking because modern empirical studies of normal cognition suggest a paradigm of reasoning, *mental models*, that is radically at odds with that presupposed by standard tests of logic (Johnson-Laird, 1995). The most obvious difference lies at the level of premises. Tests of deductive logic provide pieces of information that are explicitly described as the material from which conclusions ought to be derived. In the real world, however, our premises are seldom laid out so neatly before us. Instead, a large portion of our mental work must go towards discriminating between relevant and irrelevant information, choosing that from which we will later derive conclusions. Since available premise-groups are usually incomplete, most "conclusions" are actually closer to be being *hypotheses*, over-inclusive sets that are then restricted by confrontation with new evidence. Johnson-Laird's experiments suggest that the capacity for recognizing counter-examples to our provisionary models, and these models' subsequent revision, are just as critical in the formation of belief systems as the inferences that initially give rise to the models (Oakhill and Johnson-Laird, 1985). Since perhaps the most characteristic feature of delusions is not the strangeness of the conclusions reached, but of their perseverance in the face of systemic evidence to the contrary (Jones and Watson, 1997) one would expect the recognition and application of counter-examples to be a cognitive ability that is seriously impaired in patients with delusions. Yet it is a phenomenon that has hardly been studied in this population.

Our central hypothesis is that it is the failure to sort premises: distinguishing relevant from irrelevant information and, in particular, the recognition and application of counter-examples, that is responsible for delusions. We suggest that such a failure may be the result of a normal prioritization of neural resources during periods of emotional stress, inappropriately activated in patients. While it may be natural for healthy individuals to initially form false or partially false models, these models are normally revised in the face of contradictory evidence. In the presence of anxiety or fear, this self-correcting mechanism may be temporarily disabled in order to devote full mental resources to avoiding the cause of threat. Once the threat has passed, the mechanism would become functional once more and false beliefs would be revised. If, in the case of patients, the amygdala (and/or other medial temporal lobe structures responsible for perceiving threat) is hyperactive, then the same process may occur with far less provocation. Under the strain of a more or less continual state of emotional stress, the recognition of counter-examples may become disabled long enough to allow false beliefs to become entrenched. The advantage of grounding the creation of delusions in emotion, rather than the reverse, is that it provides an explanation for the relative thematic uniformity found between different patients' beliefs. The patient who experiences a state of fear or anxiety without a clear referent for it will presumably feel the need to explain that feeling; the explanatory structure that the patient creates, however, will very likely be flawed because of the mechanism hypothesized above.

The view of paranoid delusions as quasi-rational stories that are created to explain pre-existing emotional states is consistent with a wide variety of preliminary clinical evidence connecting psychosis and affect, including vague feelings of fear and anxiety reported during the prodromal (pre-symptomatic) period in schizophrenia (Henmi, 1993; Wiedemann *et al*, 1994) and correlations between levels of perceived stress and preoccupation with delusions (Myin-Germeys, 1999). It has long been known that there is a strong correlation between relapse in schizophrenia and even minor increases in stress (Doering et al, 1998). At a physiological level, schizophrenic patients' MRI's are characterized by lower hippocampal volumes than that of controls (Kegeles *et al*, 2000), these patients appear to have increased cortisol levels (a "stress hormone") (Goldman *et al*, 1993), and increased cortisol levels over sustained periods of time are known to be toxic to the hippocampus (Sapolsky *et al*, 1990). Other studies have linked prenatal damage to the limbic system with later development of schizophrenia (Lipska *et al*, 1993; Hanlon, Astur, and Sutherland, 1999). It may be that a vulnerable amygdala and/or medial temporal lobe abnormalities could be both responsible for the increased cortisol levels as well as for the common generalized feelings of anxiety and fear associated with the emergence of psychosis. It may also be the case that insult to the hippocampus and structures also involved with the circuit associated with arousal, such as cortical regions and the thalamus/hypothalamus, could be at least partly responsible for whatever defect in reasoning is responsible for the development of delusional beliefs. Most provocatively, this line of thought, if valid, could

shed light on the relationship between delusional ideation, clinically defined, and the anecdotal truism that even normally rational individuals become irrational when they are upset (or angry, or fearful, or euphoric, for that matter). It might also eventually provide a link between a particular symptom—delusions—and its neural mechanism among disorders, such as mania, psychotic depression, and schizophrenia, that are currently considered to be unrelated.

For our study, we tested the following hypotheses:

I. Patients with delusions are impaired in their ability to distinguish relevant from irrelevant premises in the formation and restriction of mental models.
II. Patients with delusions use logical inferences equivalently to healthy and patient controls.
III. All individuals (patients and controls) are impaired in their ability to distinguish relevant from irrelevant information when they are subject to emotional stress. We hypothesize that this task of sorting is more vulnerable to the effects of emotional stress than the task of logical inference-making.
IV. Patients are more vulnerable to stress, and therefore require less emotional provocation than do controls in order to reach the level at which this cognitive mechanism is impaired.

The Logical Reasoning Task

Dr. LR Mujica-Parodi and Dr. Harold Sackeim developed a *Logical Reasoning Task* (LRT) that avoids many of the problems present in previous attempts to test logic deficits in patients. Unlike previous tests, this task tests both logical inferences and the choice and evaluation of premises. The evaluation of premises sections are essentially an inverse of the inferences sections, in which the conclusion is given, and the subject is instructed either to identify information that would support the conclusion or to identify information that would contradict it. Items that are identical in form are presented within both affect-neutral and affect-laden contexts, counter-balanced for order. The affect-laden sections use threatening language in order to provoke a state of mild arousal, measured by visual analog scale and skin galvanic response. Responses are circled from a list of possible answers that are randomly spaced along the page. Extensive pilot testing ensured that LRT avoids floor effects by testing only inferences that most (60%-70%) healthy adults without any formal education in logic found to be intuitive and representative of "everyday reasoning." Most importantly, we established that both controls and patients are able to well-tolerate the task. The LRT is comprised of 60 items, 30 for the neutral section and 30 for the affect-laden section. For each section, 10 items test inferences, propositional premise choice, and class (quantifier) premise choice, respectively. The entire LRT takes approximately one hour to complete. Items on the LRT are scored in a manner that permits one to discriminate between errors of premise over-inclusion, errors of premise exclusion, and errors of contradiction. Subjects are able to choose more than one response. A score of 1 point is given for every item that includes the correct answer as long as there is no contradiction entailed by the subjects' responses. In Example 1 shown below (Table 1), the subject's choice of the second and third responses or the third and fourth responses would entail a contradiction. If the subject chooses more responses than are necessary, this is indicated by separate scoring for over-inclusion (with 1 additional point for each additional response). Similar scoring is done for under-inclusion. Separate scoring also records number of contradictions and choice of "not-enough-information" responses (which also indicate, with under-inclusion, degree of premise exclusion). Subjects are given five practice items before beginning the test, three of which have the correct answers marked, and two of which the subject completes.

Table 1: Examples from the Logical Reasoning Task

Example 1: Inferences, Affect-Neutral Condition
If John has missed the bus, then he will be late.
John has missed the bus.
What follows from this?
Nothing follows.
John will not be late
John will be late. ←
John has not missed the bus.
contradiction
contradiction

Example 2: Inferences, Affect-Laden Condition
If they are stabbing me, then they will kill me.
They are stabbing me.
What follows from this?
Nothing follows.
They will not kill me.
They will kill me. ←
They are not stabbing me.

Example 3: Premise Choice, Affect-Neutral Condition (Propositional logic, Counter-example)
John says that he will be late.
What information, together, makes you think that he is wrong?
John is late.
John has missed the bus.
Nothing makes me think that he is wrong.
John has not missed the bus. ←
Only if John is late, will he then miss the bus.
Only if John misses the bus, will he then be late. ←

Study Design

Subjects:
For these preliminary data we looked at responses from 28 patients, divided by cognitive symptom profiles, and 16 healthy controls. Of the patients, 10 had well-developed delusional systems, 5 were thought-disordered,

and 13 were neither delusional nor thought-disordered (with hallucinations as the primary symptom). Our completed data will contain 50 subjects in each of the 3 groups, as well as a group of healthy controls with high degrees of magical ideation. Diagnosis and symptom severity were determined using the Diagnostic Interview for Genetic Studies (DIGS). Patients were matched for symptom severity and medication status. For our final analysis, all three groups will be matched for Verbal IQ (using the Weschler Adult Intelligence Scales), age, and education. For our preliminary analysis, patients and controls were not matched due to the relatively small N, although ANCOVA's determined that covariates of education, gender, age, and education were not significant confounds. Both patient groups and controls were relatively well-educated, averaging two years of college, with no formal training in logic.

In our sample there were more male patients (21) than female patients (7), reflecting the general distribution in schizophrenia, and more female control subjects (10) than male control subjects (6). All subjects signed informed consent for this Institutional Review Board-approved study. Patients were recruited from the New York State Psychiatric Institute's Schizophrenia Research Unit, the Washington Heights Community Unit, and affiliated out-patient clinics. Controls were recruited from the local community, and were screened using the Psychosis Proneness Scales developed by Chapman and Chapman (Chapman et al, 1994).

Procedures:
Control subjects were screened using the Psychosis Proneness Scales. All subjects were administered the LRT and several sections of the WAIS (testing spatial inferences, abstraction without use of counter-examples, vocabulary, and working memory). Blind symptom-profiling for patients was performed post-hoc to avoid bias.

Results:
We performed ANOVA to determine differences between subject groups and test types (with post hoc t-tests), ANCOVA to screen confounds of age, sex, and education, and paired t-tests for individual subjects on affect neutral/laden condition to test the effects of arousal on performance. The results are summarized below in Table 2.

Differences between patient and control groups reached statistical significance for all types of reasoning (inferences: p=.005; premises(prop): p=.024; premises(class): .012). Performance was highest for healthy controls, followed by—in descending order—delusional patients, patients hallucinating only, and thought-disordered patients. This same pattern was present for all three types of reasoning. Healthy controls displayed a slight (non-significant) drop in performance when assessing relevance under mild arousal (premises(class): p=.083/df=15). Delusional patients displayed the same pattern, but in a significantly exaggerated form (inferences: p=.004/df=9; premises(class): p=.033/df=9) This suggests that delusional patients have a relatively intact ability to reason under neutral conditions, with a particular vulnerability toward emotional arousal. Thought-disordered patients displayed an inverse pattern, improving under emotional arousal (premises(class): p=.035/df=4), which perhaps reflects an inability to maintain adequate arousal under normal conditions. Healthy controls, delusional patients, and thought-disordered patients may be viewed as occupying different initial locations on an inverted "U," where—following the Yerkes-Dodson Law— performance initially improves with small degrees of arousal but suffers with increasing amounts of stress. In this case, stress-level is held constant, with different vulnerabilities to arousal accounting for the different locations on the curve. The types of errors made were also different for different groups. Delusional patients generally, and particularly under arousal, showed a tendency to shut out relevant information (p=.067/df=9), again an exaggeration of the healthy controls' response to stress (interestingly, patients who only hallucinated showed even more of an exaggeration in this regard). Thought-disordered patients, on the other hand, were more likely than other groups to assign inappropriate weight to irrelevant information (p=.05/f=2.3).

Table 2: 3 Types of Reasoning x 4 Subject Groups

	Inference	Prop	Class	
Del	0.54±22	0.31±28	0.46±.28	
Th Dis.	0.24±.18	0.11±.09	0.12±.08	**NEUTRAL**
Halluc	0.48±.20	0.29±.15	0.40±.27	**CONDITION**
HC	0.65±.19	0.45±.23	0.62±.28	
	Inference	Prop	Class	
Del	0.35±.24	0.24±.19	0.33±.25	
Th Dis.	0.26±.19	0.15±.10	0.20±.13	**AROUSED**
Halluc	0.41±.25	0.21±.13	0.28±.20	**CONDITION**
HC	0.67±.23	0.41±.24	0.52±.29	

Three important preliminary conclusions to be drawn from this data are that: a) all patients groups show significant deficits in both inferences and cognitive gating relative to controls; b) of all patients, delusional patients infer most similarly to healthy individuals, except in the presence of emotional material, which also seems to affect their reasoning most dramatically; and that c) the "irrationality" of delusional patients (which is affect-driven and shuts out relevant information) appears to be quite different from that of thought-disordered patients (which is affect-independent and reflects and inability to "screen-out" irrelevant information). This last point is of particular relevance because it suggests differences

between symptoms-types that may be relevant not only from the point of view of etiology, but of treatment (delusional patients may benefit from adjunctive benzodiazepines and/or cognitive-behavioral-therapy in a manner that thought-disordered patients may not, for instance). Interestingly, delusional patients' abnormal vulnerability to arousal, combined with healthy controls' less dramatic decline in cognitive gating under arousal, raise provocative questions regarding the degree to which delusional patients' reasoning may resemble normal controls' reasoning when normal controls are under more pronounced levels of stress.

Implications and Future Work

"Cognitive" versus "Sensory" Neural Gating

As shown above, our data suggests that all three patient groups had significant deficits in cognitive gating. In investigating the neurobiological roots to such a deficit, we have considered the possibility that deficits in cognitive gating may be rooted in a more fundamental "lower-level" deficit in "sensory-gating," whose deficits have been shown to be ubiquitous to schizophrenia. While "cognitive gating" is defined as the discrimination between relevant and irrelevant conceptual information, "sensory gating" can be similarly understood as the discrimination between relevant and irrelevant sensory data.

One explanation that could link cognitive and sensory gating is a "hierarchical" hypothesis for information processing, in which sensory data is sorted through a series of neural "filters," traveling first through a "coarse-grained" filter, then through filters that are progressively more "fine-grained" as attentional levels (and presumably levels of abstraction) are increased. This hypothesis is testable, for it predicts that poor performance on tests of higher-order filtering will also entail poor performance on tests of lower-order filtering. However, the converse is not true; performance on tests of lower-order filtering will not entail equivalent performance on tests of higher-order filtering (since information may be adequately filtered at a lower level before stumbling at a faulty higher-level filter). We would expect the brain activation associated with performance to behave equivalently. Thus we would predict that areas of the brain activated during higher-order filtering will entail (i.e., will correlate positively with) activation of areas of the brain associated with lower-order filtering (since all information that that passed through higher-order filters had to be processed through lower-order filters first). However, the converse will not be true; areas of the brain activated during lower-order filtering will not entail areas of the brain activated during higher-order filtering. M.-Marsel Mesulam [1998] proposes a similar hierarchical "critical gateway" model in which "lower" processing of sensory information contributes to "higher" processing of cognitive material along certain pre-established pathways. In Dr. Mesulam's model, the level of neurological impairment (i.e., whether related to "lower-order" global deficits like multimodal anomia or "higher-order" deficits like category-specific anomias) also results from the point in the hierarchy at which processing is disrupted. We are currently conducting a neuroimaging (fMRI) study looking at different information-gating processes (including the Logical Reasoning Task) operating at different "levels" to test this hypothesis.

While prominent, delusions are only one of many cognitive and perceptual symptoms of schizophrenia, which include thought-disorder and hallucinations. The elucidation of filter-level deficits, and their relationship to *specific* signs and symptoms of schizophrenia, is important in responding to a cogent criticism placed by Frith (1979), that the disabling of a sensory filtering mechanism in schizophrenia, if it exists, cannot be at a general level. This is because a generalized gating deficit would result in far more neurological disability than is actually clinically observed. However, the degree to which patients are disabled varies quite a bit, varying between extreme disorganization and intricately-constructed delusional belief systems. As Frith (1979) earlier has suggested, the precise symptomatology of the illness may be based on the *degree* to which gating is impaired. It may be that paranoid patients have relatively well-preserved lower-level filtering, while only higher-order filtering is impaired. Thought-disordered patients, on the other hand, may represent a more severe form of the illness in which lower as well as higher level gating is affected. Drawing these connections will be important not only theoretically, in establishing a unitary model of schizophrenia which accounts for varied symptoms, but also potentially in developing medications that are tailored to treating specific symptoms. If the mechanism associated with neural filtering does exist upon a continuum, ranging from the basic sensory gating involved in attention (at the lowest end), to the cognitive gating required to separate relevant from irrelevant information (at the highest end)—with each level dependent upon the one "beneath" it—then it may be the case that the stage at which filtering is impaired is responsible for the cognitive symptom picture (thought-disordered vs delusional, for instance) with which a schizophrenic patient presents. Higher-order filter deficits, with lower-order sensory gating that is still intact, may produce symptoms that look like belief systems that are flawed because they are constructed based on inappropriate choice of information (including inattention to counter-examples), but which remain fundamentally self-consistent. Patients whose filtering deficits have progressed to include *both* higher and lower-order gating, may produce a more general disorganization in which inferences, and therefore self-consistency, may no longer be possible. This picture is akin to Frith's model (Frith,

1979); he suggests that delusional ideation may exist as a less severe form of the disease, in which some aspects of cognition are still well-preserved. Based on the literature on sensory/sensorimotor gating and selective attention, we will be primarily looking at activation in the dorsolateral prefrontal cortex, hippocampus, striatum, and thalamus/hypothalamus.

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