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Application of Stochastic Modeling to the Assessment of Group and Individual Differences in Cognitive Functioning

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This article begins with a guiding schema of relations among cognitive science, clinical science, and assessment technology. Emphasis is placed on stochastic modeling of cognitive processes. Basic models are adjusted so as to parsimoniously accommodate performance deviations occurring with psychopathology. Modified portions of models indicate functions affected by disorder, whereas portions remaining intact indicate spared functions. Findings from clinical cognitive science are applied to the individual case using Bayesian procedures. Methods are instantiated with respect to cognitive psychopathology of paranoid schizophrenia. The authors address observations and issues arising from this application, including integration of these methods with current assessment practices.

In the first edition of his *Handbook of Abnormal Psychology*, Hans J. Eysenck (1961) began his introduction with a quote from Roger Bacon (1214–1286): "Physicians must know that their science is impotent if they do not apply to it the power of mathematics." Eysenck and other contributors to this anthology went on to demonstrate the apparent power of then state-of-the-art multivariate methods in testing theoretical predictions about the symptomatological fabric and measurement of psychopathology. The theoretical formulations that were addressed comprised mainly conjectures regarding factor structures of signs and symptoms, and distributions across disorders of multivariate aggregate scores, such as discriminant-function variates. Few theoretical formulations took the form of quantitative logical-deductive systems, whose predictions were derived as closed-form formulae or proven propositions (e.g., Braithwaite, 1968; although cf. Furneaux, 1961). Apropos of empirical testing, over and against theory construction, the above volume and other works of the time represented a watershed, ushering in application of advanced data-analytic methods to clinical science and assessment.

It seems that theory construction in this field may be poised for a similar transition toward increased formality. Stochastic modeling of cognitive processes stands at the ready for operationalizing oft-used explanatory constructs such as processing capacity, efficiency of capacity deployment, parallel–serial architecture of processing systems, and cognitive-task load.

Below, we describe a proposed strategy for appropriating formal cognitive science in the form of stochastic modeling. The claimed advantages of this strategy are considered in light of past treatises on challenges to valid inferences in clinical science. Stochastic modeling of cognitive psychopathology then is instantiated in the case of paranoid schizophrenia. This development exemplifies a progression of steps from basic cognitive science through its clinical-science implementation, to direct implications for assessment of clinically significant functions. Lastly, we expound issues and observations stemming from this application.

Formal Process Models of Cognitive Performance and Challenges to Valid Inferences in Clinical Science

Overview of Proposed Formal-Modeling Strategy

The current perspective on relations between cognitive science and clinical assessment is presented in Figure 1. The route from basic cognitive science to clinical assessment ideally encompasses seamless transitions from one sphere of investigative activity to the next.

The procedure begins with a formal account of cognitive-task performance. Modeled response parameters typically entail one or a combination of the following: speed, accuracy, judgment of item

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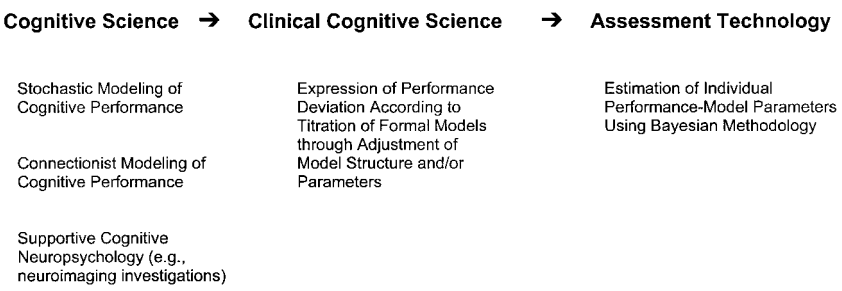

Figure 1. Linkages among domains of research.

properties (e.g., attractiveness or inter-item similarity), or response categories (e.g., item-recall or recognition in memory studies).

Stochastic models take account of the inevitable uncertainty intrinsic to prediction. Doob (1953) stated, " . . . a stochastic model is the mathematical abstraction of an empirical process whose development is governed by probabilistic laws" (p. v). Accordingly, predictions take the form of probabilistic statements, and stochastic distributions become the raw materials of modeling, along with their quantiles, moments, and other distributional properties. Volumes on the topic addressed to behavioral-science audiences include Townsend and Ashby (1983), Luce (1986), and Wickens (1982). More general treatments can be found in Johnson, Kotz, and Balakrishnan (1994, 1995). Presentations of stochastic-process models tailored to the context of clinical assessment have been presented by Carter, Neufeld, and Benn (1998), Neufeld (1998), and Neufeld, Vollick, and Highgate (1993).

Such modeling addresses key attributes of the processing system. Included is its architecture or organization with respect to task or task-component transaction. In performing a memory-search task, for example, when encoding a presented alphanumeric item so as to ascertain its presence in a memorized set of items, features of the presented item such as lines, intersection, and curves of the letter or digit may be implemented serially or in parallel. Certain structural variations may occur within an overall parallel-processing design (e.g., Townsend & Nozawa, 1995), whereas still other architectures may contain both serial and parallel elements (e.g., Schweikert, Giorgini, & Dzhafarov, 2000; Townsend, 1984; Townsend & Ashby, 1983). Additional aspects of model architecture pertain to the temporal arrangement of processing operations, such as encoding, scanning of memorized items, and response mechanisms (e.g., Townsend, 1984).

Other important processing-system attributes comprise parameters incorporated by the system architecture. Included are parameters aligned with the magnitude of a given process, such as the number of constituent operations (subprocesses) involved in item encoding (corresponding possibly to the number of item features that are implemented). Included as well may be a parameter expressing the speed of process transaction, operationalized as the rate of subprocess completions per unit of time. Note that model attributes may be regarded as random variables, changing probabilistically over observational units such as individual participants. Thus, response parameters (e.g., latency of process-completion) are deemed to be random; but now so are features of the posited model of task-performance on individual trials. That is to say, probabilistic behavior of dependent variables, such as completion latency, potentially is governed by model features which themselves are probabilistic in nature. For example, the serial-versus-parallel strategy of negotiating encoding subprocesses may vary across individuals, with certain probabilities of one or the other being in place for any one participant (i.e., "compound processing models"; Townsend & Ashby, 1983).

Similarly, parameters of the processing system, such as the rate of subprocess completion (processing capacity), may be designated as random variables. As such, their specific values would be distributed stochastically across observational units (e.g., Morrison, 1979). Consequently, performance data from a given sample in toto results from a mixture of parameter values that are randomly distributed across participants. Expanded models accommodating such additional sources of variation are known as *mixture models.* Mixture models of this nature are emphasized in the developments presented below.

With the formal model of normal task performance in hand, performance aberrations of clinical samples are confronted. Models of normal performance now are titrated so as to generate observed patterns of deviation. Titration entails the required adjustment of architectural structure, and/or parameter values, or both.[1] For parsimony, only the minimal adjustment necessary to achieve the altered performance is sought.

Architectural aspects, or parameter values of the normal model that are modified, are indicative of affected functions, whereas parts that remain intact are indicative of functions that are spared. In our experience, changes in the theoretical architecture of the processing system by and large are not indicated (Carter & Neufeld, 1999; Neufeld & Broga, 1981). Rather, changes in values of model parameters are sufficient (Neufeld, Vollick, & Highgate, 1993). In cases of mixture models, where these parameters are designated as random variables, it is sufficient now simply to shift their theoretical distributions (Neufeld & Williamson, 1996).

Such models lend themselves to Bayesian estimation of individual parameter values (Batchelder, 1998; Berger, 1985; see Appendix Equation A1 for Bayes's theorem). This option therefore represents a nexus for clinical science and clinical assessment technology. Within the Bayesian framework, a distribution of parameter values becomes a Bayesian prior. It affords a context in which data from an individual client can be placed.

Amalgamation of the prior distribution of parameter values, with the obtained performance sample, leads to the "Bayesian

[1] The titration metaphor is used liberally. More accurate would be the modification of the normal model so as to assay its own composition, rather than adjusting it to assay the composition of deviant performance.

posterior distribution" of parameter values, a conditional distribution given the obtained performance sample. The posterior distribution potentially affords markedly increased accuracy in estimation of performance-model parameters for the person at hand.

The above layout has focused on stochastic models. Confluence of results from this level of cognitive-science analysis with complementary levels enlarges the picture of cognitive operations and their abnormalities in psychopathology. Connectionist modeling comprises one such complementary level (see Siegle & Hasselmo, 2002). Computer simulation of the operation of networks of processing units, or unit modules, can serve as a metaphor for connectionist aspects of brain functioning, in principle disclosing information unique to this level of analysis (Marr, 1982).

Another complementary level of investigation is the neurophysiological, notably neuroimaging studies of brain activation accompanying cognitive activity. As with connectionist and stochastic modeling, functional magnetic resonance imaging (fMRI) of brain activation and stochastic modeling may be mutually informative. Stochastic modeling of cognitive performance stands to elucidate the functional significance of fMRI brain-activation patterns. Such patterns may become aligned with a stringently specified model architecture and/or parameter values. Clinical significance, in turn, is endowed to the degree that titrated features (see above) can be identified with measured deviations in activation (Carter, Neufeld, & Benn, 1998).

Note that the current framework points to a certain avenue of evaluating validity of models of normal cognitive performance. Models that lend themselves to adjustments accommodating clinical performance deviations are preferable to those that are strained. In this way, there is a potentially productive give-and-take between cognitive science and clinical cognitive science. Clinical samples affording marked departures in performance from the norm may provide a unique and useful opportunity to test model robustness (see Busemeyer & Wang's, 2000, exposition of generalization testing as a method of model evaluation).

Challenges to Valid Inferences in Clinical Science

The current investigative strategy can be examined in light of three classic challenges to valid inferences in clinical science. One involves Maher's (1974) Bull-in-a-Worcester-China-Shop Scenario; the second pertains to Chapman and Chapman's (1973) Differential-Deficit Psychometric-Artifact Postulate; and the third entails Meehl's (1978) Cowardly Conjecture Syndrome.

Maher observed that clinically disturbed populations fare poorly on nearly any measure of cognitive performance. He noted that much clinical science resembles one's releasing a bull into a Worcester China shop. The hypothesis is that breakage will occur. Accordingly, no matter where one looks, confirming evidence is available, especially if the control condition involves releasing a mouse into a similar test environment. Maher called for studies where performance on commensurately challenging control tasks indicated that associated functions did not decline with the investigated psychopathology (demonstration of differential deficit). A theoretical integration of the stream of reported performance deficits—a "parsimonious theory of the bull," as it were—was also needed to replace the exhaustive reporting of the bull's devastation.

Chapman and Chapman's (1973, 1978) psychometric-artifact postulate impinges on a subset of observations put forth by Maher (1974). Differential deficit on measures of alternate functions should transcend differences between the respective measures in their psychometric sensitivity to their addressed functions.

Meehl's (1978) Cowardly-Conjecture Syndrome indicts the customary practice of zero intervariable relation null-hypothesis testing, whose rejection is inevitable with sufficient statistical power. Progress in "soft psychology" requires theory that can afford Popperian bold conjectures. Such conjectures furnish falsifiability: Predicted events are predicated on theory verisimilitude, and nonfulfilment either seriously jeopardizes or decimates the theory. Quantitative, "numerical-point predictions" exemplify what is needed.

The currently proposed approach engages these challenges, as follows. Differential deficit potentially is established within a formal theoretical framework around which the multiplicity of findings of performance-decline can be arrayed (Maher, 1974). In addition, the configuration of performance patterns across experimental conditions and diagnostic groups, prescribed by precise model titration, defies tenable explanation as an artifact of group-discriminating psychometric-precision acting in concert with generalized deficit (Chapman & Chapman, 1978; cf. Carter & Neufeld, 1999; Knight & Silverstein, 2001).

Finally, predictions are of a quantitative format and refer to combinations of values comprising the above patterns of performance across conditions and groups (Meehl, 1978). Stringent tests of theoretical predictions are available and reveal when competing models can coexist and when one or the others must be discounted (Schweikert et al., 2000; Townsend & Nozawa, 1995, 1997).

A formal theoretical infrastructure seems entirely in keeping with the formulation of bold predictions. Assumptions are constrained to be explicit, and predictions are derived from precise rules of transformation within the logical-deductive system, be it mathematical, computer-language-based, or symbolic logic (cf., Staddon, 1984).

Interestingly, the boldness of conjecture extolled above can be cast into a Bayesian formulation. Doing so provides a relevant angle on the composition of strong theory–evidence relations making for predictive risk. Consider Bayes's theorem, stated in Appendix Equation A1. The denominator $Pr(B)$ is equal to $[Pr(A)\,Pr(B|A) + Pr(not\ A)(Pr(B|not\ A)]$. Let A represent a proposed theory, and B correspond to empirical observation. If the probability of the observation essentially vanishes apart from the proposed theory A, then by Equation A1, $Pr(A|B)$ necessarily approaches 1.0. It must do so even if $B|A$ seems remote, with an associated small subjective probability.

Reversing theory and observation in Bayes's theorem, let A now stand for observation and B the proposed theory. If the probability of the theory being tenable in the absence of the predicted observation $Pr(B|not\ A)$ is negligible, again by Equation A1 the observation is virtually inevitable if the theory is tenable, or $Pr(A|B)$ approaches 1.0.

The Bayesian dissection of strong theory–evidence relations in the first instance (as noted above) indicates that tenability of the designated observation is unique to the proposed theory. The second instance highlights the role of falsifiability. We would contend that the strategy depicted in Figure 1 should edge clinical science closer to the ideals expressed here. Such credentials for the

depicted strategy are claimed largely in the name of the decidedly formal theoretical apparatus by which quantitative cognitive science proceeds. Unveiled are experimental manipulations and predicted results that otherwise seem difficult or impossible to contrive. Second, predictions can be diagnostic of competing formal theoretical models (e.g., Busemeyer & Townsend, 1993; Townsend & Nozawa, 1995, 1997).

From Cognitive Science to Clinical Assessment: Stochastic Modeling of Stimulus-Encoding Latency in Paranoid Schizophrenia

The tack to implementing cognitive science drafted in Figure 1 can be illustrated with an empirical example. We begin by describing a paradigm used to examine the comparative performance of individuals with paranoid schizophrenia on a variant of a memory-search task (Sternberg, 1975). Relevant developments in cognitive science are then brought to bear in deciphering observed performance deficits. The latter take the form of delayed encoding of presenting stimulation into a task-facilitative format. A theoretical architecture of encoding and associated parameters are described; parameters are deemed to vary randomly across participants, their distributions being altered with psychopathology, and by task conditions.

We then mediate results from these analyses to individual samples of task performance, using Bayesian procedures. The individualized parameter estimates are used to construct tailor-made encoding-latency distributions. Finally, we consider clinical significance of the customized distributions.

Paradigm and Findings

A study whose results are representative of those for schizophrenia performance on the above memory-search and related tasks is that of Highgate and Neufeld (1986). Memory-search tasks require the comparison of a presented item, often a digit or letter (probe item), with a set of previously memorized items (memory set).

Briefly, individuals are asked to indicate as quickly and accurately as possible if the probe item, or one of its properties (e.g., its real-life size), is present among the previously memorized set of items. Memory-set size varies from trial to trial, but remains at sub-span values. Similarly, presence versus absence of the probe, or its property (positive vs. negative trials), vary independently from trial to trial.

Processes putatively tapped by this task include, first, encoding of the probe item into a cognitive format facilitating comparison to members of the memory set, or their properties; second, the memorial comparisons themselves; and third, response processes. Results from Highgate and Neufeld's (1986) study are representative in terms of apparent integrity of memory search amid deficient probe-item encoding.

Two of the groups participating in this study were paranoid schizophrenia patients and nonpatient controls (see, e.g., Nicholson & Neufeld, 1993). Participants completed a relatively demanding memory-scanning task. They were asked to indicate whether the presented probe or target item (either an animal or an object) resembled a memory-set item with respect to its real-life size (or overall magnitude; see Paivio, 1975). Memory-set sizes ranged from one to four. One half of the participants in each group were presented with similar-sized drawings of target items, whereas the other half were presented with names of the items. Thus, encoding requirements presumably were greater in the latter case, inasmuch as the initial verbal representation of the target item would require referral to the imagery system to access the necessary size properties (see also George & Neufeld, 1984, for elaboration of Paivio's, 1986, dual-coding theory as implemented in research on schizophrenia). In each condition, encoding demands exceeded considerably the mere extraction of presenting physical features, as might be sufficient in simple template matching.

Memory scanning also was considered to be relatively demanding. It required comparison between the size properties of the target item and the respective memory set items, and, additionally, the setting of these contrasts against subjective criteria of similarity in size (see Hockley & Murdock, 1987; Wright, 1977). Memory-set items were spaced such that their means were at least two standard deviations of their Thurstonian discriminal-difference size-distributions apart (essentially, at least two standard deviations of the distribution difference scores, between their normative size ratings; detailed in Highgate & Neufeld, 1986). Responses were identified as correct or incorrect according to whether they conformed to the following determination of negative and positive trials. Negative trials, meaning that the target item's real-life size did not resemble that of a memory set item, occurred as follows. The target's mean normative size rating was at least one standard deviation of the Thurstonian discriminal-difference distribution from the normative mean rating of each item in the trial's memory set. Positive trials were those where the target mean and the mean of a memory-set item by and large were the same. Participants were acquainted with task requirements and the nature of correct responding with the aid of practice trials; a similar number of such trials was required for each group.

Furthermore, it was ascertained that the normative ratings of item sizes (Paivio, 1975) were similarly applicable to all groups. Other precautions involve absence of speed–accuracy trade-offs and provision for extraneous demographic and clinical variables (e.g., age, education, IQ, hospitalization, medication).

Findings indicated that encoding of presented items took longer under the higher encoding-load condition (probe-item name) for both groups. Furthermore, mean encoding completion was delayed by a similar amount under each encoding-load condition for the paranoid schizophrenia participants, relative to their nonpatient counterparts. Memory-scanning and response processes, on the other hand, evidently were unimpaired (as detailed in Highgate & Neufeld, 1986; and Neufeld et al., 1993). Stimulus encoding, therefore, is the focus of analysis.

Convergent evidence for this source of deficit comes from studies using complementary paradigms (reviewed in Neufeld, 1991a; Neufeld & Williamson, 1996). Convergent evidence for the modeled composition of this deficit (see below) emanates from investigations using divergent paradigms and forms of analysis. Included are studies addressed expressly to the architecture and parameters of item encoding (Vollick, 1994; Vollick & Neufeld, 2002); memory traces pertaining to multidimensional stimuli (Carter & Neufeld, 1999); and connectionist-computational aspects of content and latency of item categorization (Carter, 2000; Carter & Neufeld, 2002).

Processing Model and Parameter Titration

To broach the formal modeling of findings, quantitative cognitive science potentially elucidating the nature of delayed encoding was brought to bear, notably developments of James Townsend and colleagues (e.g., Townsend & Ashby, 1983). A single architecture tenably expressed the encoding process for both high- and low-encoding loads and for each diagnostic group. Furthermore, the speed of transacting components of the encoding process was intact. The parameter whose adjustment most parsimoniously accommodated the observed configuration of encoding latencies was one depicting the extent of encoding. This parameter corresponded to the number of elements or subprocesses composing the encoding process.

A relevant angle on this parameter is afforded by an automotive analogy. An automobile's internal combustion engine may be in good working order. Traveling a required distance, however, may take longer because the transmission may be substandard or damaged. Consequently, more revolutions (themselves proceeding at the same rate per unit time) are required per measure of distance covered. Paralleling the analogy, in turn, processing capacity evidently has been spared, but efficiency of its deployment is impaired.

Behavioral correlates of this increased parameter value have been tendered elsewhere (e.g., Neufeld & Williamson, 1996). Among other possibilities included are "disengagement deficit," whereby stimulus features which although relevant to the given process are unnecessary (Cromwell & Dokecki, 1968); and "priming of the system" (e.g., Russell & Knight, 1977) to perform the processing requirements, resulting in an added preliminary set of subprocesses.

Note that results can be expressed in terms of a mixture model (as noted above). This model casts the observed encoding-load and psychopathology-related effects specifically as a shift in the distribution of parameter values (Neufeld & Williamson, 1996). The distribution of parameter values expressing the number of encoding subprocesses is moved upward under the higher encoding-load condition; a similar upward movement accompanies psychopathology (not unlike an endogenously increased load).

Model Specifics

The model architecture expressing the encoding process is known as the *Erlang distribution.* It depicts a given instance of encoding as a series of subprocesses, k' in number,[2] proceeding at rate v per unit time (seconds, in the present case). This idealized mechanism naturally is affected by ambient influences both endogenous and exogenous to the processing system. Consequently, the encoding latency is distributed stochastically over time t, this distribution's mean being k'/v and its variance being k'/v^2. Its probability-density function, conveying the relative frequency of process completion over time, is

$$(vt)^{k'-1}/(k'-1)!\ ve^{-vt}. \tag{1}$$

The Erlang distribution is displayed in Figure 2. The figure shows four sets of parameter values, with associated changes in the profile of encoding-process completion. In the present modeling approach, each pair of parameter values is identified with an individual research participant.

Within the mixture-model framework, then, values of k' and v themselves are distributed randomly over observational units or participants (mixing distributions).[3] The dispersion of the parameter v is regarded as gamma, a distribution with parameters k and r. The associated probability density function, conveying the relative frequency of individuals having a particular value of v under the prevailing values of k and r, is

$$(rv)^{k-1}/\Gamma(k)\ re^{-rv}. \tag{2}$$

In this formula, Γ is the gamma function, a continuous-variable analogue of the factorial, where k is an integer, $\Gamma(k) = (k-1)!$ (see, e.g., Beyer, 1984).

Justification for gamma as the chosen mixing distribution for v has been addressed elsewhere (Neufeld & Carter, 2000). For example, depending on the parameter k, it can approximate a normal distribution or become an exponential distribution (see, e.g., Neufeld, 1998). Also, it serves effectively in the present case, as compared with competitors (e.g., Jeffrey's uninformed prior; see Berger, 1985).

The parameter r has been aligned with stress-related detraction from cognitive performance, whereas k has been used to express competence-related strengthening of performance. Analytical and empirical support for these interpretations has been presented in other sources (Neufeld, 1991b, 1994, 1996; Neufeld & Carter, 2000). Of importance here is the observation that values of r and k tenably are constant across the present combinations of encoding conditions and diagnostic groups (Neufeld & Williamson, 1996; Vollick & Neufeld, 2002). Moment-fitting parameter estimation (e.g., Townsend, 1984) yielded a value of .03735 for r, and 2.5044 for k. For clarity of interpretation, these values were estimated using correct response latencies occurring under negative trials (Neufeld & Gardner, 1990; Neufeld et al., 1993). The resulting mixing distribution of v applicable to all four factorial combinations of encoding load and diagnostic status is presented in Figure 3.

The subprocess-number parameter k' too is deemed to vary randomly over individuals. It is held to be dispersed according to a Poisson distribution (see, e.g., Evans, Hastings, & Peacock, 2000), the latter entailing one parameter m. This "intensity" parameter is regarded as indicating propensity toward encoding-

[2] Although the encoding subprocesses can be taken as transpiring in serial, parallel processing models whose distributions mimic those of serial models are viable. Rigorous exposition of parallel–serial model mimicking is presented by Townsend and Ashby (1983, ch. 14; see also Townsend, 1990). Parallel architectures potentially applicable to the present study are discussed in Neufeld et al. (1993).

[3] In the present numerical development, results from Highgate and Neufeld (1986) are appropriated, as follows. Empirical moments (means and variances) guiding model construction (e.g., Neufeld et al., 1993) were computed across trials, within the respective participants. These amounts were then averaged over participants nested within encoding-load and diagnostic classification (homogeneous-participant methodology, cf. Townsend, 1984). In effect, the current exposition allows aggregates of trials with shared parameter values to stand for individuals. Task-performance samples, in turn, correspond to trials within such aggregates. Note that this qualification does not diminish the thrust of the present developments in any way.

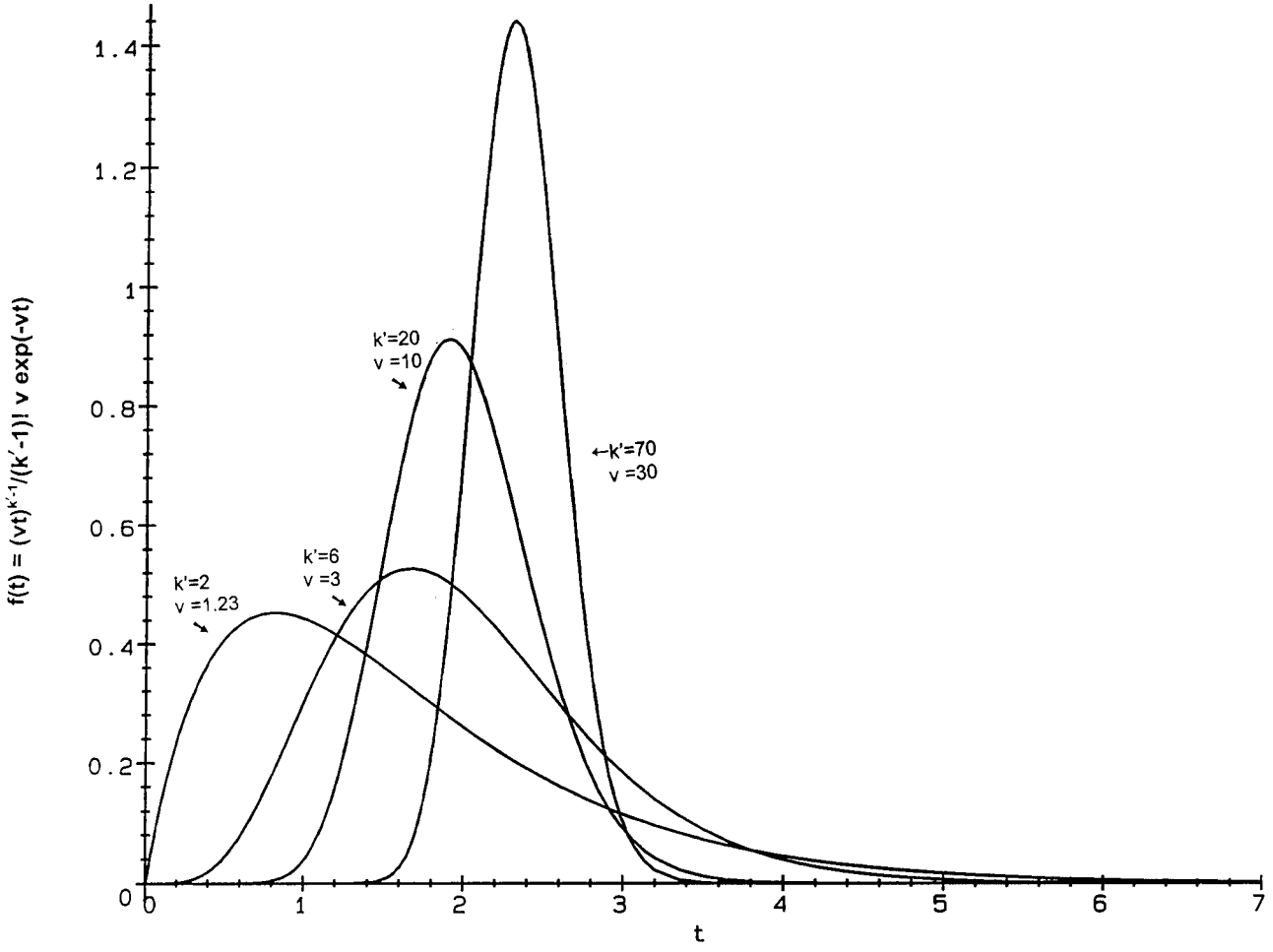

Figure 2. Erlang distributions, with hypothetical individual differences in values for parameters k' and v.

subprocess recruitment on probe-item engagement.[4] The mean of the Poisson distribution of k' is m, as is its variance. This parameter takes on a base value m' for nonpatients under the low-encoding-load condition. The value is increased by an amount h for both paranoid schizophrenia and control participants under the high-encoding-load condition. It is incremented as well by a value g with paranoid-schizophrenia status, whether under the high- or low-encoding-load condition.

The estimated value of m' was .00001. This value indicates that for the nonpatients, little if any processing was involved in encoding the clear and simple drawings of probe items. The estimated value of h was 19.73900 and that of g was 70.0. Inferred values of m under the low- and high-encoding loads for the nonpatients therefore were .00001 and 19.73901, respectively. Corresponding values for the paranoid schizophrenia participants were 70.00001 and 89.73901. Modeled expression of disorder effects thus takes the form of the above additive incrementation in the intensity parameter of the Poisson distribution.

Recall that the subprocess number k' is governed by m stochastically and thus varies randomly over observational units according to a "Poisson process." The probability under the prevailing value of m of any integer k' from 0 to infinity being the operational value in any one case is given by

$$m^{k'}/k'!\ e^{-m}.$$

The mean value of k' nevertheless is m, as is its variance. Theoretical effects of heightened encoding load, and of disorder, therefore are such as to shift the distribution's central tendency and

[4] This interpretation can be expanded on in terms of m's binomial-distribution counterpart np (where n is the number of trials, and p is the probability of success on any trial), n now being very large and p being very small. In keeping with the interpretation, let n represent the pool of encoding subprocesses *potentially* transacting the encoding process for any participant. The term p thus indicates the probability of a latent subprocess being engaged in the encoding operation. Allow n in turn to equal $m' + h + g + x$, where the new term x is simply some very large value. The term p above now approximates m/n, where m continues to have a base value of m', with the addition of h and/or g depending on the presence of exogenous (experimentally induced) and/or endogenous (paranoid-schizophrenia related) sources of elevation in encoding subprocesses. The terms h and g thus convey *increases* in the probability of involving in the encoding operation any one of the subprocesses in the pool of n. It is the combination of n and p into the product $np \approx m$ that is then brought to bear on the probability of any particular value of the subprocess variable k'. In fact, as n approaches the limiting value of infinity, and np approaches the limiting value of m, the binomial and Poisson distributions converge.

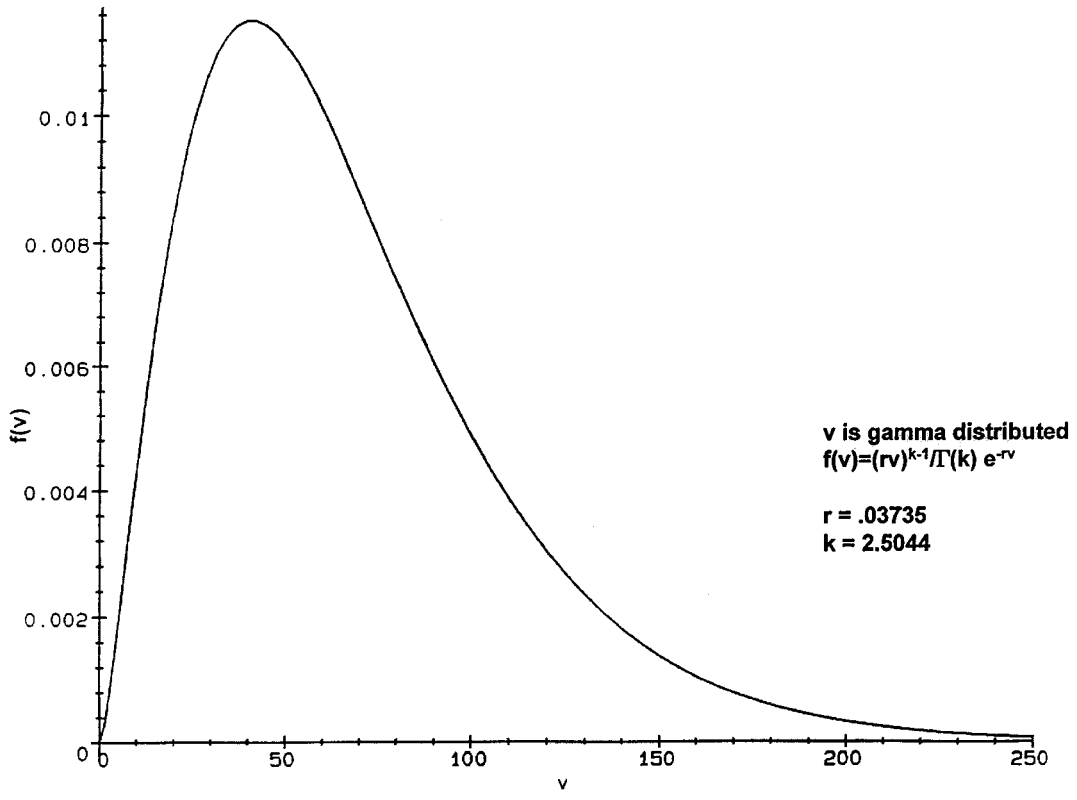

Figure 3. Mixing distribution of v, common to all combinations of encoding load and diagnostic status.

dispersion upwards by the prescribed amounts. These effects are expressed in the mixing distributions of k', illustrated in Figure 4 for the respective values of m (shown above).

The overall design of the resulting mixture model is portrayed in Figure 5. The gamma distribution of v and the Poisson distributions of k' are the parameter mixing distributions of the mixture model, corresponding to those portrayed in Figures 3 and 4. The Erlang distribution of latencies t to which the parameters v and k' apply in the individual case (exemplified in Figure 2) is the mixture model's base distribution. With this distributional arrangement in hand, the spread of parameter values for a particular participant in principle can be substantially narrowed (or individualized). The increased accuracy is achievable thanks to Bayes's theorem, as follows.

Parameter Estimation in the Individual Case

Samples of encoding latency obtained from individual participants are now brought to bear on parameter estimation. These samples are integrated with the mixing, or prior, distributions, including the Poisson distributions expressing heightened encoding demands and paranoid-schizophrenia cognitive psychopathology. Four samples of performance are considered, one from each diagnostic group under each encoding-load condition. Each sample, in turn, is integrated with the respective prior distributions associated with the diagnostic-group, encoding-load divisions. This Bayesian integration of information on parameter values, supplied by the prior distribution, with obtained performance data, then produces the potentially more exact Bayesian posterior distribution of parameter values at the individual-participant level of analysis.

The Bayesian prior in principle accommodates performance samples of its membership at large. The posterior distribution, in contrast, represents a mixing distribution that is applicable to a subset of individuals with certain performance values, to which the participant at hand belongs. The array of posterior mixing distributions resulting from the combination of performance samples with the differing priors reveals an influence of each source of information.

The narrowed posterior parameter mixing distributions, in turn, are used to construct those of encoding latencies t applicable to the individual's sample of data. Certain distribution properties are then considered with respect to both potential clinical significance and findings from other levels of study (e.g., those involving fMRI).

Performance samples. A sample of encoding-latency performance was obtained for each of the diagnostic-group, encoding-load conditions. An initial pool of 360 model-relevant data points were available in each case: nine negative trials under each of four memory-set sizes performed by each of 10 individuals. For the present purposes of tractable illustration, four values for a given individual were extracted from each group–load combination, one under each memory-set size. These values were adjusted to throw encoding into relief by subtracting estimated times for memory scanning and response-movement time (Vollick, 1994; Vollick &

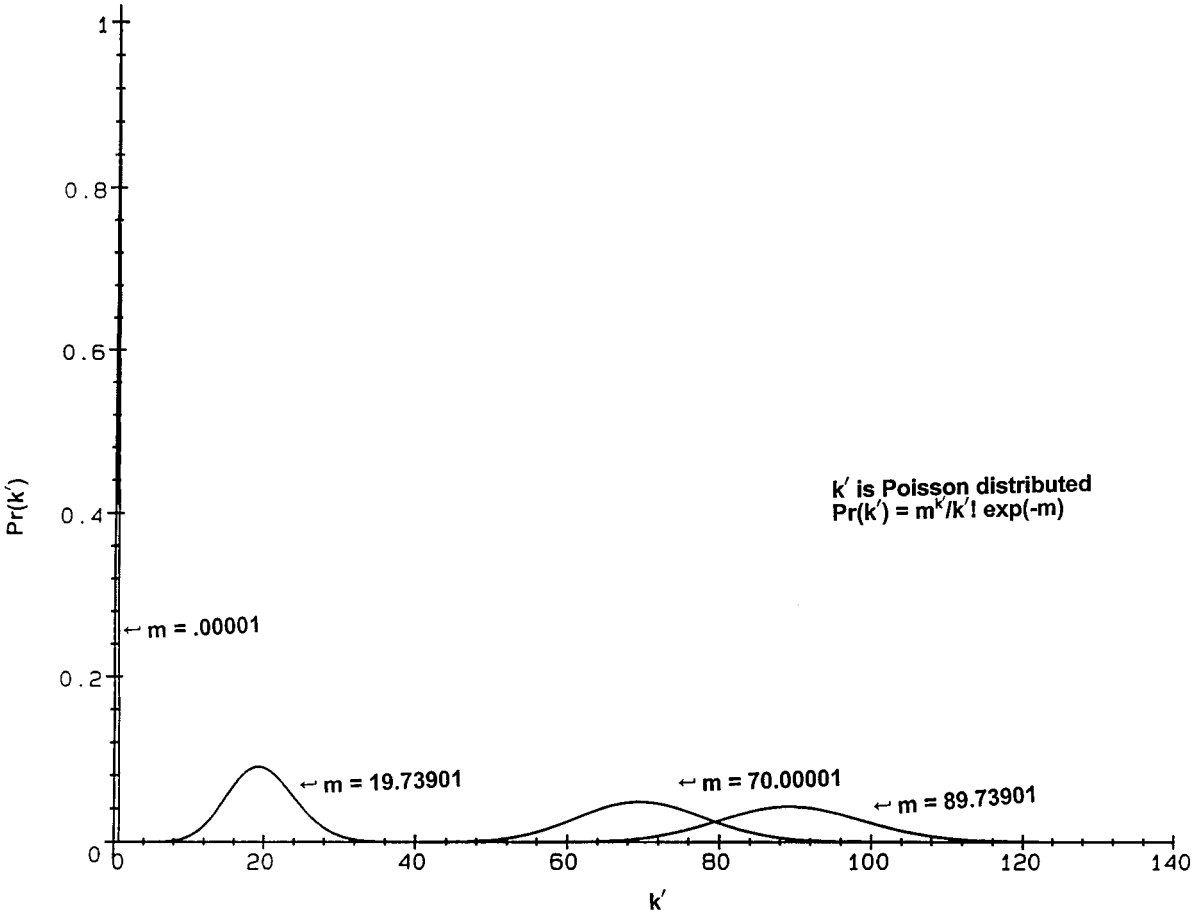

Figure 4. Mixing distributions of k', varying with encoding load and diagnostic status.

Neufeld, 2002). The adjusted sets of values $\{t_1, t_2, t_3, t_4\}$ are presented in Table 1.

Posterior distributions of parameter values. The means of the posterior distributions of v and k', $E(v|\{t_1, t_2, t_3, t_4\})$ and $E(k'|\{t_1, t_2, t_3, t_4\})$, and the standard deviations of these distributions are presented in Table 1 (formulae underlying figures and tables are presented in the Appendix). These values can be compared with the mean value of the prior distribution of v, which is 67.05, and with its standard deviation of 42.37. In each instance, the posterior distribution for v is substantially thinner than the prior distribution for v.

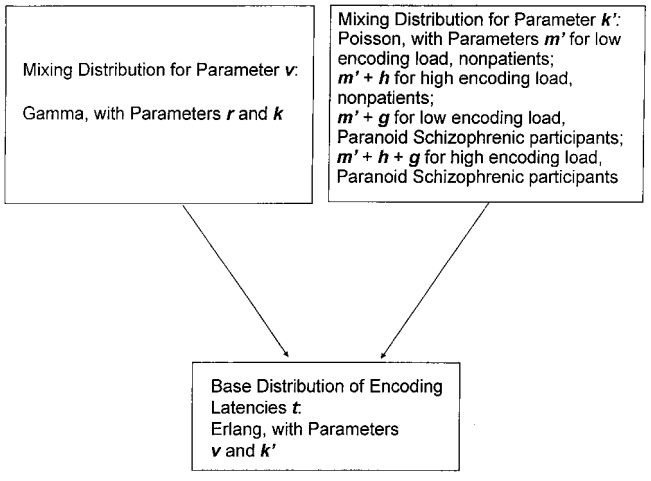

Figure 5. Design of parameter-mixture model.

The standard deviations of the posterior distributions of k', presented in Table 1, also tend to be less than those of the priors (recall that the variance of the prior is m). An exception occurs in the case of low encoding for the nonpatients. In the three instances where posterior distributions are tractable for this combination, the performance sample compels both a higher mean and standard deviation of the posterior distribution of k' than those of the prior. Interestingly, the posterior distribution specifies a single subprocess for each of the calculable posteriors. This number is the minimum required for tenability of the base distribution. As might be expected where k' is 1.0, the resulting posterior encoding-latency distribution evinces an exponential-like shape (see the distribution for $m = .00001$ of Figure 8).

One of the more dramatic instances of changes in both means and variances of the posterior parameter distributions from those of the priors occurs where $m = 70.00001$ and the performance

Table 1
Effects of Bayesian Priors and Individual Performance Samples on Estimates of Individual Encoding-Latency Distribution Parameters

		Representative individual-participant latency sets			
		Paranoid schizophrenia		Control	
Classification	Bayesian-posterior estimate	Low-encoding load {2.177, 3.184, 2.411, 2.498}	High-encoding load {3.476, 7.150, 9.00, 4.238}	Low-encoding load {1.037, 0.554, 0.191, 6.390}	High-encoding load {1.137, 3.204, 1.371, 2.207}
Paranoid schizophrenia					
Low-encoding load[a]	$E(v\|\{\cdot\})$	27.4835	9.4667	3.1077	27.0505
	$S.D.(v\|\{\cdot\})$	3.56	1.3628	1.0760	3.9857
	$E(k'\|\{\cdot\})$	70.1945	55.9255	5.7519	53.1798
	$S.D.(k'\|\{\cdot\})$	8.1000	7.2208	1.8110	7.0285
High-encoding load[b]	$E(v\|\{\cdot\})$	34.7436	11.9826	3.5487	34.0506
	$S.D.(v\|\{\cdot\})$	4.0222	1.5396	1.1795	4.5029
	$E(k'\|\{\cdot\})$	88.9026	70.6540	6.6571	67.1036
	$S.D.(k'\|\{\cdot\})$	9.2214	8.1744	2.0100	7.9548
Control					
Low-encoding load[c]	$E(v\|\{\cdot\})$	0.6325	—	0.7923	0.8175
	$S.D.(v\|\{\cdot\})$	0.2463	—	0.8510	0.3206
	$E(k'\|\{\cdot\})$	1.001	—	1.0000	1.0000
	$S.D.(k'\|\{\cdot\})$	0.0085	—	0.0017	0.0074
High-encoding load[d]	$E(v\|\{\cdot\})$	8.9426	3.1751	1.8179	9.1387
	$S.D.(v\|\{\cdot\})$	1.9280	0.7426	0.7255	2.1755
	$E(k'\|\{\cdot\})$	22.4176	18.3467	3.1049	17.5516
	$S.D.(k'\|\{\cdot\})$	4.3499	3.8668	1.1332	3.7660

Note. $\cdot = \{t_1, t_2, \ldots, t_N\}$, where in this table $N = 4$. Dashes indicate computations could not proceed, as $ujd\{t_1, t_2, \ldots, t_N\}$, under the specified values of r, k, and m essentially is 0.0.
[a] Parameters of prior distribution: $r = .03735$; $k = 2.5044$; $m = 70.00001$. [b] Parameters of prior distribution: $r = .03735$; $k = 2.5044$; $m = 89.73901$. [c] Parameters of prior distribution: $r = .03735$; $k = 2.5044$; $m = .00001$. [d] Parameters of prior distribution: $r = .03735$; $k = 2.5044$; $m = 19.73901$.

sample is {1.037, 0.554, 0.191, 6.390}. The prior and posterior distributions of v are presented in Figure 6 and those for k' are presented in Figure 7.

Note that posterior distributions of parameter values presented in Table 1 are constrained by the modeled effects of encoding load and of disorder. Specifically, the column under each performance sample displays an increase in the posterior mean k', that is, $E(k'|[\{\cdot\}])$ attendant to both these factors. They are constrained as well by the performance sample in the following way. To comply with the general level of the performance latencies, the above increases in the posterior distributions of k' are accompanied by increases in the posterior distributions of v. (Recall that the mean base-distribution latency is k'/v.)

Posterior latency distributions. The overall slenderized posterior mixing distributions can be used to construct posterior encoding-latency distributions, tailored to the prevailing performance sample. Such distributions are exemplified for the performance sample obtained from the selected paranoid schizophrenia participant performing under the low-encoding-load condition (i.e., {2.177, 3.184, 2.411, 2.498}).

This data array is implemented to compute the posterior latency distribution under each of the four prior distributions specified in Table 1. Doing so is revelatory in the following way. It indicates the effect of disorder and encoding load on the extrapolated encoding-latency base distribution. The same performance sample, in other words, is interpreted in different ways, depending on the putative group membership of the individual.

Taken into account by the posterior distribution, then, is whether the performance sample emanated from an intact or psychopathology-perturbed system and whether it occurred under low- or high-encoding demands. In this sense, the posterior latency distribution adjusts for the context of performance, as registered in initial titration of the Bayesian prior. Certain parameter values, for example, may be highly likely according to the performance sample, but they may not be sustainable when considering their prior probabilities, or densities, in the contexts from which they emanate (see Appendix Equation A6 and related commentary).

The posterior latency distributions for the above performance sample, under the respective priors, are displayed in Figures 8 and 9. Marked variation in the shapes of these distributions is apparent. Their means and standard deviations are presented in Table 2. For contrast, presented alongside are the means and standard deviations for the latency distributions prescribed by the prior mixing distribution of parameter values, unmodified by the observed performance sample (computational formulae are presented in Neufeld & Williamson, 1996).

Clinical and Cognitive-Experimental Properties of Individual Latency Distributions

The posterior latency distributions for an individual with the current performance sample can be examined with respect to their inherent substantive significance, namely potential clinical impli-

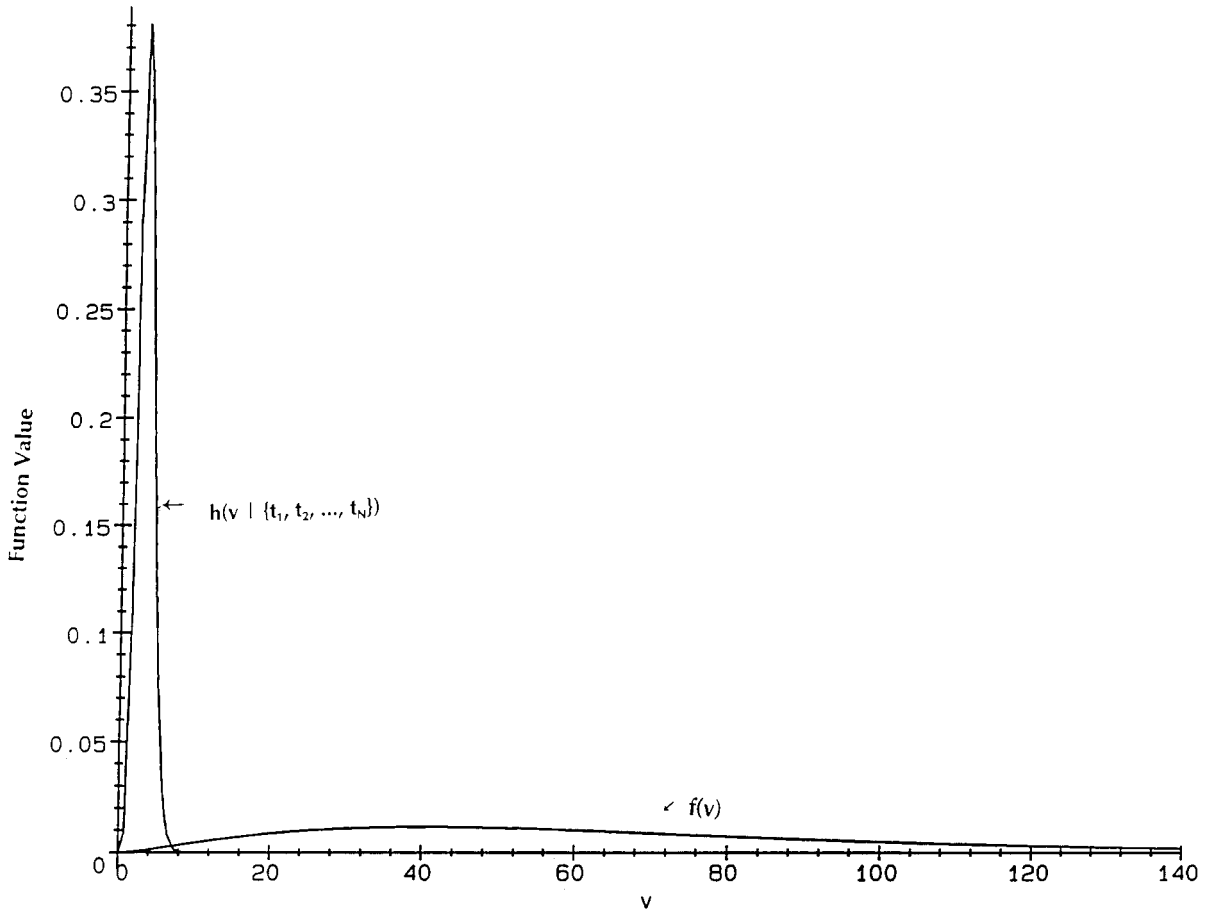

Figure 6. Prior and posterior distributions, $f(v)$ and $h(v\{t_1, t_2, \ldots, t_N\})$, of rate-parameter v.

cations. The latter entail nuances of distribution features afforded by formal developments.

The means of the posterior latency distributions are similar across the respective priors, with the exception of $m = .00001$. Not surprisingly, they are generally close to the observed performance mean of 2.5675. What noticeably distinguishes these posterior distributions are their shapes.

Note first that under the low-encoding load, the paranoid schizophrenia patient, represented by the curve for $m = 70.00001$ in Figure 8, is alienated from the exponential-like distribution ($m = .00001$). The latter distribution obviously is favorable to very early encoding completion. Other implications of the differing shapes are more subtle.

Consider the probability-distribution functions $F(t|\{t_1, t_2, \ldots, t_N\})$ for the latency distributions presented in Figure 9. These functions express the probability of completing the encoding process at or before time t. Such probabilities come to the fore where the opportunity for encoding is of limited duration (as with quickly changing scenes, or by extension to the auditory modality, normally rapid speech). With a protracted encoding process, the risk of incomplete assembly of time-limited "presenting data" is increased (cf. Yates, 1973).

The modeled effect of paranoid schizophrenia disorder on the current performance sample's posterior probability distribution function, under the high-encoding load, is presented in Figure 10. The disorder evidently puts the individual at a disadvantage during shorter time limits for encoding. This disadvantage disappears for longer durations, where it evidently gives way to a relative advantage.

Along with the risk of incomplete encoding during the briefer windows of time goes the risk of missing cues about the objective significance of events, objects, or individuals' actions. With such fractional information, veridicality of inferences stands to be compromised, even if other functions are intact (e.g., manipulations in working memory).

When it comes to symptomatology of paranoid schizophrenia, notably thought-content disorder (delusions and thematic hallucinations; *Diagnostic and Statistical Manual of Mental Disorders,* 4th ed., American Psychiatric Association, 1994, p. 275), the above risk of faulty deduction can be exacerbated by a tendency toward inordinate confidence in one's proclamations (reviewed in Neufeld, 1991a). The present developments lend a formal aspect to viewing such symptomatology as consequential to certain variations on normal cognition (Maher, 1988; see also Lange & Houran, 2000).

The above deficit during shorter time apertures for encoding completion also impinges on selected forms of stress negotiation. Briefly, *decisional control,* a prominent form of coping (cf. Lees &

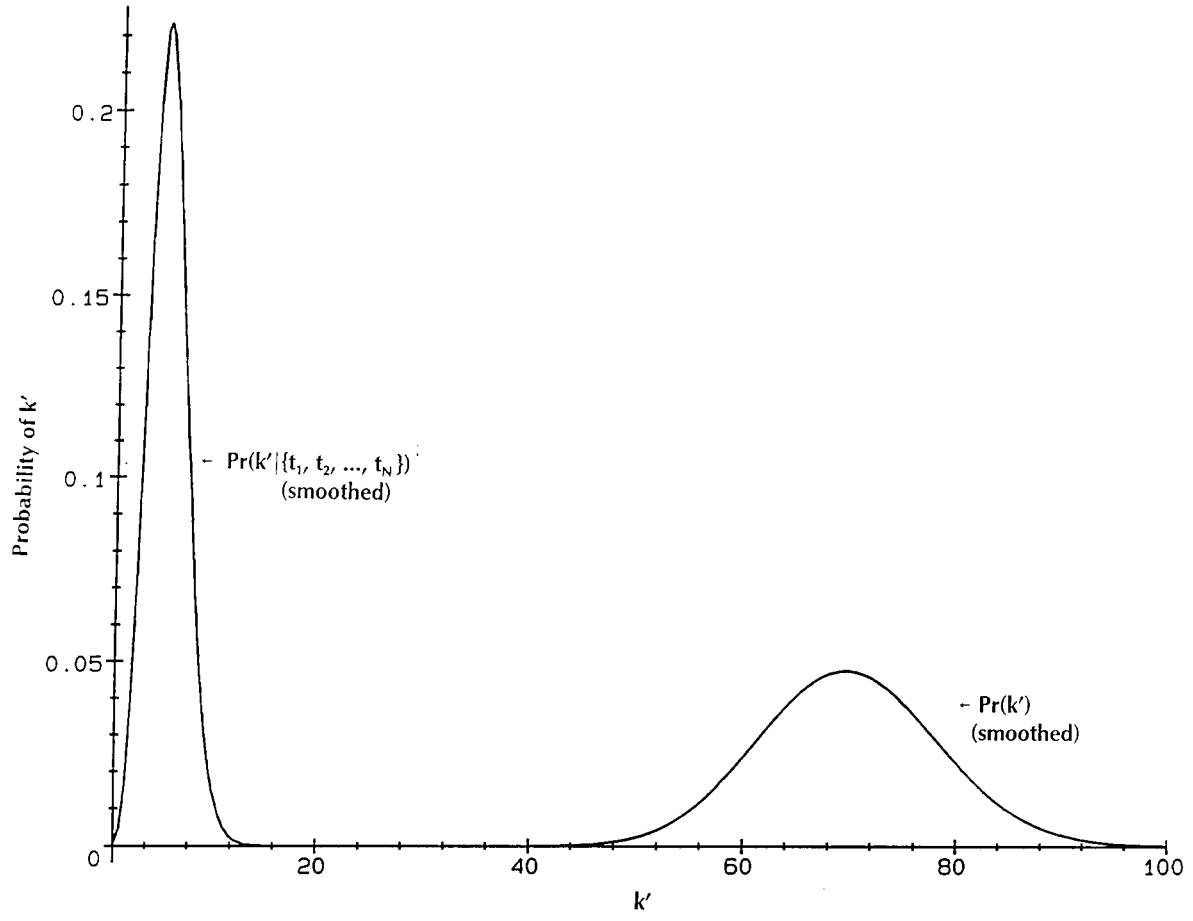

Figure 7. Prior and posterior distributions, $Pr(k')$ and $Pr(k'|\{t_1, t_2, \ldots, t_N\})$, of subprocess-number parameter, k'.

Neufeld, 1999), involves positioning oneself in a multifaceted stressing situation so as to engage the situational option of minimal threat. Formal expressions of decisional control's cognitive demands and threat-diminishing benefits have indicated the involvement of memory- and visual-search processes, including encoding of option-relevant properties (Morrison, Neufeld, & Lefebvre, 1988; Neufeld, 1999b). Elongated encoding would be expected to compromise the information necessary to mediate benefits of this important form of coping. Vulnerability to missing the critical complement of information has been put forth as a source of stress-susceptibility amongst these patients (Neufeld, 1991a).

Potentially aggravating the above susceptibility among individuals with paranoid schizophrenia are the adverse effects of stress itself on the speed of encoding (Neufeld, 1999a). Within the present formal context, these effects comprise an increase in the parameter r of the mixing distribution of v. Quantitative expressions of these exacerbating effects have been presented in other venues (Neufeld, 1994; Neufeld & Carter, 2000; Neufeld & McCarty, 1994).

Not all aspects of schizophrenia cognitive performance examined here are adversely affected or simply retain normal levels. As stated above, inspection of the graphs of $F(t|\{t_1, t_2, \ldots, t_N\})$ in Figure 10 discloses certain regions of potential superiority to nonpatient counterparts. The individual with the present performance sample theoretically is advantaged once the probabilities of encoding completion tend to exceed certain values (approximately .60, in Figure 10). This difference makes for specific empirical predictions: Individuals with these characteristics essentially should outperform their nonpatient counterparts when the available encoding period t is not curtailed and exceeds the point of distribution-function crossover.

Domains of cognitive weaknesses and strengths also may beneficially inform the design of therapeutic milieus. The latter may be crafted to take into account the unfolding of comparative probabilities of encoding process finalization and to take advantage of the putative benefits of prolonged epochs.

Finally, an individual's performance sample may have direct implications for monitoring progress in therapy. The degree to which an individual's task-performance sample typifies that occurring under clinical conditions of interest is available, as follows. The obtained sample can be submitted to Bayesian priors issuing from competing sources, in the present case diagnostic samples under alternate encoding loads. Available is the *unconditional joint density* (*ujd*, analogous to unconditional joint probability) of the performance sample, as seen in the denominator of Appendix Equation A2 and instantiated in the present case as the denominator of Appendix Equation A3.

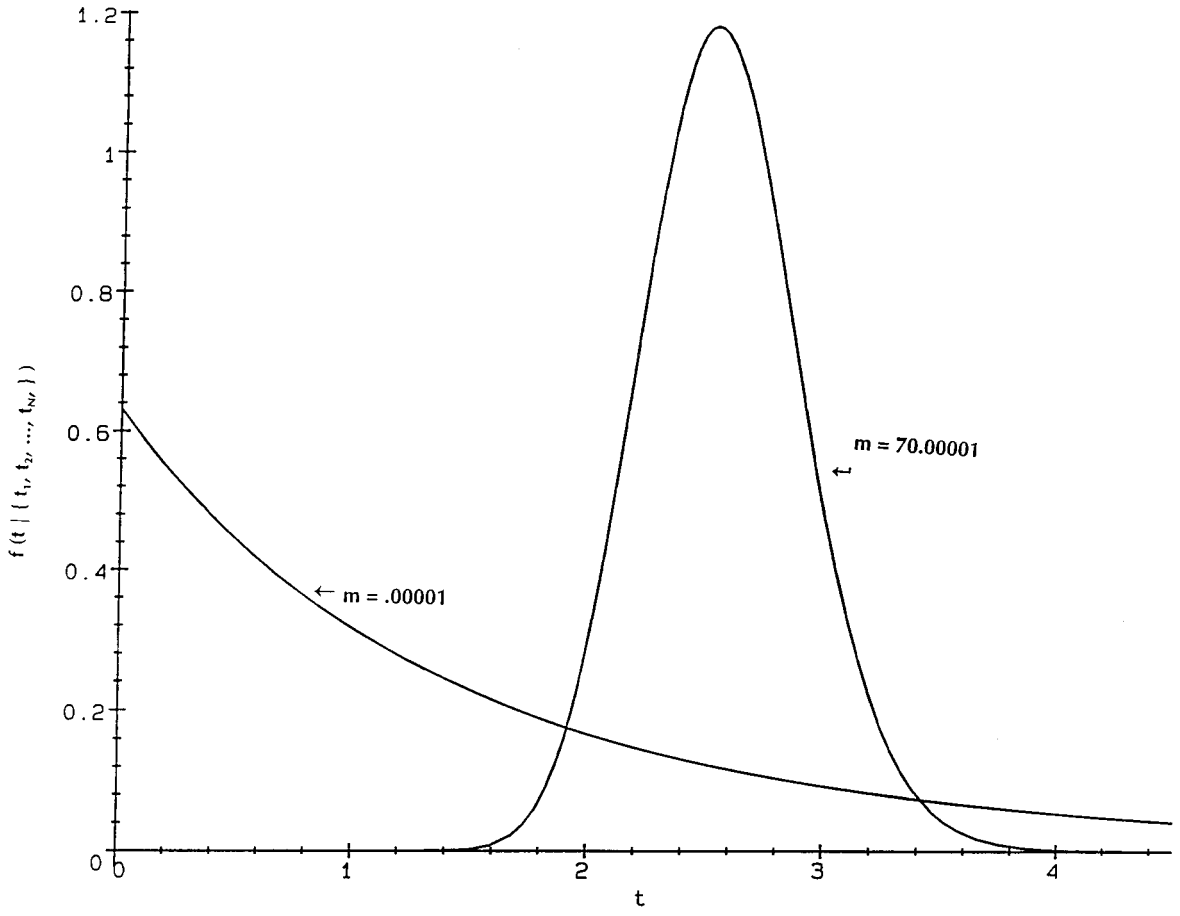

Figure 8. Effects of Bayesian prior distributions for paranoid-schizophrenia participants ($m = 70.00001$) and for nonpatients ($m = .00001$) on the posterior latency distribution for a given sample of performance under low encoding-load conditions.

Computed values for the current samples are presented in Table 3. As seen in the diagonals of the table, the *ujd* tends to be maximized for the encoding-load/diagnostic-status combination from which the sample was initially obtained. In terms of odds-ratios, the sample of performance acquired from the nonpatient under high encoding demands, for example, is $.3696(10^7)$ as likely to characterize performance from such a source as that from a paranoid-schizophrenia participant performing under the same encoding load. To take another example, the performance sample {2.177, 3.184, 2.411, 2.498} is $.4960(10^{11})$ as likely to be produced by someone who suffers from paranoid schizophrenia and is undertaking the low encoding load as by a nonpatient undertaking the same load.

Improvement in key aspects of cognitive efficiency, as an effect of therapeutic agents, is a goal increasingly touted in certain pharmaceutical circles. The above *ujd*s and odds ratios may help monitor whether obtained samples of performance indeed are being edged closer to their normal-functioning targets.

Complementing Levels of Study

Charting the stochastic distributions of selected cognitive functions, as exemplified here, in principle can contribute to investigations taking place at other levels of analysis. Included are connectionist modeling of cognitive psychopathology (e.g., Carter, 2000) and neuroimaging investigations of brain activation.

Apropos of the latter undertaking, Bayesian methodology illustrated in this article can help establish the stochastic trajectory of process completion. Confidence intervals for process perseveration can be ascertained for individuals from larger groups whose performance samples are similar (consider Figures 8–10). Indeed, such information can be used to homogenize subgroups of participating samples (cf. Frackowiak, Friston, Frith, Dolan, & Mazziotta, 1997; Reiman, Lane, Van Petten & Bandettini, 2000; cf. Carter, Neufeld, & Benn, 1998).

Candidate neuroanatomical loci for formally educed encoding abnormality have been proposed according to findings from mainstream cognitive neuropsychology (Neufeld & Williamson, 1996) and those from spectroscopy MRI (Carter & Neufeld, 1999). Such candidate sites represent "regions of interest" (ROIs) for fMRI studies of psychopathology. These analyses supposedly indicate *where* to look for neurophysiological substrata of encoding prolongation. Mapping the stochastic dynamical trajectory of encoding completion may complement

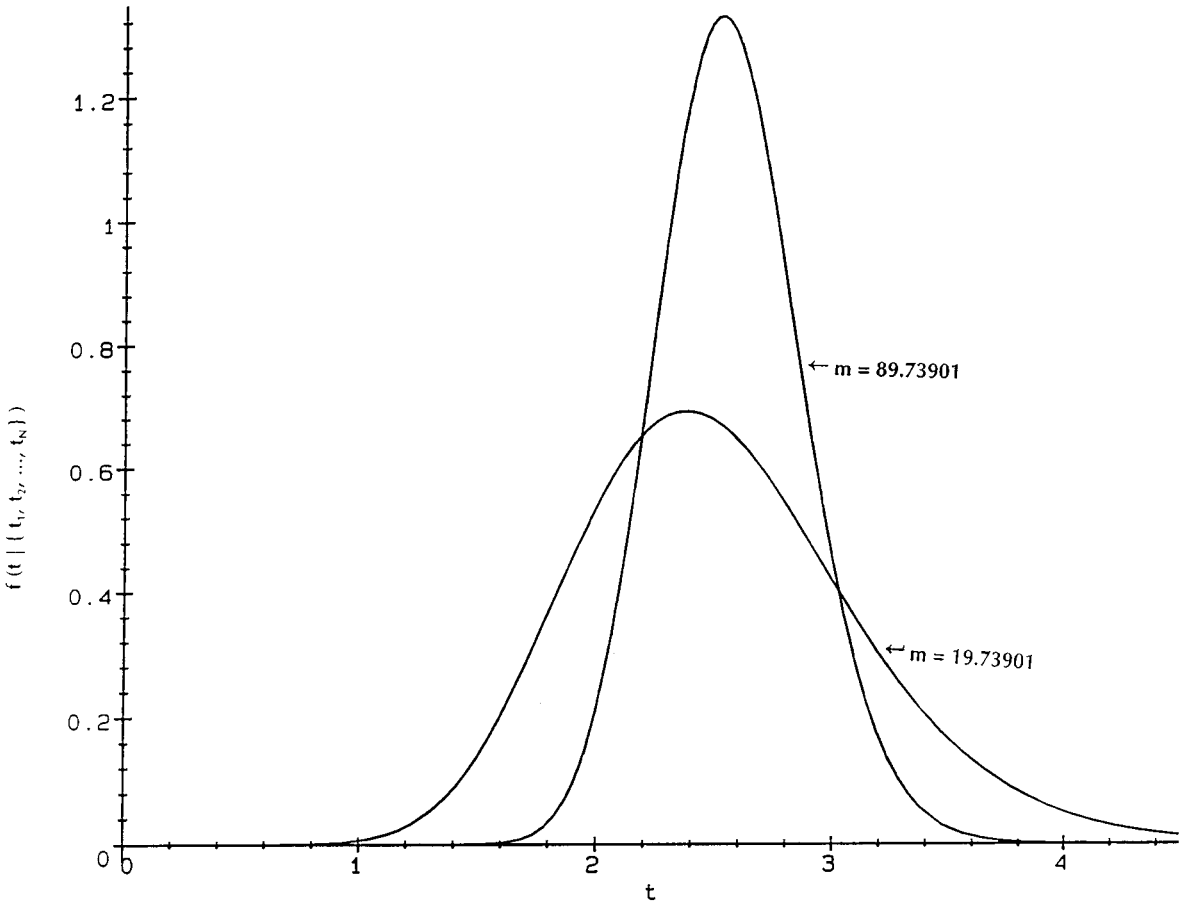

Figure 9. Effects of Bayesian prior distributions for paranoid-schizophrenia participants ($m = 89.73901$) and for nonpatients ($m = 19.73901$) on the posterior latency distribution for a given sample of performance under high encoding-load conditions.

these analyses by indicating *when* to look for activation deviations. Formally prescribed "times of interest," complementing ROIs, should contribute to the time-and-space coordination of MRI measurement. Such directions currently are being implemented in the present setting, conjoint with the spatial and temporal resolution of MRI measurement at a strength of 4.0 Tesla.

Discussion

In this article, we described a progression of investigative steps, from cognitive science to applied clinical assessment. Experimental findings on cognitive psychopathology have been embedded in stochastic modeling available from the domain of cognitive science. Results have been rigorously integrated with performance samples, yielding performance models adapted to individuals.

Individualized estimation of cognitive-performance model parameters is seen to usher in construction of performance features, such as process-completion latency. Such features, in turn, harbor potential significance for cognitive symptomatology, stress negotiation, and complementing levels of analysis, such as fMRI studies of brain activation patterns. Several observations and issues are prompted by the application of these methods in the numerical example involving paranoid schizophrenia.

Methodology and Rigor of Individualized Stochastic Modeling

In our numerical example, Bayesian posterior distributions were constructed for individuals whose task performance itself contributed to the development of prior distributions of model parameters. This lack of complete independence between the two sources of information, however, is somewhat trivial in the present instance. For each performance sample, four latency values were extracted from an original sample of 360 observations. Moreover, independence of performance specimens, and the priors to which they are referred, is not at issue where samples are obtained from new clients.

The construction and use of individualized (posterior) distributions of model parameters, using empirical samples of the very performance being modeled, nevertheless may seem troublesome. However, complete separation of explanatory constructs, such as substantively meaningful model parameters, and observations in which they theoretically participate, is not essential to the explan-

Table 2
Means and Standard Deviations in Encoding Latencies for Bayesian Priors and Bayesian Posteriors

	Prior		Posterior, given latencies {2.177, 3.184, 2.411, 2.498}	
Classification	Mean latency	Standard deviation of latency	Mean latency	Standard deviation of latency
Paranoid schizophrenia				
Low-encoding load[a]	1.7374	2.4990	2.5269	0.3448
High-encoding load[b]	2.2280	3.1892	2.5658	0.3062
Control				
Low-encoding load[c]	0.2483 (10^{-6})	0.1198 (10^{-8})	1.8726	2.2502
High-encoding load[d]	0.4904	0.7407	2.5327	0.6122

[a] Parameters of prior distribution: $r = .03735$; $k = 2.5044$; $m = 70.00001$. [b] Parameters of prior distribution: $r = .03735$; $k = 2.5044$; $m = 89.73901$. [c] Parameters of prior distribution: $r = .03735$; $k = 2.5044$; $m = .00001$. [d] Parameters of prior distribution: $r = .03735$; $k = 2.5044$; $m = 19.73901$.

atory constructs' scientific legitimacy. As aptly stated by Flanagan (1991),

> In physics there are many explanatory constructs, electrons for example, which cannot be measured independently of the observable situations in which they figure explanatorily. What vindicates the explanatory use of such constructs is the fact that, given everything else we know about nature, electrons best explain the observable processes in a wide array of experimental tests, and lead to successful predictions. (p. 380)

From a clinical perspective, the present procedures are not unlike those entailing the diagnostic use of blood samples. A specimen is innocuously obtained from the patient and assayed according to the body of knowledge underlying the assessment technology. The extracted sample can then be referred to the relevant store of information, making for a customized picture of disorder with potential treatment and prognostic implications. By extension, clinical assessment may include the development of Bayesian priors, paralleling the current practice in psychological assessment of establishing test-performance distributions of standardization samples.

Conceptual Status of Models

The modeling illustrated here entails certain assumptions regarding both prior distributions of parameter values and base distributions of performance latency. Although not beyond challenge, the selected priors certainly are defensible, as previously noted.

The base distributions assume that the durations of subprocesses are distributed exponentially. This assumption has undergone its own share of challenges, but is not lacking in empirical support (e.g., Ashby, 1982; Ashby & Townsend, 1980; Ashby, Tein, & Balakrishnan, 1993; Townsend & Ashby, 1983). Moreover, the exponential assumption is more forgiving than many in terms of robustness of inferences vis à vis potential violation of the speci-

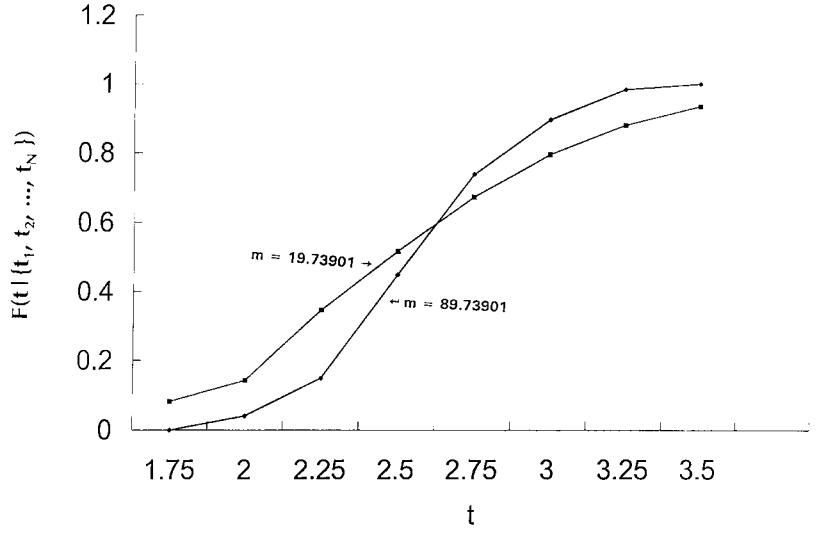

Figure 10. Effects of Bayesian prior distributions for paranoid-schizophrenia participants ($m = 89.73901$) and for nonpatients ($m = 19.73901$) on the probability of encoding-completion at or before *t*.

Table 3
Unconditional Joint Densities of Encoding-Latency Performance Samples Under Alternate Bayesian Priors

	Representative individual-participant-latency sets			
	Paranoid schizophrenia		Control	
Classification	Low-encoding load {2.177, 3.184, 2.411, 2.498}	High-encoding load {3.476, 7.150, 9.00, 4.438}	Low-encoding load {1.037, 0.554, 0.191, 6.390}	High-encoding load {1.137, 3.204, 1.371, 2.207}
Paranoid schizophrenia				
Low-encoding load[a]	.00737	.999776 (10^{-11})	.23614 (10^{-32})	.70547 (10^{-9})
High-encoding load[b]	.00684	.17176 (10^{-12})	.29397 (10^{-40})	.54107 (10^{-11})
Control				
Low-encoding load[c]	.1486 (10^{-12})	Pr{ · } → 0.0	.65324 (10^{-12})	.80080 (10^{-12})
High-encoding load[d]	.00083	.32930 (10^{-7})	.75783 (10^{-13})	.00002

[a] Parameters of prior distribution: $r = .03735$; $k = 2.5044$; $m = 70.00001$. [b] Parameters of prior distribution: $r = .03735$; $k = 2.5044$; $m = 89.73901$. [c] Parameters of prior distribution: $r = .03735$; $k = 2.5044$; $m = .00001$. [d] Parameters of prior distribution: $r = .03735$; $k = 2.5044$; $m = 19.73901$.

fied intercompletion latencies (see, e.g., Schweikert & Townsend, 1989). The challengeable shape of subprocess completions, on their convolution into total completion times, furthermore can issue in (or at least approximate) a normal distribution, whose own shape is ubiquitous.

At the same time, developments in distribution-free modeling of processing architecture, and constructs such as processing capacity, are available (e.g., Townsend & Nozawa, 1995). Additionally, Bayesian methods have been extended in distribution-general directions (Chechile, 1998). These advances point to potentially important future implementations in this setting.

Note, however, that the predictions put forth under the present assumptional framework, nevertheless conform to patterns of empirical distribution-moments across diagnostic groups and task conditions (for model-discerning properties of the second moment, see Townsend, 1984, and Townsend & Ashby, 1983, p. 153; for a discourse on some important relations between model means and variances, see Townsend & Diedrich, 1996). Despite frailties of moments in addressing certain properties of the distributions producing them, it seems tenable that the actual properties should be coextensive with those that are assumed, at least in ways constrained by the above predictions.

More generally, Busemeyer and Townsend (1993) have depicted stochastic modeling's role in the study of human cognition, as follows:

> . . . we can never know all the factors that influence an individual's [covert processing transactions] from one moment to the next. The best we can hope to do is determine some important characteristics of the [probability distribution of events] as a function of the few known factors that we can identify or observe. (p. 434)

Again, regarding epistemological status of cognitive-process modeling,

> Of course, we do not mean to imply that [human information processors] consciously carry out the computations indicated by the [composition of the processing model that manifests its product stochastically over continuous time]. Instead, those computations are assumed to be realized by an underlying neural system, and the [mathematical portrayal] is an abstract representation of its essential dynamic properties . . . . (pp. 444–445)

Appropriating this depiction to clinical abnormalities, the behaviorally significant deviations from normal in the above underlying neural system ostensibly exhibit outcroppings in the abstract representation of the system's essential dynamic properties.

Isomorphic association(s) (as described above) might imply that the present modeling takes on more of an instrumentalist than realist tenor. That is, model value is judged against predictive utility, over and against strict alliance of its constructs with real-world observables (Casti, 1989). Flanagan's distinction between simulation and duplication of cognitive events provides a related slant on possible model status. At minimum, the present formal apparatus tenders a decidedly quantitative platform for potential explanation, measurement, and prediction.

Integration With Psychometric Measures

Individual parameter estimates can be synthesized with psychometric measures to which they conceivably relate. For example, psychometric measures potentially tapping encoding efficiency may be correlated to the present Bayesian estimates of k' (see, e.g., Paivio & Harshman, 1983, for IDQ scoring key). Similarly, psychometric measures of stress susceptibility may be correlated to the parameter v (e.g., Endler, Edwards, Vitelli, & Parker, 1989). Doing so lends construct-representation validity to the psychometric measures and helps validate the substantive interpretation of the model parameters. Furthermore, it may be possible to use highly predictive psychometric measures as surrogates for model-parameter estimates, if desired for practical or other reasons (see Carter et al., 1998, for elaboration).

Challenges to Valid Clinical Inferences

We return now to the earlier posed challenges to valid inferences in clinical science (Maher, 1974; Chapman & Chapman, 1973, 1978; Meehl, 1978). Apropos of Maher's (1974) Bull-in-a-Worcester-China-Shop Scenario and ubiquitous damage to the

processing system, affected and spared functions have been proposed. Stimulus encoding is affected, and memory scanning and response processes evidently are spared. Furthermore, the parametric composition of disturbed encoding has been tendered, and presents a picture of reduced efficiency in deploying intact processing capacity. The "nature of the bull" has been put forth in terms of a model parameter that provides a nexus for findings that cut across multiple cognitive paradigms (e.g., Carter & Neufeld, 1999; Neufeld & Williamson, 1996).

The dissection of strong theory–evidence relations (Meehl, 1978) from the Bayesian perspective discussed above can be applied to predictions emanating from the present formal theoretical platform. Implicated alongside is the viability of the interplay of generalized deficit and psychometric properties of dependent-variable measures as a parsimonious explanation of empirical collectives (Chapman & Chapman, 1978).

Strong predictions in this context have certain parallels in meteorological forecasting (cf. Meehl, 1978). A weak weather prediction, for instance, would have it that there will be precipitation in the forthcoming November. A stronger prediction would venture the amount involved. A genuinely strong and bold prediction moreover would invoke a dynamical element and specify the actual distribution of quantities across the 30 days of November, whereby the predicted total is realized (cf. Busemeyer & Stout, 2002). Nondynamical configurations similarly qualify as strong predictions. In either case, key to prediction strength comprises an observational set whose combined occurrence essentially hangs on tenability of the proposed theory (cf. Knight & Silverstein, 2001).[5]

In the present instance, means and intertrial variances of performance latencies, aggregated at encoding levels and diagnostic groups, correspond to mixture-model-prescribed patterns (see Figure 5). Other distribution properties also could come into play (quantiles, such as the median; "hazard-function" shapes, and so on; descriptions of which are available in multiple sources, including Neufeld, 1998; Carter & Neufeld, 1999). Furthermore, to the extent that it applies to results from several paradigms, the tendered function abnormality, and its parametric makeup, resembles Flanagan's, 1991, plausible explanation of a corpus of findings (see convergent evidence for encoding and its espoused parametric composition, described in the *Paradigm and Findings* and *Processing Model and Parameter Titration* sections).

Note that the present Bayesian developments potentially issue in multiple sets of predictions, each set arising from individualized posterior parameter distributions (illustrated in Figures 6 and 7). Customized distributions, or cumulative distributions of encoding-process latencies (as illustrated in Figures 8–10) should be predictive of their empirical counterparts. Indeed, one such set of predictions attends the performance of each parametrically homogeneous enclave of participants—identified as those with similar performance samples, and to whom the same Bayesian prior applies (performance samples themselves, of course, can be withheld from the empirical distribution subjected to prediction).

Finally, psychometric properties of measured response parameters, such as latencies, in principle can be introduced into formal process models. Psychometric measurement errors, or model-exogenous noise (described in Carter et al., 1998), stand to be integrated with cognitive-process completion latencies (technically, by convolving the posited distribution of measurement errors with that of encoding-process latencies, the latter portrayed in Figures 2 and 8–10). Measurement errors thus are expressed in an expanded version of the theoretical base distribution of trialwise latencies (see Figure 5).

This expansion, in turn, duly infiltrates the Bayesian posterior distribution of parameter values (addressed in Figures 6 and 7 and Table 1) and customized latency distributions (Figures 8–10). It can be seen to do so according to its modification of values entering into computation of the above distributions, specifically, modification of $cjd(\{t_1, t_2, \ldots, t_N\}|k, v)$ and $ujd(\{t_1, t_2, \ldots, t_N\})$ of Appendix Equation A2 and $f(t|k', v)$ of Equation A8.

In a similar vein, psychometric measurement error can be funnelled into non-Bayesian parameter estimates (cf. Busemeyer & Stout, 2002; Treat et al., 2002). Layouts of such estimates across participant samples, incorporating both between- and within-sample sources of dispersion (Busemeyer & Stout, 2002), can furnish a comprehensive context for interpreting estimate values in particular cases.

Concluding Comments

Results from analytical procedures described here may appear specific to the experimental paradigm to which they were applied. Indeed, there is precedent for model architectures to be sensitive to paradigmatic nuances (e.g., Schweikert, 1978). However, processes such as the encoding of presenting stimulation into a task-facilitative format seem ubiquitous, by whatever architecture they may assume for a particular task. Encoding, and its deficit in schizophrenia, evidently occurs in a multiplicity of tasks, ranging from card-sort tasks, through memory-search tasks, to eye-tracking of a moving target (reviewed in Neufeld & Williamson, 1996).

Transcendence of task morphology by component processes raises sanguinity about similar transcendence of process parameters. The parameters may apply in instances well beyond the task by which they are initially assessed, thus increasing incentive for precise estimation. Routine accurate estimation has become more and more feasible with available computational capacity and computer-algebra software. It is hoped that the quantitative methods illustrated here will facilitate the more general implementation of cognitive science in clinical assessment.

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[5] More formally, let $\{O_1, O_2, \ldots, O_j, \ldots, O_p\}$ be a set of p observations whose probability of co-occurrence, or intersection in typical notation, is

$$Pr\left(\bigcap_{j=1}^{p} O_j\right).$$

The O_j conceivably are independent, apart from the theory-related coalescence. The probability of their intersection then is the product of their individual probabilities,

$$\prod_{j=1}^{p} Pr(O_j),\ Pr(O_j) > 0.$$

As the number of outcomes increases, this product becomes 0:

$$\lim_{p\to\infty} \prod_{j=1}^{p} Pr(O_j) = 0.$$

The convergence on 0 is hastened if the O_j represent precise quantities, because the independent $Pr(O_j)$ themselves are diminished, apart from tenable theory.

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Appendix

Formulae Underlying Figures and Tables

Recall that Bayes's theorem states

$$Pr(A|B) = [Pr(A)Pr(B)|A)]/Pr(B). \quad \text{(A1)}$$

As applied in the present context, the probability density function of parameter-value combination $V = v \cap K' = k'$, given latency-sample $\{t_1, t_2, \ldots, t_N\}$, in turn, is

$$g(v, k'|\{t_1, t_2, \ldots, t_N\}) = \frac{f(v)Pr(k')cjd(\{t_1, t_2, \ldots, t_N\}|k', v)}{ujd(\{t_1, t_2, \ldots, t_N\})}. \quad \text{(A2)}$$

Where v is gamma distributed, with shape-parameter k and rate-parameter r, $f(v)$ is

$$\frac{(rv)^{k-1}}{\Gamma(k)} re^{-rv};$$

where k' is Poisson-distributed, with "intensity parameter" m,

$$Pr(k') = \frac{m^{k'}}{k'!} e^{-m};$$

and where t is Erlang distributed, with parameters k' and v, the conditional joint density of $\{t_1, t_2, \ldots, t_N\}$, $cjd(\{t_1, t_2, \ldots, t_N\}|k', v)$ is

$$\prod_{i=1}^{N} \frac{(vt_i)^{k'-1}}{(k'-1)!} ve^{-(vt_i)},$$

and the unconditional joint density of $\{t_1, t_2, \ldots, t_N\}$, $ujd(\{t_1, t_2, \ldots, t_N\})$, or ujd for short, is that taken across all combinations of v and k'. Therefore,

$$ujd = \int_0^\infty \sum_{w=0}^{\infty} \frac{(rx)^{k-1}}{\Gamma(k)} re^{-rx} \frac{m^w}{w!} e^{-m} \prod_{i=1}^{N} \frac{(xt_i)^{w-1}}{(w-1)!} xe^{-xt_i} dx$$

$$= \frac{r^k e^{-m}}{\Gamma(k)} \sum_{w=0}^{\infty} \frac{m^w}{w!} \frac{\Gamma(Nw+k)}{[r + \sum_{i=1}^{N} t_i]^{Nw+k}} \prod_{i=1}^{N} \frac{t_i^{w-1}}{(w-1)!},$$

and for convenience, we let

$$\frac{ujd\,\Gamma(k)}{r^k e^{-m}} = D.$$

We have, then,

$$g(v, k'|\{t_1, t_2, \ldots, t_N\}) = \frac{\frac{(rv)^{k-1}}{\Gamma(k)} re^{-rv} \frac{m^{k'}}{k'!} e^{-m} \prod_{i=1}^{N} \frac{(vt_i)^{k'-1}}{(k'-1)!} ve^{-vt_i}}{ujd}, \quad \text{(A3)}$$

$$h(v|\{t_1, t_2, \ldots, t_N\}) = \sum_{k'=0}^{\infty} g(v, k'|\{t_1, t_2, \ldots, t_N\}),$$

$$= \frac{\sum_{k'=0}^{\infty} v^{Nk'+k-1} e^{-(r+\sum_{i=1}^{N} t_i)v} \frac{m^{k'}}{k'!} \prod_{i=1}^{N} \frac{t_i^{k'-1}}{(k'-1)!}}{D}, \quad \text{(A4)}$$

and

$$Pr(k'|\{t_1, t_2, \ldots, t_N\}) = \int_0^\infty g(v, k'|\{t_1, t_2, \ldots, t_N\})dv$$

$$= \frac{\frac{m^{k'}}{k'!} \frac{\Gamma(Nk'+k)}{[r + \sum_{i=1}^{N} t_i]^{Nk'+k}} \prod_{i=1}^{N} \frac{t_i^{k'-1}}{(k'-1)!}}{D}. \quad \text{(A5)}$$

Figures 6 and 7 are based on Equations A4 and A5, respectively.

$$E(v|\{t_1, t_2, \ldots, t_N\}) = \sum_{k'=0}^{\infty} \frac{\frac{\Gamma(Nk'+k+1)}{[r + \sum_{i=1}^{N} t_i]^{Nk'+k+1}} \frac{m^{k'}}{k'!} \prod_{i=1}^{N} \frac{t_i^{k'-1}}{(k'-1)!}}{D}. \quad \text{(A6)}$$

The above equation displays how the mean value of v, given the encoding-performance sample of N latencies $\{t_1, t_2, \ldots, t_N\}$, entails an admixture of the maximum-likelihood estimate of v based exclusively on the performance sample,

$$\frac{Nk'}{\sum_{i=1}^{N} t_i},$$

and the mean value of the prior distribution of v, k/r (consider the bracketed portions of the first ratio in the numerator of the summed term).

$$Var(v|\{t_1, t_2, \ldots, t_N\}) = \sum_{k'=0}^{\infty} \frac{\frac{\Gamma(Nk'+k+2)}{[r + \sum_{i=1}^{N} t_i]^{Nk'+k+2}} \frac{m^{k'}}{k'!} \prod_{i=1}^{N} \frac{t_i^{k'-1}}{(k'-1)!}}{D}$$

$$- [E(v|\{t_1, t_2, \ldots, t_N\})]^2. \quad \text{(A7)}$$

(*Appendix continues*)

The conditional density function for encoding latencies is

$$\frac{\int_0^\infty \sum_{k'=0}^{\infty} \frac{(rv)^{k-1}}{\Gamma(k)} re^{-rv} \frac{m^{k'}}{k'!} e^{-m} \prod_{i=1}^{N} \left(\frac{(vt_i)^{k'-1}}{(k'-1)!} ve^{-vt_i} \right) \frac{(vt)^{k'-1}}{(k'-1)!} ve^{-vt} dv}{ujd}$$

$$= \frac{\sum_{k'=0}^{\infty} \frac{m^{k'}}{k'!} \frac{\Gamma[k'N+k'+k]}{[r+t+\sum_{i=1}^{N} t_i]^{k'N+k'+k}} \prod_{i=1}^{N} t_i^{k'-1} \frac{t^{k'-1}}{((k'-1)!)^{N+1}}}{D}. \quad \text{(A8)}$$

Figures 8 and 9 are based on Equation A8. The conditional distribution function

$$F(t|\{t_1, t_2, \ldots, t_N\}) = \int_0^t f(t'|\{t_1, t_2, \ldots, t_N\}) dt'$$

$$= \sum_{k'=0}^{\infty} \frac{m^{k'}}{k'!} \frac{1}{D} \left[\frac{\Gamma(k'N+k) \prod_{i=1}^{N} t_i^{k'-1}}{[r+\sum_{i=1}^{N} t_i]^{k'N+k} ((k'-1)!)^N} - \sum_{j=0}^{j=k'-1} \frac{\Gamma[k'N+j+k] \prod_{i=1}^{N} t_i^{k'-1} t^j}{j![r+t+\sum_{i=1}^{N} t_i]^{k'N+j+k} ((k'-1)!)^N} \right]$$

$$= 1.0 - \frac{\sum_{k'=0}^{\infty} \frac{m^{k'}}{k'!} \sum_{j=0}^{j=k'-1} \frac{\Gamma[k'N+j+k] \prod_{i=1}^{N} t_i^{k'-1} t^j}{j![r+t+\sum_{i=1}^{N} t_i]^{k'N+j+k} ((k'-1)!)^N}}{D}. \quad \text{(9)}$$

Figure 10 is based on Equation (A9).

Note that the final form of Equation (A9) makes apparent that the conditional probability, given $\{t_1, t_2, \ldots, t_N\}$, of completing an encoding process comprising k' subprocesses by time t is 1.0 minus the conditional probability of completing $k'-1$ or fewer subprocesses by time t (consider the second summation operator in the numerator of the term subtracted from 1.0).

Finally,

$$E(T^n|\{t_1, t_2, \ldots, t_N\}) = \int_0^\infty f(t|\{t_1, t_2, \ldots, t_N\}) t^n dt$$

$$= \frac{\sum_{k'=0}^{\infty} \frac{m^{k'}}{k'!} \frac{\Gamma(Nk'+k-n)(k'+n-1)!}{[r+\sum_{i=1}^{N} t_i]^{Nk'+k-n}} \frac{\prod_{i=1}^{N} t_i^{k'-1}}{((k'-1)!)^{N+1}}}{D}, \quad \text{(A10)}$$

for $(Nk'+k) > n$. The conditional mean and variance then are available as

$$E(T^{n=1}|\{t_1, t_2, \ldots, t_N\}),$$

and

$$E(T^{n=2}|\{t_1, t_2, \ldots, t_N\}) - (E(T^{n=1}|\{t_1, t_2, \ldots, t_N\}))^2.$$

Similarly,

$$E(k'^n|\{t_1, t_2, \ldots, t_N\}), = \sum_{k'=0}^{\infty} \frac{\frac{\Gamma(Nk'+k)}{[r+\sum_{i=1}^{N} t_i]^{Nk'+k}} \frac{m^{k'}}{k'!} \prod_{i=1}^{N} \frac{t_i^{k'-1}}{(k'-1)!} k'^n}{D}.$$

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