Jumping to conclusions: A network model predicts schizophrenic patients' performance on a probabilistic reasoning task

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This article extends computational models of schizophrenia that focus on the negative aspects of this syndrome to behavioral biases that are associated with a positive symptom of schizophrenia, namely delusions. The phenomenon studied is the "jump-to-conclusions" style of reasoning that is characterized by delusional patients—in comparison with controls—whereby they make less-informed decisions when an option to collect more decision-specific information is available. Simulations show that these differences can be mimicked by modulating the gain parameter—associated with variations in dopamine level—in a simple network model.

Disturbance in the prefrontal cortex (pfc)—mesocortical dopaminergic (DA) systems is implicated in the aetiology of schizophrenia (Meltzer & Stahl, 1976). Cohen and Servan-Schreiber (1992) focused on those behaviors associated with the negative symptoms of schizophrenia and proposed an abstract model that accounted for the behavioral differences between patients and controls. Through modeling the modulatory effect of DA on behavior, they argued that a reduction in DA explained performance biases associated with the negative symptoms of schizophrenia. The present article extends Cohen and Servan-Schreiber's observations to examine those behaviors associated with the positive symptoms of schizophrenia and to therefore explain a different aspect of the delusional-psychotic state that modelers and theorists have not previously considered.

The layout of this article is as follows: First, we review empirical research demonstrating behavioral biases on the "beads" task between controls and patients who exhibit the positive symptoms of schizophrenia. We then propose an abstract model that—we hypothesize—captures the behavioral differences between delusional patients and controls. Next, we present simulations that show how varying a parameter that is associated with variation in DA levels changes the model's behavior from normal to delusional performance.

Reasoning on the Beads Task and Schizophrenia

Reasoning biases may contribute to delusion formation and maintenance (Garety & Freeman, 1999; Garety &

Hemsley, 1994; Garety, Hemsley, & Wessely, 1991; Young & Bentall, 1997). A small variety of reasoning tasks have been employed to explore reasoning biases (e.g., syllogistic tasks and conditional inference tasks), often yielding inconsistent results (Kemp, Chua, McKenna, & David, 1997). Such inconsistencies have led researchers (Garety & Freeman, 1999) to suggest that—rather than a generalized reasoning bias—there is a more specific data-gathering bias, or a "jump-to-conclusions" style of reasoning that contributes to delusion formation and maintenance. To explore this bias, the most common approach has been to employ probabilistic judgment tasks, in particular the beads task (Garety, 1991; Garety & Freeman, 1999; Garety & Hemsley, 1994; Garety et al., 1991; Huq, Garety, & Hemsley, 1988).

In this article, we focus on the behavioral phenomenon jump-to-conclusions, as exhibited on the beads task. This task has a standard paradigm in which participants are required to judge from which jar (out of two) different colored beads are being drawn. One jar might contain 85 beads of one color (e.g., red) and 15 beads of another color (e.g., blue). The second jar contains the same number of beads, but with the reverse distribution (e.g., 15 red and 85 blue). Participants—knowing a priori the distribution of beads in the jars—are shown a series of beads that are drawn one at a time from one of the two jars, with each bead being replaced in its original jar after the participant has seen it. Participants are required to indicate when they are confident enough to make a judgment on which jar the beads are being drawn from.

Garety and colleagues (Garety et al., 1991) examined behavioral differences between clinical groups using the beads task. Four groups of participants were recruited: a schizophrenic group (whose members were also delu-

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sional), a paranoid-delusional disorder group, a clinical control group (anxiety disorder), and a "normal" control group. Two conditions were measured using the beads task. First, belief formation was measured by recording the number of drawn beads that was required for each participant to reach a decision. The second condition used by Garety et al. (1991) measured the process of belief formation and maintenance by asking participants to indicate the subjective probability of the bead being drawn from Jar A or Jar B on each experimental trial. The number of colored beads drawn in each trial was predetermined, and the trial was terminated after 20 beads had been drawn.

A significant effect of group was found in the first condition: The schizophrenic and the paranoid-delusional disorder groups requested fewer beads before making their decisions. In the second condition, which examined belief formation and maintenance, Garety et al. (1991) found a stronger effect of disconfirming evidence in the paranoid-schizophrenic and paranoid-delusional groups. Participants in these groups were more likely to revise their estimate of which jar the beads had been drawn from when presented with one item of disconfirming evidence (i.e., being shown a bead of the opposing color). With the same evidence, the two control groups were more likely to make no change, or even to slightly increase their initial estimate. No differences were found between the paranoid-schizophrenic group and the paranoid-delusional disorder group. To summarize, Garety et al. (1991) found that deluded patients required less evidence (i.e., requested fewer beads) to reach a decision, but were more willing to change their mind in light of minimal disconfirmatory evidence. Garety et al. termed this pattern of behavior in patients a *jump-to-conclusions* style of reasoning.

The jump-to-conclusions style of reasoning appears robust and has been replicated (Dudley, John, Young, & Over, 1997a). However, a secondary factor that emerged from further research (Dudley et al., 1997a) is that—although patients require less information before making their judgment—both controls and patients are invariably correct when they make their choice. This observation suggests that the variation in performance is not the consequence of a deficit in performance; rather, it is a modulation of performance. The present model seeks to mimic the bias for less information while still retaining the ability to make the correct decision.

The Model

Intraindividual differences are modeled as a variation in a network model's gain parameter. The gain parameter is associated with DA function, and variations in DA function are associated with delusional states. The DA hypothesis of schizophrenia implicates enhanced DA function in the pathophysiology of the positive symptoms (e.g., delusions) associated with schizophrenia (Abi-Dargham et al., 1998; Breier et al., 1997; Lieberman, Sheitman, & Kinon, 1997; Meltzer & Stahl, 1976; Seeman, 1987). The association between DA and the delusional state has been deduced using a variety of techniques. Neuroleptics

that block DA receptors also improve psychotic symptoms (Seeman, 1987), whereas psychostimulants (e.g., amphetamines and cocaine) that enhance presynaptic DA neurotransmission can induce psychotic symptoms (Connell, 1958). Cohen and Servan-Schreiber (1992) modeled the negative symptoms associated with schizophrenia as a decrease in the gain parameter. In the present model, the delusional state associated with the positive symptoms of schizophrenia is represented as an increase in the gain parameter.

The gain parameter (see Equation 2) modulates the input-output relationship for each node in the network, and this parameter has been associated with mesocortical dopaminergic function (Ashby, Isen, & Turken, 1999; Cohen & Servan-Schreiber, 1992; Gasbarri, Introini-Collison, Packard, Pacitti, & McGaugh, 1993; Hollerman & Schultz, 1998; Kandel, Schwartz, & Jessell, 1995; Servan-Schreiber, Printz, & Cohen, 1990) in the human cortex. Neurons—as suggested by Servan-Schreiber (1990)—operate in "noisy" environments. Interconnected neurons receive signals from other neurons, and these input signals govern the likelihood of that neuron firing. However, these signals are received embedded in background noise that may mask the input signal from an adjacent neuron and thus affect the likelihood of that neuron firing. Thus, any change to the signal-noise ratio across a network of neurons will alter the likelihood of the neurons in that network firing when given some input signal. A widely used function relating input to output is of a logistic form (see Equation 2) that incorporates a free parameter (θ), known as the *gain parameter* (see Figure 1). In the to-be-presented simulations, we examine model performance under different values of θ .

Previous research supporting our hypothesis that DA is involved with the behavioral biases that are associated with delusional states also suggests loci at which DA operates. In Krickhaus, Donahoe, and Morgan's (1992) review, they argued that hippocampal hyperactivity—in particular, DA₂ receptors in the CA₁ region of the trisynaptic loop—is primary in mediating delusional states. The role of the hippocampus is further confirmed in research employing a variety of methodologies. Heckers (2001) reviewed studies demonstrating how results from neuroimaging complement postmortem and behavioral studies that have shown that abnormalities in the hippocampus are associated with a history of delusions. Krickhaus et al. (1992) further suggested that delusions are a consequence of an imbalance between the association cortices and the hippocampus; this in turn mediates the generation of representations. Furthermore, neural network models proposed by Chen (1994, 1995) further support the role of associative cortices and help delineate the cognitive processes involved with the behavior of interest.

Chen (1994, 1995) modeled the role of DA in delusional behavior, targeting hippocampal-cortical interactions. He used an attractor neural network (ANN; Hopfield, 1982) that was specified using a Hebbian learning rule (Sejnowski, 1977). A typical use of ANNs (the one employed by Chen, 1994) is to model the formation

Figure 1. A graphic example of the logistic function for two values of the gain parameter: For the same input level, output increases as the gain parameter increases.

of memory states. In the learning stage, ANN is presented with stimuli that cause stimulus-specific activity. Hebbian learning rules are used to adjust synaptic weights so that the ANN can actively sustain the representations of stimuli. For example, according to the Hebbian rule, the weight between two nodes that are simultaneously active will be increased, whereas the weights will be decreased between asynchronous nodes. In this manner, ANN is able to learn—through repeated presentation of stimuli—to store representations of those stimuli. ANN's ability to recall (or correctly identify) those representations is tested through the presentation of a partial or noisy version of a previously presented stimulus.

Chen (1994, 1995) hypothesized that greater DA could be represented as less noise in an ANN. By decreasing noise levels and examining how ANN recognized noisy stimuli, he argued that the network's performance began to exhibit properties that were characteristic of delusional thought. A reduction of noise in ANN increases the probability that spurious attractors will dominate, given the presentation of a test stimulus. Under circumstances of greater DA, a test stimulus can be interpreted as something that it is not—"a delusional interpretation of input overrides normal interpretation" (Chen, 1994, p. 366). As such, Chen's (1994, 1995) model is most applicable to the misattribution of perceptual stimuli, or hallucinations, associated with psychosis.

Broadly, three cognitive processes might be involved with delusional behavior and thus the jump-to-conclusions style of reasoning: perceptual, attentional, and representational. Although Chen's (1994, 1995) model explained perceptual misattribution, it is unlikely that perceptual misattribution is responsible for variation in performance on the beads task. In the beads task, participants are required to recognize a bead as either red or blue. While misattribution may occur, according to Chen's (1994, 1995) model, it would most likely entail—

for example—red beads recognized as blue. Cohen and Servan-Schreiber's (1992) model implicated pfc attentional systems. However, modulation of these systems in delusional patients is associated with the modulation of the mesocortical DA systems, not the mesolimbic DA systems believed to be responsible for delusional behavior. We therefore argue that the most likely source of delusional behavior arises from mesolimbic DA systems modulating representational systems.

The DA hypothesis of delusions has further implications for the relationship between positive and negative symptoms. The argument that DA activity modulates the emergence of positive and negative symptoms and that these symptoms reside along a continuum would suggest that positive and negative symptoms cannot co-occur. Evidence suggests otherwise. Eaton, Thara, Federman, Melton, and Liang (1995) aggregated symptom ratings into monthly count data for 90 schizophrenic patients over a 10-year period. These data showed that some patients exhibit both positive and negative aspects of schizophrenia across one month and that symptom clusters appear to vary independently. However, aggregate count data of this form cannot be used to select between candidate causal models (i.e., one common cause, such as DA, or two discrete causes). Aggregated count data determine the prevalence, not the functional relationship between outcomes. It is plausible that general DA system volatility (i.e., abrupt swings from extremely high to extremely low levels of DA) might explain why some patients exhibit both positive and negative symptoms, whereas a persistent bias toward either a reduction or increase in DA might characterize patients that predominantly exhibit negative or positive symptoms, respectively. Alternatively, mesolimbic and mesocortical DA systems may vary independently, thus accounting for the apparent independence of symptoms in Eaton et al.'s (1995) study; we will return to this issue in the Discussion. In summary, we hypothesize

that the computational realization of dopaminergic innervation as variation in a network's gain parameter can be used to mimic reasoning biases demonstrated by delusional patients on the beads task. However, physiological evidence suggests that it is variation in the mesolimbic DA system rather than the mesocortical DA system that is most likely involved in the jump-to-conclusions reasoning bias.

Modeling the Beads Task

In the standard version of the beads task, participants are shown a sequence of beads from one of two jars (Jar A and Jar B hereafter). Participants know a priori that one jar contains more of one color than the other (e.g., a ratio of 85:15 red to blue in Jar A and a 15:85 ratio of red to blue in Jar B). Across repeated draws from a jar, they are instructed to state from which of the two jars the beads are being drawn as soon as they are very confident they can predict the correct jar. In this task, we assume that participants contrast two complementary hypotheses specific to each jar (H_A and H_B), where the information pertinent to each hypothesis is represented as activity in working memory systems characterized as McCulloch–Pitts (McCulloch & Pitts, 1943) neurons in a network: As more evidence for one or the other hypothesis increases, activity increases asymptotically. We develop this assumption from observations made in single-cell recording research. These data indicate that neural activity associated with choice options gradually rises as the strength of evidence for each choice option increases (Gold & Shadlen, 2001; Shadlen & Newsome, 2001).

Shadlen and colleagues (Gold & Shadlen, 2001; Shadlen & Newsome, 2001) trained nonhuman primates to make a categorical decision on the basis of the visual presentation of information (moving dots). The participants were presented—over a period of 1 sec—with a series of randomly positioned dots and were trained to decide whether there was a tendency for the dots to move in one direction or another (e.g., up or down). The task was made difficult by only allowing a few of the dots to present this lawful behavior. There were two choice options (the dots were moving upward or downward). Therefore, one means of formally representing information that was pertinent to the decision would have been to posit a single vector that was changed in light of new information. Analyses suggested, however, that distinct cell assemblies represent information pertinent to each decision option. Furthermore, these researchers observed that the variations in neuronal activity led to a decision when that activity crossed a given threshold (Shadlen & Newsome, 2001). When activity in one or another neuronal ensemble reached a particular level, that decision option was selected (see also, Brown et al., 2005).

The change in activity pertinent to H_A and H_B is weighted by prior knowledge of the distribution of beads in each jar (see Attneave, 1959). For example, if a red bead is drawn first, H_A is updated by a factor of 0.15 and H_B by a factor of 0.85 (see Figure 2). The model we posit differs from more usual network models in one impor-

tant way. Although most models employ learning rules to adjust weights over repeated trials, the model developed in this article fixes the weights a priori. This reflects the nature of the beads task we are modeling. The beads task is not a learning task, since participants are given information about the distribution of beads in the two jars. We suggest that this sets up two hypotheses: that the beads are being drawn from Jar A or that they are being drawn from Jar B. In order to recognize this prior information, we set the network weights to reflect the known probability that a bead is drawn from one jar or the other. In setting these weights, we further assumed a linear relationship between expected probability and weight. Although there are small biases in how people use probabilities in their decision making (see Prelec, 2000), the purpose of the research presented here was to mimic the deviations exhibited between delusional patients and controls, not to capture performance on the beads task itself. Because changing the weights in this model affects the proposed number of draws to a decision, we assume that the stated weighting is sufficient for the purposes of this paper and that these weights do not covary with the prevalence of delusions.

Activity is increased asymptotically, such that the change in activation for hypothesis i (ΔA_i) is proportional to current activation (A_i) and maximal activation,

$$\Delta A_i = (1 - A_i)h, \quad (1)$$

where h is proportional to the informational value of the bead drawn (0.15 or 0.85 in the above example). As with Servan-Schreiber and Blackburn (1995), we assume pre- and postsynaptic activation is functionally related by a logistic function with a constant negative bias,

$$P(A) = \frac{1}{1 + e^{-\theta(A_i)}}, \quad (2)$$

where θ represents the gain parameter.

In sum, we argue that weighted perceptual input modifies activity in neural representations of information pertinent to each hypothesis. Activation feeds into upstream attentional systems that select one or the other decision option. Following our earlier discussion of Chen's (1994, 1995) ANN model of delusional behavior, we argue that

Figure 2. Model schematic. Weighted (w_{ij}) perceptual input [red (R) and blue (B) beads] updates activation in hypothesis-specific nodes H_A and H_B .

delusional behavior is most likely associated with the mesolimbic DA modulation of representational systems. With reference to the model proposed here (see Figure 2), we argue that the logistic transformation of activation captures the expression of prior information. We further argue that weighted perceptual input (R and B in Figure 2) and upstream attentional systems (ΔP in Figure 2)—although important to the decision—are not biased in the case of delusional behavior.

We further assume that a decision is based on a combination of activation pertinent to each hypothesis, and we define this combination probabilistically as ΔP , where ΔP acts as a proxy to the “confidence” in either decision option (Jar A and Jar B). The greater this value, the higher the probability that a decision will be made. ΔP is defined as

$$\Delta P = P(A) - P(B), \quad (3)$$

where $P(A)$ is the probability of the source being Jar A, and $P(B)$ is the probability of the source being Jar B. Since $-1 < \Delta P < 1$, then $\Delta P = -1$ represents a strong belief that the beads were being drawn from Jar B in this simulation, and $\Delta P = 1$ represents a strong belief that the beads were being drawn from Jar A.

Three variables were considered in the following simulations: ΔP , the number of trials to decision (n), and the gain parameter (θ). There are two versions of the beads task, one where participants passively accumulate information pertinent to the decision to be made, and another where participants are required to indicate their confidence that the beads are being drawn from one jar or the other. The latter version of the beads task was critical to the model presented here, since—in the former version—a fourth “threshold” variable can explain differences between groups. In other words, ΔP remains constant between groups, and it is a lower activation threshold that needs to be met before a decision is made. As will be discussed, performance differences on the latter task, where participants stated their confidence, suggest that differences between groups are not attributable to a difference in threshold, but are due to differences in output. We did not, therefore, include a threshold variable.

SIMULATIONS

Standard Beads Task

In Dudley, John, Young, and Over (1997b), deluded participants selected significantly fewer beads than control groups (depressed and normal controls) for a 60:40 beads task (see Table 1). In another experiment, Dudley et al. (1997a) presented two versions of the beads task (with and without a memory aid) to participants (deluded and two control groups: depressed and matched) for both a 60:40 and an 85:15 beads task (see Table 2). For both scenarios, deluded patients selected fewer beads than controls.

In order to simulate these tasks, two models were constructed, identical except for the weighting (see Equation 1). For example, a red bead updated Node A (representing Jar A where there was a known ratio of red to blue beads of 60:40) by a factor of 0.6 and Node B by a factor of

0.4. Activity in both nodes was initially set at zero, thus $P(A) = P(B) = .5$. New information accumulated according to Equation 1, and the output function (Equation 2) gave $P(A)$ and $P(B)$, while Equation 3 gave ΔP . For the simulations, beads were randomly presented according to the desired ratio on each trial for 20 trials, and ΔP was calculated at each trial. This simulation was then run 100 times and the mean ΔP was calculated for each trial across all models. We argued that if ΔP increased more rapidly as the gain parameter increased, then the probability of an early decision would be similarly increased.

Referring to Figures 3 and 4, for the same value of ΔP the model shows that increasing the gain parameter increases the rate of ascent for ΔP . We thus argue that for a high-gain parameter, the probability of an early decision is increased and—consistent with the empirical data discussed in this section—that the model predicts participants will select fewer beads.

Change in Confidence in Light of Disconfirmatory Evidence

Garety et al. (1991), in Condition 2 of their paper, examined the effect of disconfirmatory evidence on their participants' strength of belief that the beads (in a standard beads task) were being drawn from one of the two jars. They presented participants with a predetermined series of beads (WWBWWWWBWBWBWBWBWB) and measured the change in confidence that the beads were being drawn from one of the two jars after Trial 4 (when the first “disconfirming” bead following three beads of the same color had been drawn). In their analysis, Garety et al. showed that deluded participants demonstrated a significantly greater change in confidence than participants in the control groups.

In order to simulate Garety et al.'s (1991) findings, we took ΔP as a proxy to subjective confidence. ΔP for Trial 4 was subtracted from ΔP for Trial 3, and this calculation was performed for five difference values of the gain parameter for one simulation.

Referring to Figure 5, as the gain parameter increases, the change in confidence between Trials 3 and 4 increases. Assuming that a high gain parameter is associated with delusional illness, this model mimics the data presented by Garety et al. (1991).

Discussion

In the simulations above, data from five research papers were examined, and the model successfully mimicked dif-

Table 1
Number of Draws Until Decision (With Standard Deviations)
for a 60:40 Beads Task (Dudley, John, Young, & Over, 1997a)

Participant Group	Content			
	Neutral		Salient	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Deluded	5.2	2.6	4.2	1.9
Depressed	7.5	3.4	7.1	3.6
Control	8.0	3.8	7.3	3.7

Table 2
Number of Draws Until Decision (With Standard Deviations)
for 60:40 and 85:15 Beads Tasks, With and Without a
Memory Aid (Dudley, John, Young, & Over, 1997b)

Participant Group	Task Condition							
	60:40				85:15			
	No Memory Aid		Memory Aid		No Memory Aid		Memory Aid	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Deluded	5.2	2.4	5.1	2.4	2.4	0.7	2.5	0.7
Depressed	8.4	2.9	7.9	2.7	4.1	1.6	4.5	1.6
Control	9.4	3.7	7.7	2.0	4.1	1.4	4.9	1.9

ferences in the behavioral data exhibited by delusional patients and controls. The characteristic of the network model that acted as the independent variable in the simulations was the gain parameter, and increasing this parameter changed the network's output from mimicking "normal" control data toward mimicking behavior on the beads task exhibited by schizophrenic patients. We further suggest that these data are consistent with the general view that increases in DA—and, therefore, the gain parameter—explain the behavioral manifestations associated with the positive symptoms of schizophrenia. As such, this article has extended those observations made by Cohen and Servan-Schreiber (1992) in a novel direction.

GENERAL DISCUSSION

An alternative account of the behavior that was observed while using the beads task with delusional patients is that patients verbalize their responses sooner than controls,

rather than exhibit a reasoning bias. Evidence that delusional states are associated with a reasoning bias rather than a "speak-too-soon" bias may be found in studies of a normal population (see, e.g., Linney, Peters, & Ayton, 1998; Sellen, Oaksford, & Gray, 2005). Sellen et al. differentiated between participants using the impulsive-nonconformity factor of the Oxford–Liverpool inventory of feelings and experiences (Mason, Claridge, & Jackson, 1995). Delusional states were observed in both affective and schizophrenic psychoses. The impulsive-nonconformity factor is argued to measure an underlying common disposition, under the assumption that affective and schizophrenic psychoses coexist along a continuum (Sellen et al., 2005). Thus, high scores on the impulsive-nonconformity factor may reflect a general psychosis proneness, or a proneness to delusions. Sellen et al. employed a conditional inference task (adapted from Cummins, 1995) to assess reasoning biases. They found that those who scored high on the impulsive-nonconformity factor were less able to take account of counterexamples to a conditional statement. This task was presented on computer; therefore, it was not consistent with a speak-too-soon explanation because it did not require participants to verbalize responses. Similarly, no differences were found on reaction-time data, suggesting that the inability to consider counterexamples was a function of a jump-to-conclusions reasoning bias rather than verbal impulsivity, per se.

Linney, Peters, and Ayton (1998) divided their sample from the normal population on the basis of their scores on Peters, Day, and Garety's (1996) delusion inventory. They conducted four tasks: a data-gathering task (the revised 2–4–6 problem; Gorman, Stafford, & Gorman, 1987), a hypothesis-testing task (the modified Wason selection task), a coin-tossing probability-judgment task, and a prior

Confidence by Learning Trial for Different Gain Parameters
for the 60:40 Beads Task

Figure 3. Simulated confidence levels (ΔP) for three values of the gain parameter on the 60:40 beads task ($n = 100$ for each value of the gain parameter). The vertical axis represents the simulated confidence level for the correct jar. Trial number on the horizontal axis represents beads drawn.

Confidence by Learning Trial for Different Gain Parameters
for the 85:15 Beads Task

Figure 4. Simulated confidence levels (ΔP) for different values of the gain parameter on the 85:15 beads task ($n = 100$ for each value of the gain parameter). The vertical axis represents the simulated confidence level for the correct jar. Trial number on the horizontal axis represents beads drawn.

probability task. Significant effects were found only on the data-gathering task and the probability-judgment task, leading Linney et al. (1998) to argue that delusion ideation is caused only by data-gathering abnormalities. The evidence from both studies involving normal populations (Sellen et al., 2005; Linney et al., 1998) demonstrate that different reasoning tasks—using different methodologies and measures of delusion proneness—result in a specific data-gathering bias. This is consistent with a jump-to-conclusions reasoning bias, not a quick-to-respond bias. In summary, given the robust evidence for the jump-to-conclusions reasoning bias in delusional patients, as well as converging evidence from normal populations and con-

sistent findings on different types of reasoning tasks, we argue that the jump-to-conclusions bias is a function of a reasoning deficit rather than any other explanation.

The beads task captures a generic aspect of real-world decision making, where a choice can be deferred at the cost of collecting additional information. For example, a farmer may face the choice of harvesting his or her crop or purchasing additional meteorological information that would assist in making that choice (see, e.g., Freixas & Kihlstrom, 1984; Gould, 1974). Another example would be the option for consumers who are shopping for a used car to purchase additional information from a third party on its mechanical circumstances (Akerlof, 1970). In the

Change in Confidence in Light of Disconfirmatory Evidence

Figure 5. Simulated predicted change in confidence [$\Delta P(4) - \Delta P(3)$] between Trials 3 and 4 in light of disconfirmatory evidence.

research discussed above—consistent with real-world decision making—differences between groups and within groups were observed. Both normal controls and patients showed variance around the mean. There are two ways of understanding this variability. Either it is due to random noise or it is due to systematic biases in cognition. The simulations presented in this article suggest the latter and have general implications for decision-making research.

Variations in the architecture supporting schizophrenia reside along a continuum (i.e., there are no discrete boundaries between the patient and the control; Claridge, 1985, 1997). Thus, extreme differences in DA function that underlie the differences between schizophrenics and normal populations may show scale invariance such that small variations in DA function within normal populations may underlie individual differences on the beads task. Furthermore, DA is implicated in decision making (Egelman, Person, & Montague, 1998; Platt & Glimcher, 1999), with evidence suggesting a relationship between DA function and the subjective utility of a prospective reward (Fiorillo, Tobler, & Schultz, 2003; Platt & Glimcher, 1999). Thus, if observations made in this paper do scale to variations in the normal population, then individual differences in DA function may underlie individual differences in decision making more generally.

Thus, two areas of further research are identified in this paper. First, little is known about how the brain represents uncertainty in decisions that involve reducing that uncertainty. The beads task has received little attention, despite growing interest in the neuroanatomical structures that are involved with general decision making. The previously presented model offers testable hypotheses on the relationship between the accumulation of hypothesis-specific information in the brain and the role of the mesolimbic DA system. Second, if variations in the architecture supporting schizophrenia reside along a continuum and psychometrics of the type used by Sellen et al. (2005) can identify such variation in normal populations, then the model presented here might suggest mechanisms that account for individual variations. Finally, although research has examined the relationship between clinical diagnosis and neuroanatomical structures that may correspond with observable behavior, definitions of schizophrenia subtypes remain imprecise. We argue that one means of defining schizophrenia may be through a more liberal use of computational modeling. Computational models are generally precise and usually not open to variations in interpretation. Moreover, models can successfully bring together a range of observations that include the neuroanatomical structures involved in the disease, together with observable behaviors.

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(Manuscript received June 15, 2005;
revision accepted for publication March 22, 2006.)