Mathematical and Computational Modeling in Clinical Psychology

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Abstract

This chapter begins with a description of clinical mathematical and computational modeling as it fits within the larger context of mathematical and computational modeling. In general, models of normal cognitive-behavioral functioning are titrated to accommodate performance deviations associated with psychopathology; model features remaining intact are taken to indicate functions that are spared, and those that are perturbed are triaged as signifying functions that are disorder affected. Clinical applications also can be used to test the generality of models originally focusing on normal functioning. Potential assets, in the form of clinical-information added-value, are introduced. Distinctions and interrelations among forms of modeling in clinical science and assessment are stipulated, with an emphasis on analytical, mathematical modeling. Preliminary conceptual and methodological considerations essential to appreciating the unique contributions of clinical mathematical and computational modeling are presented. Following are concrete examples illustrating the benefits of modeling as applied to specific disorders, such as chronic substance abuse, autism spectrum disorders, developmental dyslexia, and schizophrenia. Emphasis in each case is on clinically-significant information uniquely yielded by the modeling enterprise. Implications for the functional side of clinical functional neuro-imaging are detailed. Challenges to modeling in the domain of clinical science and assessment are described, as are tendered solutions. The chapter ends with a description of continuing challenges and future opportunities.

Introduction

"The important point for methodology of psychology is that just as in statistics one can have a *reasonably precise theory of probable inference*, being 'quasi-exact about the inherently inexact,' so psychologists should learn to be sophisticated and rigorous in their metathinking about open concepts at the substantive level. … In social and biological science, one should keep in mind that *explicit definition* of theoretical entities is seldom achieved in terms of initial observational variables of those sciences, but it becomes possible instead by theoretical reduction or fusion (Meehl, 1978; p. 815)." Mathematical and computational modeling of clinical-psychological phenomena can elucidate clinically-significant constructs by translating them into variables of a quantitative system, and lending them meaning according to their operation within that very system (e.g., Braithwaite, 1968). Availed are new explanatory properties, options for clinical-science measurement, and tools for clinical-assessment technology. This chapter is designed to elaborate upon the above assets by providing examples where otherwise intractable or hidden clinical information has been educed. Issues of practicality and validity, indigenous to the clinical setting, are examined, as is the potentially unique contribution of clinical modeling to the broader modeling enterprise. Emphasis is on currently prominent domains of application, and exemplary instances within each. Background material for the current developments is available in several sources (e.g., Busemeyer & Diedrich, 2010; Neufeld, 1998; 2007a).

We begin by considering an overall epistemic strategy of clinical psychological modeling. Divisions of modeling in the clinical domain then are distinguished. Exemplary implementations are presented, as are certain challenges sui generis to this domain.

Figure 1 summarizes the overall epistemic strategy of clinical psychological modeling. In its basic form, quantitative models of normal performance, typically on laboratory tasks, are titrated to accommodate performance deviations occurring with clinical disturbance. The requisite model tweaking, analogous to a reagent of chemical titration, in principle discloses the nature of change to the task-performance system taking place with clinical disturbance. Aspects of the model remaining intact are deemed as aligning with functions spared with the disturbance, and those that have been perturbed are triaged as pointing to functions that have been affected.

Insert Figure 1 about here

Accommodation of performance deviations by the appropriated model infrastructure in turn speaks to validity of the latter. Successful accommodation of altered performance amongst clinical samples becomes a source of construct validity, over and against an appropriated model's failure, or strain in doing so. This aspect of model evaluation, an instance of "model-generalization testing" (Busemeyer & Wang, 2000), is one in which performance data from the clinical setting can play an important role.

To illustrate the above strategy, consider a typical memory-search task (Sternberg, 1969). Such a task may be appropriated to tap ecologically significant processes: cognitively preparing and transforming (encoding) environmental stimulation into a format facilitating collateral cognitive operations; extracting and manipulating material in short-term or working memory, on which informed responding rests; and preparing and delivering the information-determined response. During each trial of the above task, a pre-memorized set of items (memory set), such as alphanumeric characters, are to be scanned, in order to ascertain "as quickly and accurately as

possible" the presence or absence of a subsequently presented item (probe item). The sub-span memory-set size varies from trial to trial, with the items within each set size also possibly varying over their presentations, or alternatively remaining constant within each set size (variable- versus fixed- set procedures). Other manipulations may be directed, for instance, to increasing in probe-encoding demands (whereby say the font of the probe item mismatches, rather than matches the font of the memory set items). The principal response property tenably is latency from probe-item onset, accuracy being high, and not compromising the validity of latency-based inferences (e.g., no speed-accuracy trade-off).

Quantitatively informed convergent experimental evidence may point to an elongated probe-encoding process as being responsible for delayed trial-wise response latencies in the clinical group. The encoding process may be represented by a portion of the trial-performance model. This portion may stipulate, for example, constituent encoding operations of the encoding process, there being *k*' in number (to use model-parameter notation consistent with the clinical modeling literature). Such subprocesses may correspond with observable stimulus features, such as curves, lines and intersections of the alphanumeric probe, extracted in the service of mental template matching to members of the trial's memory set. The intensity of processing applicable to each of the respective *k*' subprocesses (loosely, speed of completion, or number transacted per unit time; e.g., Rouder, Sun, Speckman, Lu, & Ahou, 2003; Townsend & Ashby, 1983) is denoted *v*. Decomposing clinical-sample performance through the model based analysis potentially exonerates the parameter *v*, but indicts *k*' as the source of encoding protraction. Specifically, model-predicted configurations of performance latency, and inter-trial variability, converge with the pattern of empirical deviations from control data, upon elevation in *k*', but not

reduction in *v*. By way of parameter assignment in the modeling literature, strictly speaking cognitive capacity (in the sense of processing speed) is intact, but efficiency of its deployment has suffered. The above example of mapping of formal theory onto empirical data is known in clinical mathematical modeling, and elsewhere, as "abductive reasoning" (see Textbox 1).

Insert Textbox 1 about here

When it comes to potential "value added" beyond the immediate increment in information, the following come to the fore. Ecological significance is imbued in terms of assembling environmental information in the service of establishing necessary responses, including those germane to self-maintenance activities, and meeting environmental demands. On this note, basic deficit may ramify to more elaborate operations in which the affected process plays a key role (e.g., where judgments about complex multidimensional stimuli are built up from encoded constituent dimensions). Deficits in rudimentary processes moreover may parlay into florid symptom development or maintenance, as where thought-content disorder (delusions and thematic hallucinations) arise from insufficient encoding of cues that normally anchor the interpretation of other information. Additional implications pertain to memory, where heightened retrieval failure is risked owing to protraction of initial item encoding. Debility parameterization also may inform other domains of measurement, such as neuroimaging. A tenable model may demarcate selected intra-trial epochs of cognitive tasks when a clinically significant constituent process is likely to figure prominently. In this way, times of measurement interest may complement brain-regions of interest, for a more informed navigation of space-time co-ordinates

in functional neuroimaging. Symptom significance thus may be brokered to imaged neurocircuitry via formally modeled cognitive abnormalities.

Modeling Distinctions in Psychological Clinical Science

There are several versions of "modeling" in psychological clinical science. Non-formal models, such as flow-diagrams and other organizational schemata, nevertheless ubiquitously are labeled "models" (cf. McFall, Townsend & Viken, 1995). Our consideration here is restricted to formal models, where the progression of theoretical statements is governed by precisely stipulated rules of successive statement transitions. Most notable, and obviously dominating throughout the history of science are mathematical models. Formal languages for theory development other than mathematics include symbolic logic and computer syntax. Within the formal modeling enterprise, then is mathematical modeling, computational modeling [computer simulation, including "connectionist", "(neural) network", "cellular automata", and "computational informatics" modeling], and nonlinear dynamical systems modeling ("chaos-theoretic" modeling, in the popular vernacular). There is of course an arbitrary aspect to such divisions. Mathematical modeling can recruit computer computations (below), while nonlinear dynamical systems modeling entails differential equations, and so on. Like many systems of classification, the present one facilitates exposition, in this case of modeling activity within the domain of psychological clinical science. Points of contact and overlap amongst these divisions, as well as unique aspects, should become more apparent with the more detailed descriptions that follow.

The respective types of formal modeling potentially provide psychopathology-significant information unique to their specific level of analysis (Marr, 1982). They also may inform each other, and provide across--level-analysis construct validity. For example, manipulation of connectionist-model algorithm parameters may be guided by results from mathematical modeling. Connectionist modeling results, in turn, may lend construct validity to mathematical model titration (above).

Before delineating subsets of formal modeling, a word is in order about so-called statistical modeling (see, e.g., Rodgers, 2010). Statistical modeling, such as Structural-Equation Modeling, including Confirmatory Factor Analysis, Hierarchical Linear Modeling, Mixture Growth Modeling, and Taxometric Analysis ("mixture-model testing for staggered, or quasi-staggered latent distributions of clinical and non-clinical groups") supply a platform for data organization, and inferences about its resulting structure. To be sure, parameters, such as path weights and factor loadings are estimated using methods shared with formal models, as demarcated here. Contra the present emphasis, however, the format of proposed model structure (typically one of multivariate covariance), and computational methods are trans-content, generic and do not germinate within the staked out theoretical-content domain with its problem-specific depth of analysis. In the case of formal modeling, it might be said that measurement models and empirical-testing methods are part and parcel of process-models of observed responses and data production. (see also Textbox 2). Extended treatment of formal-model distinctives and assets in clinical science is available in alternate venues (e.g., Neufeld, 2007b; Neufeld, Shanahan & Townsend, 2010).

Forms of Modeling in Clinical Science.

Mathematical modeling. Clinical mathematical modeling is characterized by analytically derived accounts of cognitive-behavioral abnormalities of clinical disorders. In most instances, models are stochastic, meaning they provide for an intrinsic indeterminacy of the modeled phenomenon (not unlike Brownian motion being modeled by a Wiener process, in Physics). Doob (1953) has described a stochastic model as a "… mathematical abstraction of an empirical process whose development is governed by probabilistic laws (p. v)". Roughly, built into the structure of stochastic models is a summary of nature's perturbation of empirical values from one observation to the next. Predictions therefore by and large are directed to properties of the distributions of observations, such as those of response latencies over cognitive-task trials. Model expressions of clinical-sample deviations in distribution properties therefore come to the fore. Such properties may include summary statistics, such as distribution moments (notably means and inter-trial variances), but can also include distribution features as detailed as the distribution's probability density function (density function, for short; proportional to the relative frequency of process completion at a particular time since its commencement; see, e.g., Evans, Hastings & Peacock, 2000; Townsend & Ashby, 1983; Van Zandt, 2000).

Grasping the results of mathematical modeling's analytical developments can be challenging, but can be aided by computer computations. Where the properties of derivations are not apparent from inspection of their structures themselves, the formulae may be explored numerically. Doing so often invokes three-dimensional response surfaces. The predicted response output expressed by the formula, plotted on the *Y* axis, is examined as the formula's model parameters are varied on the *X* and *Z* axes. In the earlier example, for instance, the

probability of finalizing a stimulus- encoding process within a specified time *t* may be examined as parameters *k*' and *v* are varied.

Note in passing that expression of laws of nature typically is the purview of analytical mathematics (e.g., Newton's law of Gravity).

Computational modeling (computer simulation). Computational modeling expresses recurrent interactions (reciprocal influences) among activated, and activating units, that have been combined into a network architecture. The interactions are implemented through computer syntax (e.g., Farrell & Lewandowsky, 2010). Some of its proponents have advanced this form of modeling as a method uniquely addressing the computational capacity of the brain, and as such, have viewed the workings of a connectionist network as a brain metaphor. Accordingly, the network units can stand for neuronal entities (e.g., multi-neuron modules, or "neurodes"), whose strengths of connection vary over the course of the network's ultimate generation of targeted output values. Variation in network architecture (essentially paths and densities of inter-neurode connections), and/or connection activation, present themselves as potential expressions of cognitive abnormalities.

Exposition of neuro-connectionist modeling in clinical science has been relatively extensive (e.g., Bianchi, Caviness,& Cash, 2012; Carter & Neufeld, 2007; Hoffman & McGlashan, 2007; Phillips & Silverstein, 2003; Siegle & Hasselmo, 2002; Stein & Young, 1992). Typically directed to cognitive functioning, the connectionist network approach recently has been extended to the study of inter-symptom relations, providing an unique perspective for example on the issue of co-morbidity (Boorsboom, & Cramer, in press; Cramer, Waldorp, van der Maas & Boorsboom, 2010).

Nonlinear dynamical system ("chaos-theoretic") modeling. This form of modeling again entails inter-connected variables ("coupled system dimensions"), but in clinical science by and large these are drastically fewer in number than usually is the case with computational network modeling (hence, the common distinction between "high-dimensional networks" and "low-dimensional nonlinear networks"). Continuous interactions of the system variables over time are expressed in terms of differential equations. The latter stipulate the momentary change of each dimension at time *t*, as determined by the extant values of other system dimensions, potentially including that of the dimension whose momentary change is being specified (e.g., Fukano & Gunji, 2012). The nonlinear properties of the system can arise from the differential equations' cross-product terms, conveying continuous interactions, or nonlinear functions of individual system dimensions, such as raising extant momentary status to a power other than 1.0.

The status of system variables at time *t* is available via solution to the set of differential equations. Because of the nonlinearity-endowed complexity, solutions virtually always are carried out numerically, meaning computed cumulative infinitesimal changes are added to starting values for the time interval of interest.

System dimensions are endowed with their substantive significance according to the theoretical content being modeled (e.g., dimensions of subjective fear, and physical symptoms, in dynamical modeling of panic disorder; Fukano & Gunji, 2012). A variation on the above description comprises the progression of changes in system-variable states over discrete trials (e.g., successive husband-wife interchanges; Gottman, Murray, Swanson, Tyson & Swanson, 2002). Such sequential transitions are implemented through trial-wise difference-equations, which now take the place of differential equations.

Model-predicted trajectories can be tested against trajectories of empirical observations, as obtained say through diary methods involving on-line data-gathering sites (e.g., Qualtrics; Bolger, Davis & Rafaeli, 2008). In addition, empirical data can be evaluated for the presence of dynamical signatures ("numerical diagnostics") generated by system-equation driven, computer-simulated time series. It is possible moreover to search empirical data for time-series numerical diagnostics that are general to system complexity of a nonlinear-dynamical-systems nature. This latter endeavour, however, has been criticized when undertaken without a precise tendered model of the responsible system, ideally buttressed with other forms of modeling (notably mathematical modeling, above) – such a more informed undertaking as historically exemplified in Physics (Wagenmakers, van der Maas & Farrell, 2012).

A branch of nonlinear dynamical systems theory, "Catastrophe Theory", has been implemented notably in the analysis of addiction relapse. For example, therapeutically significant dynamical aspects of aptitude-treatment intervention procedures (e.g., Dance & Neufeld, 1988) have been identified through a Catastrophe-Theory based reanalysis of data from a large-scale multi-site treatment-evaluation project (Witkiewitz, van der Maas, Hufford & Marlatt, 2007). Catastrophe Theory offers established sets of differential equations ("canonical forms") depicting sudden jumps in nonlinear dynamical system output (e.g., relapse behaviour) occurring to gradual changes in input variables (e.g., incremental stress).

Nonlinear dynamical systems modeling-- whether originating in the content domain, or essentially imported, as in the case of Catastrophe Theory-- often may be the method of choice when it comes to macroscopic, molar clinical phenomena, such as psychotherapeutic interactions (cf. Molenaar, 2010). Extended exposition of nonlinear dynamical systems model development,

from a clinical-science perspective, including that of substantively-significant patterns of dimension trajectories ("dynamical attractors"), has been presented in Gottman, et al (2002), Levy, Yao, McGuire, Vollick, Jette, Shanahan, Hay & Neufeld (2012), and Neufeld (1999).

Model Parameter Estimation in Psychological Clinical Science

A parameter is "an arbitrary constant whose value affects the specific nature but not the properties of a mathematical expression" (Borowski & Borwein, 1989, p. 435). In modeling abnormalities in clinical samples, typically it is the values of model parameters, rather than the model structure (the model's mathematical organization) that are found to shift away from control values.

The clinical significance of the shift depends on the meaning of the parameter(s) involved. Parameters are endowed with substantive significance according to their roles in the formal system in which they are positioned, and their mathematical properties displayed therein. For example, a parameter of a processing model may be deemed to express "task-performance competence". Construct validity for this interpretation may be supported if the statistical moments of modeled performance-trial latencies (e.g., their mean or variance) are sent to the realm of infinity, barring a minimal value for the parameter. If this value is not met, a critical corpus of extremely long, or incomplete task trials ensues, incurring infinite response-latency moments in the formalized system. From a construct-validity standpoint, this type of effect on system behavior—severe performance impairment, or breakdown-- is in keeping with the parameter's ascribed meaning (Neufeld, 2007b; see also Pitt, Kim, Navarro & Myung, in press).

Added to this source of parameter information-- "analytical construct validity"-- is "experimental construct validity". Here, estimated parameter values are selectively sensitive to experimental manipulations, diagnostic-group differences, or variation in psychometric measures on which they purportedly bear (e.g., additional constituent operations of a designated cognitive task, other demands being equal, resulting in elevation specifically in the parameter *k*', above).

In clinical science and assessment, values of model parameters to a large extent are estimated from empirical data. Such estimation differs from methods frequently used in the physical sciences, which more often have the luxury of direct measurement of parameter values (e.g., measuring parameters of liquid density and temperature, in modeling sedimentation in a petroleum-extraction tailings pond). Parameter estimation from the very empirical data being modeled -- of course with associated penalization in computations of empirical model fit -- however, is only superficially suspect. As eloquently 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)

How, then might parameter values be estimated, with an eye to possible constraints imposed in the clinical arena? Multiple methods of parameter estimation in clinical science variously have been used, depending on desired statistical properties (e.g., "maximum likelihood", "unbiasedness", "Bayes"; see, e.g., Evans, et al, 2000) and data-acquisition constraints. Note that selection of parameter estimation methods is to be distinguished from methods of selecting from among competing models, or competing model variants (for issues of model selection, see especially Wagenmakers & Vandekerckhove, this volume).

Moment matching. One method of parameter estimation consists of moment matching. Moments of some stochastic distributions can be algebraically combined to provide a direct estimates. For example, the mean of the Erlang distribution of performance-trial latencies, expressed in terms of its parameter implementation k' and v, above, is k'/v (e.g., Evans, et al, 2000). The inter-trial variance is k'/v^2. From these terms, an estimate of v is available as the

empirical mean divided by the empirical variance, and an estimate of *k*' is available as the mean, squared, divided by the variance.

Maximum likelihood. Maximum-likelihood parameter estimation means initially writing a function expressing the likelihood of obtained data, given the data-generation model. The maximum-likelihood estimate is the value of the model parameter that would make the observed data maximally likely, given that model.

Maximum likelihood estimates can be obtained analytically, by differentiating the written likelihood function with respect to the parameter in question, setting it to 0, and solving (the second derivative being negative). For example, the maximum-likelihood estimate of *v* in the Erlang distribution, above, is $\frac{Nk'}{\sum_{i=1}^{N} t_i}$, where the size of the empirical sample of latency values t_i is *N*. With multiple parameters, such as *v* and *k*', the multiple derivatives are set to 0, followed by solving simultaneously.

As model complexity increases, with respect to number of parameters, and possibly model structure, numerical solutions may be necessary. Solutions now are found by computationally searching for the likelihood-function maximum, while varying constituent parameters iteratively and reiteratively. Such search algorithms are available through *R*, through the MATLAB OPTIMIZATIOMN TOOLBOX, computer-algebra programs, such as Waterloo MAPLE, and elsewhere.

As with other methods that rely exclusively on the immediate data, stability of estimates rests in good part on extensiveness of data acquisition. Extensive data acquisition say on a cognitive task, possibly amounting to hundreds or even thousands of trials, obtained over

multiple sessions, may be prohibitive when it comes to distressed patients. For these reasons, Bayesian estimates may be preferred (below).

Moment fitting and related procedures. Another straightforward method of parameter estimation is that of maximizing the conformity of model-predicted moments (typically means and inter-trial standard deviations, considering instability of higher-order empirical moments; Ratcliff, 1979), across performance conditions and groups. An unweighted least-squares solution minimizes the sum of squared deviations of model predictions from the empirical moments. Although analytical solutions are available in principle (deriving minima using differential calculus, similar to deriving maxima, in the case of maximum-likelihood solutions), use of a numerical search algorithm most often is the case.

Elaborations on unweighted least-squares solutions include minimization of Pearson χ^2, where data are response-category frequencies fr_i:

$$\min \left(\sum_{i=1}^{p} \frac{\left(fr_{i,\,observed} - fr_{i,\,model-predicted} \right)^2}{fr_{i,\,model-predicted}} \right),$$

where there are p categories. Note that frequencies can include those of performance-trial latencies falling within successive time intervals ("bins").

Where the chief data are other than categorical frequencies, as in the case of moments, parameter values may be estimated by constructing and minimizing a pseudo- χ^2 function. Here, the theoretical and observed frequencies are replaced with the observed and theoretical moments (Townsend, 1984; Townsend & Ashby, 1983, chap. 13). As with the Pearson χ^2, the squared differences between the model-predicted and observed values are weighted by the inverse of the model-predicted values.

It is important to take account of logistical constraints when it comes to parameter-estimation and other aspects formal modeling in the clinical setting. In this setting, where available data can be sparse, the use of moments may be necessary for stability of estimation. Although coarse, as compared to other distribution properties, moments encompass the entire distribution of values, and can effectively attenuate estimate-destabilizing noise (cf. Neufeld & Gardner, 1990).

Observe that along with likelihood maximization, the present procedures are an exercise in function optimization. In the case of least-squares and variants thereon, the exercise is one of function minimization. In fact, often an unweighted least-squares solution also is maximum likelihood. Similarly, where likelihood functions enter χ^2 or approximate-χ^2 computations ("Likelihood-Ratio G^2), the minimum χ^2 is also maximum likelihood.

Evaluation of the adequacy of parameter estimation amidst constraints intrinsic to psychological clinical science and assessment ultimately rests with empirical tests of model performance. Performance obviously will suffer with inaccuracy of parameter estimation.

Bayesian parameter estimation. Recall that Bayes' Theorem states that the probability of an estimated entity *A*, given entity-related evidence *B* ("posterior probability of *A*") is the pre-evidence probability of *A* (its "prior probability") times the conditional probability of *B*, given *A* ("likelihood"), divided by the unconditional probability of *B* ("normalizing factor"):

$$Pr(A|B) = \frac{Pr(A)Pr(B|A)}{Pr(B)} . \qquad (1)$$

As applied to parameter estimation, *A* becomes the candidate parameter value θ, and *B* becomes data *D* theoretically produced by the stochastic model in which θ participates. Recognizing θ as continuous, (1) becomes

$$g(\theta|D) = \frac{f(\theta)Pr(D|\theta)}{\int_{-\infty}^{+\infty} f(\theta)Pr(D|\theta)d\theta} \quad , \qquad (2)$$

where *g* and *f* denote density functions, over and against discrete-value probability *Pr*. The data *D* may be frequencies of response categories, such as correct versus incorrect item recognition.

Note that the data *D* as well may be continuous, as in the case of measured process latencies. If so, the density functions again replace discrete-value probabilities. For an Erlang distribution, for instance, the density function for a latency datum t_i is

$$\frac{(vt_i)^{k'-1}}{(k'-1)!} v e^{-vt_i} .$$

Allowing *k*' to be fixed, for the present purposes of illustration (e.g., directly measured, or set to 1.0, as with the exponential distribution), and allowing θ to stand for *v*, then for a sample of *N* independent values,

$$Pr(D|\theta)$$

in (2) becomes the joint conditional density function of the *N* t_i values, given *v* and *k*',

$$\prod_{i=1}^{N} \frac{(vt_i)^{k'-1}}{(k'-1)!} v e^{-vt_i} .$$

The posterior density function of θ [e.g., (2)] can be computed for all candidate values of θ, the tendered estimate then being the mean of this distribution (the statistical property of this estimate is termed "Bayes"; see especially Kruschke & Vanpaemel, this volume). If an individual participant or client is the sole source of *D*, the Bayes estimate of θ de facto has been individualized accordingly.

Bayesian parameter estimation potentially endows clinical science and practice with demonstrably important advantages. Allowance for individual differences in parameter values can be built into the Bayesian architecture, specifically in terms of the prior distribution of performance-model parameters (Batchelder, 1998). In doing so, the architecture also handles the issue of "over-dispersion" in performance-data, meaning greater variability than would occur to fixed parameter values for all participants (Batchelder & Riefer, 2007).

Selecting a prior distribution of θ depends in part on the nature of θ. Included are "strong priors", such as those from the Generalized Gamma family (Evans,et al, 2000) where $0 \leq \theta$; the Beta distribution, where $0 \leq \theta \leq 1.0$; and the normal or Gaussian distribution. Included as well are "gentle" (neutral) priors, notably the uniform distribution, whose positive height spans the range of possible non-0 values of θ. (see also Berger, 1985, for Jeffreys' uninformed, and other prior distributions). Grounds for prior-distribution candidacy also includes "conjugation" with the performance process model. The practical significance of distributions being conjugate essentially is that the resulting posterior distribution becomes more mathematically tractable, allowing a closed-form solution for its probability density function.

Defensible strong priors (defensible on theoretical grounds, and those of empirical model fit) can add to the arsenal of clinically significant information, in and of themselves. To illustrate, the Bayesian prior distribution of θ values has its own governing parameters ("hyper-parameters"). Hyper-parameters, in turn, can be substantively significant within the addressed content domain (e.g., the task-wise competence parameter, described at the beginning of this section; or another expressing psychological-stress effects on processing speed;

a).

Because of the information provided by a strong Bayesian prior, the estimate of θ can be more precise and stable in the face of a smaller data set *D*, than would be necessary when the estimate falls entirely to the data at hand (see, e.g., Batchelder, 1998). This variability-reducing influence on parameter estimation is known as "Bayesian shrinkage" (e.g., O'Hagan & Forster, 2004). Bayesian shrinkage can be especially valuable in the clinical setting, where it may be unreasonable to expect distressed participants or patients to offer up more than a modest reliable specimen of task performance. Integrating the performance sample with the prior-endowed information is analogous to referring a modest blood sample to the larger body of haematological knowledge in the biomedical assay setting. Diagnostic richness of the proffered specimen is exploited because its composition is subjected to the pre-existing body of information that is brought to bear. Other potentially compelling Bayesian-endowed advantages to clinical science and assessment are described in the section, **Special Considerations Applying to Mathematical and Computational Modeling in Psychological Clinical Science and Assessment**, below. (See also Textbox 2 for an unique method of assembling posterior density functions of parameter values to ascertain the probability of parameter differences between task-performance conditions.)

Illustrative Examples of Contributions of Mathematical Psychology to Clinical Science and Assessment.

Information conveyed by rigorous quantitative models of clinically significant cognitive-behavioral systems is illustrated in the following examples. Focus is on uniquely disclosed

aspects of system functioning. Results from generic data-theory empirical analyses, such as group by performance-conditions statistical interactions, are elucidated in terms of their modeled performance-process underpinnings. In addition to illuminating results from conventional analyses, quantitative modeling can uncover group differences in otherwise conflated psychopathology-germane functions (e.g., Chechile, 2007; Riefer, Knapp, Batchelder, Bamber & Manifold, 2002). Theoretically unifying seemingly dissociated empirical findings through rigorous dissection of response configurations represents a further contribution of formal modeling to clinical science and assessment (White, Ratcliff, Massey & McKoon, 2010a).

Moreover, definitive assessment of prominent conjectures on pathocognition is availed through experimental paradigms exquisitely meshing with key conjecture elements (Johnson, Blaha, Houpt & Townsend, 2010). Such paradigms emanate from models addressing fundamentals of cognition, and carry the authority of theorem-proof continuity, and closed-form predictions (Townsend & Nozawa, 1995; see also Townsend & Wenger, 2004a).

At the same time, measures in common clinical use have not been left behind (e.g., Yechiam, Veinott, Busemeyer, & Stout, 2007). Mathematical modeling effectively has quantified cognitive processes at the root of performance on measures such the Wisconsin Card Sorting Test, the Iowa Gambling Task, and the Go—No-Go task (taken up under *Cognitive modeling of routinely used measures in clinical science and assessment*, below).

Further, formal models of clinical-group cognition can effectively inform findings from clinical neuroimaging. Events of focal interest in "event-related imaging" are not the within- nor between-trial transitions of physical stimuli embedded in administered cognitive paradigms, but

rather the covert mental processes to which such transitions give rise. Modeled stochastic trajectories of the symptom-significant component processes that transact cognitive performance trials can stipulate intra-trial epochs of special neuroimaging interest. The latter in can complement brain regions of interest, together facilitating the calibration of space-time measurement co-ordinates in neuroimaging studies.

Multinomial processing tree modeling of memory and related processes; unveiling and elucidating deviations among clinical samples. Possibly the most widely used mathematical modeling in clinical psychology is Multinomial Processing Tree Modeling (MPTM). Essentially MPTM models the production of categorical responses, such as recall or recognition of previously studied items, or judgments of items as to their earlier source of presentation (e.g., auditory versus visual-- potentially bearing on the nature of hallucinations; Batchelder & Riefer, 1990; 1999).

Responses in such categories are modeled as having emanated from a sequence of stages, or processing operations. For example, successful recall of a pair of semantically linked items, such as “computer, internet”, entails storage and retrieval of the duo, retrieval itself necessarily being dependent on initial storage. The tree in MPTM consists of branches emanating from nodes; processes branching from nodes proceed from other processes, on which the branching process is conditional (as in the case of retrieving a stored item).

Each process in each successive branch has a probability of successful occurrence. The probability of a response in a specific category (e.g., accurate retrieval of an item pair) having

taken place through a specific train of events, is the product of the probabilities of those events (e.g., probability of storage times the conditional probability of retrieval, given storage).

Parameters conveying the event probabilities are viewed as "capacities of the associated processes". Criteria for success of constituent processes implicitly are strong, in that execution of a process taking place earlier in the branching is a sufficient precondition for the process being fed; the probability of failure of the subsequent process falls to that process itself. In this way, MPTM isolates functioning of the individual response-producing operations, and ascertains deficits accordingly.

The model structure of MPTM is conceptually tractable, thanks to its straightforward processing tree diagrams. It has, however, a strong analytical foundation, and indeed has spawned innovative methods of parameter estimation (Chechile, 1998), and notably rigorous integration with statistical science (labelled "cognitive psychometrics"; e.g., Batchelder, 1998; Riefer, et al, 2002). Computer software advances have accompanied MPTM's analytical developments (see Moshagen, 2010, for current renderings). Exposition of MPTM measurement technology has been substantial (e.g., Batchelder & Riefer, 1990; Chechile, 2004; Riefer & Batchelder, 1988), including that tailored to clinical-science audiences (Batchelder, 1998; Batchelder & Riefer, 2007; Chechile, 2007; Riefer, et al, 2002; Smith & Batchelder, 2010).

Issues in clinical cognition potentially form natural connections with the parameters of MTM. Just as the categories of response addressable with MPTM are considerable, so are the parameterized constructs it accommodates. In addition to essential processes of memory, perception, and learning, estimates are available for the effects of guessing, and for degrees of

participants' confidence in responding. Moreover, MPTM has been extended to predictions of response latency (Hu, 2001; Schweickert, 1985; Schweickert & Han, 2012).

Riefer, et al (2002) applied MPTM in two experiments, to decipher the nature of recall performance among schizophrenia and brain-damaged alcoholic participants. In each experiment, the clinical group and controls (nonpsychotic patients, and non–organic-brain-syndrome alcoholics) received six trials of presentation and study of semantically-related item pairs (above), each study period being followed by recall of items in any order. In both experiments, the research design comprised a "correlational experiment" (Maher, 1970). A correlational experiment consists of the diagnostic groups under study performing under multiple conditions of theoretical interest – a prominent layout in psychological clinical science (e.g., Yang, Tadin, Glasser Glasser, Hong & Park, 2013).

Initial analyses of variance (ANOVA) were conducted on sheer proportions of items recalled. In each instance, significant main effects of groups and study-recall trials were obtained; a statistically significant trials by groups interaction, the test of particular interest, was reported only in the case of the brain-damaged alcoholic participants and their controls (despite liberal degrees of freedom for within-subjects effects). As noted by Riefer, et al. (2002), this generic empirical analysis betrayed its own shortcomings for tapping potentially critical group differences in faculties subserving recall performance. Indeed, Riefer, et al simply but forcefully showed how reliance on typical statistical treatments of data from correlational experiments can generate demonstrably misleading inferences about group differences in experimentally addressed processes of clinical and other interest.

Riefer, et al's theory-disciplined measures precisely teased apart storage and recall-retrieval processes, but went further in prescribing explicitly theory-driven significance tests on group differences (see also Link, 1982; Link & Day, 1992; Townsend, 1984). Pursuant to the superficially parallel group performance changes across trials, schizophrenia participants failed to match the controls in improvement of storage efficiency specifically over the last 3 trials of the experiment. Moreover, analysis of a model parameter distinguishing rate of improvement in storage accuracy as set against its first-trial "baseline", revealed *greater* improvement among the schizophrenia participants during trials 2 and 3, but a decline relative to controls during the last 3 trials. In other words, this aspect of recall-task performance arguably was decidedly spared by the disorder, notably during the initial portions of task engagement. Analysis of the model parameter distinguishing rate of improvement in retrieval, as set against its first-trial baseline now indicated a significantly slower rate among the schizophrenia participants throughout. The interplay of these component operations evidently was lost in the conventional analysis of proportion of items recalled. It goes without saying that precisely profiling disorder-spared and affected aspects of functioning, as exemplified here, can inform the navigation of therapeutic intervention strategies. It also can round out the "functional" picture brought to bear on possible functional neuroimaging measurement obtained during recall task performance.

Applying the substantively-derived measurement model in the study on alcoholics with organicity yielded potentially important processing specifics buried in the nevertheless now significant groups-by-trials ANOVA interaction. As in the case of schizophrenia, the brain-damaged alcoholic participants failed to approximate the controls in improvement of storage

operations specifically over the last 3 trials of the experiment. Also, significant deficits in retrieval again were observed throughout. Further, the rate of improvement in retrieval, relative to the trial-1 baseline, more or less stalled over trials. In contrast to retrieval operations, the rate of improvement in storage among the sample of alcoholics with organicity kept up with that of controls—evidently a disorder-spared aspect of task execution.

The mathematically derived performance-assessment MPTM methodology demonstrably evinced substantial informational added-value, in terms of clinically significant measurement and explanation. Multiple dissociation in spared and affected elements of performance was observed within diagnostic groups, moreover with further dissociation of these patterns across groups.

Estimates of model parameters in these studies were accompanied by estimated variability therein (group and condition-wise standard deviations). Inferences additionally were strengthened with flanking studies supporting construct validity of parameter interpretation, according to selective sensitivity to parameter-targeted experimental manipulations. In addition, validity of parameter-estimation methodology was attested to through large-scale simulations, which included provision for possible individual differences in parameter values (implemented according to “hierarchical mixture structures”).

In like fashion, Chechile (2007) applied MPTM to expose disorder-affected memory processes associated with developmental dyslexia. Three groups were formed according to psychometrically identified poor, average, and above average reading performance. Presented items consisted of sixteen sets of 6 words, some of which were phonologically similar (e.g., blue,

zoo), semantically similar (bad, mean), orthographically similar (slap, pals), or dissimilar (e.g., stars, race). For each set of items, 6 pairs of cards formed a 2 by 6 array. The top row consisted of the words to be studied, and the second row was used for testing. A digit-repetition task intervened between study and test phase, controlling for rehearsal of the studied materials. Testing included that of word-item recall, or word position, in the top row of the array. For recall trials, a card in the second row was turned over revealing a blank side, and the participant was asked what word was in the position just above. For recognition trials, the face-down side was exposed to reveal a word, with the participant questioned as to whether that word was in the corresponding position, in the top row. In some instances, the exposed word was in the corresponding position, and in others it was in a different position.

A 6-parameter MPTM model was applied to response data arranged into 10 categories. The parameters included 2 storage parameters, one directed to identification of a previous presentation, and one reserved for the arguably deeper storage required to detect foils (words in a different position); a recall-retrieval parameter; 2 guessing parameters, and a response-confidence parameter. As in the case of Riefer, et al (2002), parameterized memory-process deviations proprietary to model implementation were identified.

Apropos of the present purposes, a highlight among other noteworthy group differences occurred as follows. Compared to above-average readers, performance data of poor readers produced lower values for the previous-presentation storage parameter, but higher values for recall retrieval. This dissociation of process strength and deficit was specific to the orthographically similar items. The above inferences moreover were supported with a decidedly

model-principled method of deriving the probability of two groups differing in their distributions of parameter values (see Textbox 2).

The reciprocal effects of stronger retrieval and weaker storage on the poor readers' performance with the orthographically similar items, evinced a non-significant difference from the above-average readers on raw recall scores ($p > .10$). Without the problem-customized measurement model, not only would group differences in memory functions have gone undetected, but the nature of these differences would have remained hidden. Again, a pattern of strength and weakness, and the particular conditions to which the pattern applied (encountering orthographically similar memory items), were powerfully exposed.

In this report as well, findings were fortified with model-validating collateral studies. Selective sensitivity to parameter-targeting experimental manipulations lent construct validity to parameter interpretation. In addition, the validity of the model structure hosting the respective parameters was supported with preliminary estimation of coherence between model properties and empirical data [*Pr*(*coherence*); Chechile, 2004]. Parameter recovery as well was ascertained through simulations for the adopted sample size. Furthermore, parameter estimation in this study employed an innovation developed by Chechile, called "Population Parameter Mapping" (detailed in Chechile, 1998; 2004; 2007).[1]

Unification of disparate findings on threat-sensitivity among anxiety-prone individuals through a common-process model. Random-walk models (Cox & Miller, 1965) are stochastic mathematical models that have a rich history in cognitive theory (e.g., Busemeyer & Townsend, 1993; Link & Heath, 1975; Ratcliff, 1978; see Ratcliff & Smith, this volume). Diffusion

modeling (Ratcliff, 1978) presents itself as another form of modeling shown to be of

Type equation here.extraordinary value to clinical science. This mathematical method allows the dissection of decisional performance into component processes that act together to generate choice responses and their latencies (Ratcliff, 1978). Application of diffusion modeling has been used to advantage in simplifying explanation, and in unifying observations on sensitivity to threat-valenced stimulation among anxiety-prone individuals (White, Ratcliff, Vasey & McKoon, 2010a; for diffusion-model software developments, see Wagenmakers, van der Maas, Dolan & Grasman, 2008).

Increased engagement of threatening stimulus content (e.g., words such as punishment, or accident) among higher anxiety-prone (HA) as compared to lower anxiety-prone (LA) individuals has been demonstrated across multiple paradigms (e.g., the Stroop and dichotic listening tasks; and the dot-probe task, where for HA individuals, detection of the probe is disproportionately increased with its proximity to threatening versus neutral items in a visual array).

Consistency of findings of significant HA-LA group differences, however, by and large has depended on presentation of the threat items in the company of non-threat items. Such differences break down when items are presented singly. Specifically, the critical HA-LA by threat—non-threat item interaction (group-item second-order difference, or two-way interaction) has tended to be statistically significant specifically when the two types of stimuli have occurred together. This pattern of findings has generated varyingly complex conjectures as to responsible

agents. The conjectures have emphasized processing competition between the two types of stimuli, and associated "cognitive control operations". Group differences have been attributed, for example, to differential tagging of the threat items, or to H-A participant threat-item disengagement deficit (reviewed in White, et al, 2010a).

The difficulty in obtaining significant second order-differences with presentation of singletons has led some investigators to question the importance, or even existence, of heightened threat-stimulus sensitivity as such among HA individuals. Others have developed neuro-connectionst computational models (e.g., Williams & Oaksford, 1992), and models comprising neuro-connectionist computational-analytical amalgams (Frewen, Dozois, Joanisse & Neufeld, 2008), expressly stipulating elevated threat sensitivity among HA individuals.[2] If valid, such sensitivity stands to ramify into grosser clinical symptomatology (Neufeld & Broga, 1981).

Greater threat-stimulus sensitivity defensibly exists among HA individuals; but for reasons that are relatively straightforward, such sensitivity may be more apparent in the company of non-threat items, as follows. The cognitive system brought to bear on the processing task stands to be one of limited capacity (see Houpt & Townsend, 2012; Townsend & Ashby, 1983; Wenger & Townsend, 2000). When items are presented together, a parallel processing structure arguably is in place for both HA and LA participants (Neufeld & McCarty, 1994; Neufeld, et al, 2007). With less pre-potent salience of the threat item for the LA participants, more attentional capacity potentially is drawn off by the non-threat item, attenuating their difference in processing latency between the two items. A larger inter-item difference would occur for the HA

participants, assuming their greater resistance to the erosion of processing capacity away from the threat item (see White, et al, 2010a, p. 674).

This proposition lends itself to the following simple numerical illustration. We invoke an independent parallel, limited-capacity processing system (IPLC), and exponentially-distributed item-completion times (Townsend & Ashby, 1983). Its technical specifics aside, the operation of this system makes for inferences about the present issue that are easy to appreciate. The resources of such a system illustratively are expressed as a value of 10 arbitrary units (essentially, the *rate* per unit time at which task elements are transacted), for both HA and LA participants. In the case of a solo presentation, a threat item fully engages the system resources of an HA participant, and ninety percent thereof in the case of an LA participant. The solo presentation of the non-threat item engages fifty percent of the system's resources for both participants. By the IPLC model, the second-order difference in mean latency then is (1/10 – 1/9) – 0 = -.0111 (that is, latency varies inversely as capacity, expressed as a processing-rate parameter).

Moving to the simultaneous-item condition, eighty percent of system processing resources hypothetically are retained by the threat item in the case of the HA participant, but are evenly divided in the case of the LA participant. The second-order difference now is (1/8-1/5) - (1/2-1/5) = -.375. Statistical power obviously will be greater for an increased statistical effect size accompanying such a larger difference. [3]

It should be possible nevertheless to detect the smaller effect size for the single-item condition, with a refined measure of processing. On that note, the larger second-order difference

in raw response times attending the simultaneous item condition, above, itself may be attenuated due to the conflation of processing time with collateral cognitive activities, such as item encoding and response organization and execution. On balance, a measure denuded of such collateral processes may elevate the statistical effect size of the solo-presentation second-order difference at least to that of the paired-presentation raw reaction time.

Such a refined measure of processing speed per se was endowed by the diffusion model as applied by White, et al (2010a) to a lexical decision task (yes-no as to whether presented letters form a word). Teased apart were speed of central decisional activities (diffusion-model drift rate), response style (covert accumulation of evidence pending a decision), bias in favor of stating the presence of an actual word, and encoding (initial preparation and transformation of raw stimulation). Analysis was directed to values for these parameters, as well as to those of raw latency and accuracy.

In three independent studies, analysis of drift rates consistently yielded significant group—item-type second-order differences, while analysis of raw latency and accuracy rates consistently fell short. The significant second-order difference also was parameter-selective, being restricted to drift-rate values, even when manipulations were conducive to drawing out possible response-propensity differences.[4]

Here too, findings were buttressed with supporting analyses. Included was construct-validity augmenting selective parameter sensitivity to parameter-directed experimental manipulations. A further asset accompanying use of this model is its demonstrable parametric economy, in the following way. Parameter values have been shown to be uncorrelated, attesting

to their conveyance of independent information. Information also is fully salvaged inasmuch as both correct and incorrect response times are analyzed (see also Link, 1982).

Parameter estimates were accompanied by calculations of their variability (standard deviations and ranges), for the current conditions of estimation. Diagnostic efficiency statistics (sensitivity, specificity and positive and negative predictive power) were used to round out description of group separation on the drift-rate, as well as raw data values, employing optimal cut-off scores for predicted classification. In each instance, the drift-rate parameter decidedly outperformed the latency mean and median, as well as raw accuracy. These results were endorsed according to signal-detection analysis, where the "signal" was the presence of higher anxiety proneness.

Altogether, the above developments make a strong case for model-delivered parsimony. Seemingly enigmatic and discordant findings are shown to cohere, as products of a common underlying process.

Measurement technology emanating from theoretical first principles: Assessing fundamentals of cognition in autism spectrum disorders. As averred by Meehl (1978; quotation at the outset of this chapter), measurement technology emanating from formal theory of longer-established disciplines has emerged from the formal theory itself [writ large in the currently prominent Higgs-boson directed Large Hadron Collider; Close (2011); see McFall & Townsend (1998) for a still-current update of Meehl's appraisal of measurement methods in clinical-science]. Systems Factorial Technology (SFT; Townsend & Nozawa, 1995; see also Townsend & Wenger, 2004a, and Chapter 3, this volume) comprises such a development in cognitive

science, and has been used to notable advantage in clinical cognitive science (Johnson, et al, 2010; Neufeld, et al, 2007; Townsend, Fific & Neufeld, 2007). Identifiability of fundamentals of cognition has been disclosed by a series of elegant theorem-proof continuities addressed to temporal properties of information processing (see Townsend & Nozawa, 1995 for details; see also Townsend & Altieri, 2012, for recent extensions incorporating the dual response properties of latency and accuracy). The axiomatic statements from which the proofs emanate, moreover, ensure that results are general, when it comes to candidate distributions of processing durations: continuity of underlying population distributions is assumed, but results transcend particular parametric expressions thereof (e.g., exponential, Weibull, etc.; see e.g., Evans, et al, 2000). The distribution-general feature is particularly important because it makes for robustness across various research settings, something especially to be welcomed in the field of clinical science.

Elements of cognitive functioning exposed by SFT include: (1), the architecture, or structure of the information-processing system; (2), the system's cognitive workload capacity; (3), selected characteristics of system control; and, (4), independence versus interdependence of constituent cognitive operations carried out by system components. Architecture pertains to whether the system is designed to handle task constituents concurrently (in parallel channels), or successively, in a serial fashion (e.g., encoding curves, lines and intersections of alphanumeric characters, simultaneously, or sequentially). Within the parallel division, moreover, alternate versions can be entertained. The channels can function as segregated units, with the products of their processing remaining distinct from each other in task completion (regular parallel architecture). Or the channels can act as tributaries to a common conduit that receives and

conveys the sum of their contributions, dispatching the collective toward task finalization (co-active parallel architecture).

Cognitive workload capacity is estimated in SFT through an index related to work and energy in physics (Townsend & Wenger, 2004b). The index registers the potential of the system to undertake cognitive transactions per unit time [analogous to the rate of dispatching Shannon-Weaver (1949) bits of information]. Third, an important aspect of system control entails cessation of processing upon sufficiency for informed responding, over and against extra-criterial continuation (operative stopping rules). Fourth, independence versus interdependence of system components refers to presence versus absence of either mutual facilitation, or cross-impedance of system channels devoted to discrete task constituents (e.g., channels handling separate alphanumeric items, or possibly item features).

Importantly, SFT mathematically disentangles these key elements of cognition. For example, cognitive-workload capacity is isolated from stopping rules and system architecture. Such elements are conflated in macroscopic speed and/or accuracy, whose relative resistance to increased task load (e.g., added items of processing; or concomitant-secondary versus single-task requirements) typically is taken to indicate system capacity (see Neufeld, et al, 2007). Disproportionate change in such behavioral data may occur, however, for reasons other than limitation in system workload capacity. Uneconomical stopping rules may be at work, such as exhaustive processing (task constituents on all system channels are finalized), when self-terminating processing will suffice (informed responding requires completion of only one, or a subset, of task constituents). It also is possible that healthy participants' seemingly greater

workload capacity actually is attributable to a more efficient architecture (e.g., the presence of co-active parallel processing).

This quantitatively disciplined measurement infrastructure takes on increased significance for clinical cognitive science, when it is realized that certain highly prominent constructs therein align with cognitive elements measured by SFT. Especially noteworthy in the study of schizophrenia, for example, is the construct of cognitive capacity (see, e.g., Neufeld, et al, 2007). In addition, system-control stopping rules impinge on so-called executive function, a construct cutting across the study of multiple disorders. Implicated by cognitive-control stopping rules are cognitive-resource conservation, and the robustness of selective inhibition.

It should not go unnoticed that SFT fundamentals of cognition also are at the heart of the "automatic-controlled processing" construct[2]. This construct arguably trumps all others in cognitive-clinical-science frequency of usage.

In identifying variants of the cognitive elements enumerated above, the stringent mathematical developments of SFT are meshed with relatively straightforward experimental manipulations, illustrated as follows. In the study of processing mechanisms in autism spectrum disorder (ASD), Johnson, et al (2010) instantiated SFT as "double factorial technology" (Townsend & Nozawa, 1995). A designated visual target consisted of a figure of a right-pointing arrow in a visual display. Manipulations included the presence or absence of such a figure. The target figure could be present in the form of constituent items of the visual array being arranged into a pattern forming a right-pointing arrow (global target), the items themselves consisting of right-pointing arrows (local target), or both (double target).

This manipulation is incorporated into quantitative indexes discerning the nature of system workload-capacity. The specific target implementation appropriated by Johnson, et al, is ideally suited to the assessment of processing deviation in ASD, because prominent hypotheses about ASD cognitive performance hold that more detailed (read "local") processing is favored.

An additional mathematical-theory driven manipulation entails target salience in the double target condition. The right-pointing item arrangement can be of high or low salience, as can the right-pointing items making up the arrangement, altogether resulting in four factorial combinations. The combinations, in lockstep with SFT's mathematical treatment of associated processing-latency distributions, complement the capacity analysis, above, by discerning competing system architectures, stopping rules, and in(inter)dependence of processing channels handling the individual targets.

A microanalysis of task-performance latency distributions (errors being homogeneously low for Johnson, et al's both ASD and control participants) was undertaken via the lens of systems factorial assessment technology.[5] Mathematically authorized signatures of double- over and against single-target facilitation of processing ("redundancy gain") was in evidence for all ASD and control participants alike. This aspect of processing evidently was spared with the occurrence of ASD.

Contra prominent hypotheses, above, all ASD participants displayed a speed advantage for global over local target processing. In contrast, 4 of the controls exhibited a local target advantage, or approximate equality of target speed. On balance, the verdict from quantitatively-disciplined diagnostics was that this property of performance was directly opposite to that

predicted by major hypotheses about ASD cognitive functioning. At minimum, a global-target processing advantage was preserved within this ASD sample.

Less prominent in the literature have been conjectures about cognitive control in ASD. However, exhaustive target processing was detected as potentially operative among 5, and definitively operative for 2 of the sample of 10 ASD participants (one case being inconclusive). In contrast, for a minority of the 11 controls – 4 in number -- exhaustive processing was either possibly or definitively operative. The analysis therefore revealed that post-criterial continuation of target processing (with possible implications for preservation of processing resources, and the processing apparatus' inhibition mechanism) may be disorder affected. System workload capacity, chronometrically measured in its own right, nevertheless was at least that of controls--an additional component of evidently spared functioning.

Observed violations of selective influence of target-salience manipulations, notably among the control participants, indicated the presence of cross target-processing-channel interactions. The violations impelled the construction of special performance-accommodating theoretical architectures. Certain candidate structures thus were mandated by the present clinical-science samples. The upshot is an example where clinical cognitive science reciprocates to non-clinical cognitive science, in this case by possibly hastening the uncovering of potentially important structures in human cognition.

Insert Textbox 2 about here

Cognitive modeling of routinely used measures in clinical science and assessment.

Measurement in clinical science and assessment frequently has been addressed to the important cognitive-behavioral domain of decision and choice. Examples include the assembling of physical objects based on a judged organizing principle, executing risky gambles, and withholding versus emitting a response to a presenting cue. These decision-choice scenarios are instantiated in the Wisconsin Sorting Test (WCST; Berg, 1948), which targets frontal lobe "executive function"; the Iowa Gambling Task (Bechara, Damasio, Damasio & Anderson, 1994), which is directed to decisions potentially abetted by accompanyingl affect; and the Go/No-Go Discrimination Task (see, e.g., Hoaken, Shaughnessy & Pihl, 2001), which is thought to engage inhibitory aspects of "cognitive control". Deficits in decisional operations are poised to be ecologically consequential, when it comes to social, occupational and self-maintenance activities [see Neufeld & Broga, 1981, for a quantitative portrayal of "critical" (versus "differential") deficit, a concept recently relabelled "functional deficit", e.g., Green, Horan & Sugar, 201].

The Expectancy Valence Learning Model (EVL; Busemeyer & Myung, 1992; Busemeyer & Stout, 2002; Yechiam, Veinott, Busemeyer, & Stout, 2007; see also Bishara, Kruschke, Stout, Bechara, McCabe & Busemeyer, 2010, and Fridberg, Queller, Ahn, Kim, Bishara, Busemeyer, Porrino & Stout, 2010, for related sequential learning models) is a stochastic dynamic model (see Busemeyer & Townsend, 1993) that supplies a formal platform for interpreting performance on such measurement tasks. The model expresses the dynamics of decisional behaviors in terms of the progression of expected values accrued by task alternatives, as governed by the record of outcomes rendered by choice-responses to date.

Dynamic changes in alternative-expectations are specified by the model structure, in which are embedded the psychological forces – model parameters—operative in generating selection likelihoods at the level of constituent selection trials. Parameters of the EVL model deliver notable psychometric "added value" when it comes to the interpretation and clinical-assessment utility of task-measure data.

Model parameters tap "motivational", "learning" and "response" domains of decision and choice. The first ascertains relative sensitivity to positive versus negative outcomes to selected alternatives (e.g., payoff versus loss to a card-selection gamble). A second performance parameter encapsulates the comparative strength of more versus less recent choice outcomes in influencing current responses. And the third parameter conveys the degree to which responding is governed by accumulated information, as opposed to momentary, ephemeral influences (informational grounding, versus impulsivity). The model therefore contextualizes the dynamics of choice responding in terms of rigorously estimated, psychologically meaningful constructs.

The EVL model, and its closely aligned sequential learning derivatives (e.g., Ahn, Busemeyer, Wagenmakers & Stout, 2008), have successfully deciphered sources of choice-selection abnormalities in several clinical contexts. Studied groups have included those with Huntington's and Parkinson's Disease (Busermeyer & Stout, 2002), Bipolar Disorder (Yechiam, Hayden, Bodkins, O'Donnell & Hetrick, 2008), and various forms of substance abuse (Bishara, et al, 2010; Fridberg, et al, 2010; Yechiam, et al, 20007), and autism spectrum disorders (Yechiam, Arshavsky, Shamay Tsoory, Yanov & Aharon, 2010). Attestation to the value of EVL analyses, among other forms, has been that of multiple dissociation of parameterized abnormalities across the studied groups.

For example, among Huntington's Disease individuals, the influence of recent outcome experiences in IGT selections has ascended over that of more remote episodes; and responding has been less consistent with extant information as transduced into alternative-outcome modeled expectations (Busemeyer & Stout, 2002; 2007). Judgments of both stimulant- and alcohol-dependent individuals, on the other hand, have tracked negative feedback to WCST selections to a lesser degree than have those of controls. Although similarly less affected by negative feedback, stimulant-dependent individuals have been more sensitive than alcohol-dependent individuals to positive outcomes attending their selection responses (Bishara, et al, 2010).

Through the psychological significance of their constituent parameters, sequential learning models have endowed the routinely used measures, above, with incremental content validity and construct representation (Embretson, 1983). Conventional measures have been understood as to their dynamical-process underpinnings. For instance, WCST performance (e.g., perseverative errors, whereby card sorting has not transitioned to a newly imposed organizing principle) has been simulated by modeled sequential-learning (Bishara, et al, 2010). Errors of commission on the Go/No-Go task have been associated with elevated attention to reward outcomes, and errors of omission have been associated with greater attention to punishing outcomes (Yechiam, Goodnight, Bates, Busemeyer, Dodge, Pettit & Newman, 2006).

Model parameters also have lent incremental nomothetic span (Embretson, 1983) to routinely used measures. Specifically, in addition to producing theoretically consistent diagnostic-group correlates (e.g., elevated sensitivity to IGT-selection rewards, among cocaine users; Yechiam, et al, 2007), parameters have been differentially linked to relevant multi-item psychometric measures (Yechiam, et al, 2008).

Moreover model-based measures have displayed diagnostic efficacy incremental to conventional measures. Individual differences in model parameters have added to the prediction of bipolar disorder, over and above that of cognitive-functioning and personality/temperament inventories (Yechiam, et al, 2008). In addition to its explanatory value for conventionally-measured WCST performance, model parameters comprehensively have captured conventional measures' diagnostic sensitivity to substance abuse, but conventional measures have failed to capture that of model parameters (Bishara, et al, 2010).

In addition to the credentials enumerated above, performance of the EVL model and its affiliates have survived the crucible of competition against competing model architectures (e.g., Yechiam, et al, 2007). Applied versions of EVL and closely related sequential learning models also have been vindicated against internal variations on the ultimately applied versions (e.g., attempts at either reduced, or increased parameterization; Bishara, et al, 2010; Yechiam, et al, 2007).

Formal modeling of pathocognition, and functional neuroimaging (the case of stimulus-encoding elongation in schizophrenia). Mathematical and computational modeling of cognitive psychopathology can provide vital information when it comes to the functional component of clinical functional neuroimaging (functional Magnetic Resonance Imaging, MagneticResonance Spectroscopy, Electroencephalography, and Electro-Magnetoencephalography). Cognitive-process unfolding, as mathematically mandated by a tenable process model, can speak to the "why, where, when and how" of neurocircuitry estimation. In so doing, it can provide an authoritative antidote to the vexatious problem of "reverse inference", whereby functions whose

neurocircuitry is purported as being charted, are inferred from the monitored neuro-circuitry itself (e.g., Green, et al, 2013; see Poldrack, 2011).

So called event-related neuroimaging seeks to track sites of neuro-activation, and inter-site co-activation, aligned with transitions taking place in experimental cognitive performance paradigms (e.g., occurrence of a probe item, for ascertainment of its presence among a set of previously memorized items, in a memory-search task; or presentation of a visual array, for ascertainment of target presence, in a visual-search task). Events of actual interest, however, are the covert cognitive operations activated by one's experimental manipulations. Estimating the time course of events that are cognitive per se arguably necessitates a tenable mathematical model of their stochastic dynamics. Stipulation of such temporal properties seems all the more important when a covert process of principal interest defensibly is accompanied by collateral processes, within a cognitive-task trial (e.g., encoding the probe item for purposes of memory-set comparison, along with response operations, above). Motivation for model construction is fuelled further if the designated intra-trial process has symptom and ecological significance. The upshot is that mathematical modeling of cognitive abnormality is uniquely poised to supply information as the "why, where, and when" of clinical and other neuroimaging. It arguably also speaks to the "how" of vascular- and electro-neruophysiological signal processing, in terms of selection from among the array of signal-analysis options.

Clinical importance of neurocircuitry measures can be increased through their analytical interlacing with clinical symptomatology. Among individuals with schizophrenia, for example, elongated duration of encoding (cognitively preparing and transforming) presenting stimulation into a format facilitating collateral processes (e.g., those taking place in so-called working

memory) can disproportionately jeopardize the intake of contextual cues, vital to anchoring other input in its objective significance (i.e., "context deficit"; e.g., Dobson & Neufeld, 1982; George & Neufeld, 1985). This combination potentially contributes to schizophrenia thought content disorder (delusions and thematic hallucinations; Neufeld, 1991; 2007c). Formally prescribed cognitive psychopathology thus can help broker symptom significance to monitored neurocircuitry.

The latter likewise can be endowed with increased ecological significance. Intact encoding, for example, may be critical to negotiating environmental stressors and demands, especially when coping is cognition intensive (e.g., where threat minimization is contingent on predictive judgments surrounding coping alternatives; e.g., Neufeld, 2007b; Shanahan & Neufeld, 2010). Impairment of this faculty therefore may be part and parcel of stress susceptibility (Normal & Malla, 1994). Compromised stress resolution in turn may further impair encoding efficiency, and so on (Neufeld, et al, 2010).

Turning to the "where" of neuro-imaging measurement, targeted cognitive functions in principle are used to narrow down neuro-anatomical regions of measurement interest, according to locations on which the functions are thought to supervene. Precision of function specification constrained by formal modeling in principle should reduce ambiguity as to what cognitively is being charted neurophysiologically. Explicit quantitative stipulation of functions of interest should sharpen imaging's functionally guided spatial zones of brain exploration.

Formal modeling of cognitive-task performance seems virtually indispensable to carving out intra-trial times of neuro-(co)activation measurement interest. Stochastic dynamic models

specify probabilistic times of constituent-process occurrence. For instance, encoding a probe item for its comparison to memory-held items would have a certain time trajectory following the probe's presentation. With encoding as the singled-out process, imaging signals identified with a trial's model-estimated epoch during which the ascendance of encoding was highly probable, would command special attention. A tenable stochastic model of trial performance can convey the stochastic dynamics of a designated symptom- and ecologically-significant process, necessary for a reasonable estimate of its corresponding within-trial neuro-circuitry (additional details are available in Neufeld, 2007c, the mathematical and neuro-imaging specifics of which are illustrated in Neufeld, et al, 2010).

A delimited intra-trial measurement epoch stipulated by a stochastic-model specified time trajectory commands considerable temporal resolution of processed imaging signals. Among analysis options, covariance of activation between measurement sites, across time ("seed-voxel, time series covariance") has been shown empirically and mathematically to have the temporal resolution necessary to monitor neuro-circuitry during the designated epoch (Neufeld, 2012; see also Friston, Fletcher, Josephs, Holmes & Rugg, 1998). In this way, formal modeling of cognitive performance speaks to the "how" of imaging-signal processing, by selecting out or creating neuro-imaging-signal analysis options with requisite temporal resolution. Note that success at ferreting out target-process neurocircuitry is supported by the selective occurrence of differential neuro-(co)activation to the modeled presence of the triaged process in the clinical sample—and conversely, its non-occurrence otherwise (also known as "multiple dissociation").

Model-informed times of measurement interest stand to complement tendered regions of interest, in navigating space-time co-ordinates of neuroimaging measurement. Moreover, symptom and ecologically significant functions remain in the company of collateral functions involved in task-trial transaction (e.g., item encoding occurs alongside the memory-scanning and response processes, for which it exists). Keeping the assembly of trial-performance operations intact, while temporally throwing into relief the target process, augurs well for preserving the integrity of the latter, as it functions in situ. Doing so arguably increases the ecological validity of findings, over and against experimentally deconstructing the performance apparatus by experimentally separating out constituent processes, and thereby risking distortion of their system-level operation.

Special Considerations Applying to Mathematical and Computational Modeling in Psychological Clinical Science and Assessment.

Clinical mathematical and computational modeling is a relatively recent development in the history of modeling activity (see, e.g., Townsend, 2008). It goes without saying that opportunities unlocked by clinical implementations are accompanied by intriguing challenges. These take the form of upholding rigor of application amidst exigencies imposed by clinical constraints, along with faithfully engaging major substantive issues found in clinical science and assessment.

Methodological challenges. One of the first initial challenges to clinical formal modeling consists of obtaining sufficient data for stability of model-property estimation. This challenge is part and parcel of the field's longstanding concern with ensuring the applicability of inferences

to a given individual, or at least homogeneous class of individuals to which the one at hand belongs (Davisdon & Costello, 1969; Neufeld, 1977).

Mainline mathematical modeling too has been concerned with this issue. The solution by and large has been to model performance based on a large repertoire of data obtained from a given participant, tested over the course of a substantial number of multi-trial sessions. Clinical exigencies (e.g., participant availability or limited endurance), however, may proscribe such extensiveness of data accumulation; the trade-off between participants and trial-number may have to be tipped toward more participants and fewer trials.[6] The combination of fewer performance trials, but more participants requires built-in checks for relative homogeneity of individual data protocols, to ensure that the modeled aggregate is representative of its respective contributors.

Collapsing data across participants within a testing session, then, risks folding systematic performance differences into their average, with a resultant centroid that is unrepresentative of any of its parts (see Estes, 1956). Note in passing that similar problems could attend participant-specific aggregation across experimental sessions, should systematic inter-session performance drift, or re-configuration occur. Methods have been put forth for detecting systematic heterogeneity (Batchelder & Riefer, 2007; Carter & Neufeld, 1999; Neufeld & McCarty, 1994), or ascertaining its possibility to be inconsequential to the integrity of modeling results (Riefer, Knapp, Batchelder, Bamber & Manifold, 2002). If present, methods of disentangling, and separately modeling the conflated clusters of homogenous data have been suggested (e.g., Carter, Neufeld, & Benn, 1998).

Systematic individual differences in model properties also can be accommodated by building them into the model architecture itself. Mixture models implement heterogeneity in model operations across individual and/or performance trials. For example, model parameters may differ across individual participants within diagnostic groups, the parameters in principle forming their own random distribution. As parameter values now are randomly distributed across individuals ("hyper distribution"), a parameter value's probability (discrete-value parameters), or probability density (continuous-value parameters) takes on the role of its Bayesian prior probability (density) for the current population of individuals. Combined with a sample of empirical task performance, on whose distribution the parameter bears ("base distribution"), an individualized estimate of the parameter is available through its Bayesian posterior parameter distribution (see *Bayesian parameter estimation*, above). A mixture model approach can be especially appealing in cases of "over-dispersion", as indicated say by a relatively large coefficient of variation (standard deviation of individual mean performance values divided by their grand mean; see "over-dispersion" under *Bayesian parameter estimation*, above). [7]

Also availed through mixture-models are customized (again, Bayesian posterior) performance distributions. If the latter apply to process latencies, for example, they can be consulted to estimate strategic times of neuro-imaging measurement within subgroups of parametrically homogeneous participants (detailed in Neufeld, et al, 2010, Section 7).

Apropos of the limitations indigenous to the clinical enterprise, mixture models potentially moderate demands on participants, when it comes to their supplying a task-performance sample. A relatively modest sample tenably is all that is required, because of the

stabilizing influence on participant-specific estimates, as bestowed by the mixing distribution's prior-held information. This obviously is boon in the case of clinical modeling, considering the possible practical constraints in obtaining a stable performance sample from already distressed individuals.

Mixture models also open up related avenues of empirical model testing. For purposes of assessing model validity, predictions of individuals' performance, mediated by modest performance samples, can be applied to separately obtained individual validation samples. In this way, model verisimilitude can be monitored, including the model's functioning at the level of individual participants (for details exposited with empirical data, see Neufeld, et al, 2010). Because of the stabilizing influence of the Bayesian prior distribution of base-distribution properties, this strategy provides a tack to the thorny issue of "small-*N* model testing" (see "Bayesian shrinkage", under *Bayesian parameter estimation*, above).

An additional asset conferred by a mixture-model platform bridges methodological and clinical substantive issues, as follows. Emerging from this model design is a statistical— and cognitive-behavioral—science principled measurement infrastructure for dynamically monitoring individual treatment response. The method exploits the distinct prior distributions of base-model properties obtained from varyingly symptomatic, and healthy groups. Now Bayes' Theorem allows the profiling of an individual at hand, with respect to the latter's current probability of belonging to group g, $g = 1,2, \ldots G$ (G being the number of referent groups), given obtained performance sample $\{*\}$: $Pr(g|\{*\})$. Such a profile can be updated with the progression of treatment.

Moreover, closely related procedures can be applied to the assessment of treatment regimens. In this case, treatment efficacy is monitored by charting the degree to which the treated sample at large is being edged toward, or possibly away from, healthier functioning (computational specifics are illustrated with empirical data in Neufeld, 2007b, and Neufeld, et al, 2010).

Clinical substantive issues. A substantive issue interlaced with measure-theoretic considerations is the so-called differential-deficit, psychometric-artefact problem (Chapman & Chapman, 1973). Abnormalities that are more pronounced than others may have special etiological importance, in part because of associated neuro-physiological substrates. False inferences of differential deficit, however, are risked because the relative amounts by which examined faculties are disorder-affected, are conflated with the psychometric properties of the instruments used to measure them. This issue retains much currency in the contemporary clinical-science literature (e.g., Gold & Dickinson, 2013).

Frailties of recommended solutions, consisting of inter-measure calibration toward equality on classical measurement properties (reliability and observed-score variance) have been noted almost since the recommendations' inception (e.g., Neufeld, 1984a; Neufeld & Broga, 1981; with augmenting technical reports, Neufeld & Broga, 1977; Neufeld 1984b). Note that the arguments countering the original classical-measurement recommendations have been demonstrated to extend from diagnostic-group to continuous-variable designs (as used, e.g., by Kang & MacDonald, 2010; see Neufeld & Gardner, 1990).

It can be shown that transducing classical psychometric partitioning of variance into formally-modeled sources, not only lends a model-based substantive interpretation to the

partitioned variance, but renders the psychometric-artefact issue (which continues to plague reliance on off-the-shelf and sub-quantitative, often clinically, rather than contemporary cognitive-science, contrived measures) essentially obsolete (Neufeld, 2007b; see also McFall & Townsend, 1998; Neufeld, Vollick, Carter, Boksman & Jette', 2002; Silverstein, 2008). Partitioned sources of variance in task performance now are specified according to a governing formal performance model, which incorporates an analytical stipulation of its disorder-affected and spared features (see Figure 1, and related prose, in the **Introduction** of this chapter). These sources of variance include classical measurement-error variance (within-participant variance); within-group inter-participant variance; and inter-group variance.

Another major substantive issue encountered in clinical science concerns the integration of so-called "cold and hot cognition". Cold cognition, in this context pertains more or less to the mechanisms of information processing, and hot cognition pertains to their informational product, notably with respect to its semantic and affective properties—roughly, "how the thought is delivered (cold cognition), and what the delivery consists of (hot cognition) ". Deviations in the processing apparatus leading to interpretations of one's environment cum responses that bear directly on symptomatology, hold special clinical significance. The synthesis of cold and hot cognition perhaps is epitomized in the work on risk of sexual coercion, and eating and other disorders, by Teresa Treat, Richard Viken, Rchard McFall, and their prominent clinical-scientist collaborators. Examples include Treat, McFall, Viken, Nosofsky, McKay & Kruschke (2002), and Treat, Viken, Kruschke & McFall (2010; see also Treat & Viken, 2010).

These investigators have shown how basic perceptual processes (Perceptual Organization) can ramify to other clinically-cogent cognitive-behavioral operations, including

those of classification in interpersonal perception, and memory. This program of research exemplifies integrative, translational, and collaborative psychological science. It has been rigorously eclectic in its recruitment of modeling methodology. Included has been multidimensional scaling; classification paradigms; perceptual-independence—covariation paradigms; and memory paradigms -- all work that brooks no compromise on state-of-the-art mathematical and computational developments. Productively studied have been deviations in cognitive mapping, classification, and memory retrieval, surrounding presented items bearing on clinically important problems comprising eating disorder, and risk sexual aggression.

A further point of contact, between formal process modeling and substantive clinical issues, involves relations between the former and multi-item psychometric inventories. Empirical associations between model properties, and multi-item measures, reciprocally contribute to each other's nomothetic-span construct validity. Correlations with process-model properties furthermore lend construct-representation construct validity to psychometric measures. Psychometric measures in turn can provide useful estimates of model properties with which they sufficiently correlate, especially if their economy of administration exceeds that of direct individual-level estimates of the model properties (Carter, Neufeld & Benn, 1998; Wallsten, Pleskac & Lejuez, 2005).

Furthermore, to liaison with multi-item psychometric inventories, responding to a test item can be viewed as an exercise in dynamical information processing. As such, modeling option selection via item response theory can be augmented with modeling item response time, the latter as the product of a formally portrayed dynamic stochastic process (e.g., Neufeld, 1998;

van der Maas, Molenaar, Maris, Kievit & Boorsboom, 2011; certain direct parallels between multi-item inventories and stochastic process models have been developed in Neufeld, 2007b).

Note that a vexing contaminant of scores on multi-item inventories is the influence of "Social Desirability "(S.D.) – that is the pervasive inclination to respond to item options in a socially desirable direction. Indeed, a prime mover of contemporary methods of test construction, Douglas N. Jackson, has stated that S. D. is the "*g*" factor of personality and clinical psychometric measures (e.g., Helmes, 2000). Interestingly, it has been shown that formal theory and its measurement models can circumvent the unwanted influence of S. D. , because of the socially neutral composition of the measures involved (Koritzky & Yechiam, 2010).

Conclusion.

Mathematical and computational modeling arguably is essential to accelerating progress in clinical science and assessment. Indeed, rather than its being relegated to the realms of an esoteric enterprise or quaint curiosity, a strong case can be made for the central role of quantitative modeling in lifting the cognitive side of clinical cognitive neuroscience out of current quagmires, and avoiding future ones.

Clinical formal modeling augurs for progress in the field, owing to the explicitness of theorizing to which the modeling endeavor is constrained. Returns on research investment are elevated because rigor of expression exposes empirical shortcomings of extant formulations; blind alleys are expressly tagged as such, thanks to the definiteness of derived formulae, so that wasteful retracing is avoided; and needed future directions often are indicated, because of the forced exactness of one's quantitative formulations.

Otherwise unavailable or intractable information is released. Moreover, existing data can be exploited by freshly viewing and analyzing it through methods opened up by a valid mathematical, computational infrastructure (Novotney, 2009).

Formal developments also stand to enjoy an elongated half-life of contribution to the discipline. Staying power of the value of rigorous and substantively important achievements has been seen in mathematical modeling generally. For instance, early fundamental work on memory dynamics (e.g., Atkinson, Belseford & Shiffrin, 1967) recently has been found useful in the study of memory in schizophrenia (Brown, Lohr, Nosestine, Turner, Ganst & Eyler, 2007). A similar durability stands to occur for clinical mathematical psychology, within the field of clinical science itself, and beyond.

Certain suspected agents of disturbance may resist study through strictly experimental manipulation of independent variables (e.g., non-model-informed correlational experiments; see *Mulitnomial processing tree modeling of memory and related processes; unveiling and elucidating deviations in clinical samples*, above). Such may be the case on ethical grounds, or because the theoretically tendered agent may elude experimental induction (e.g., organismically endogenous stress activation suspected as generating cognition-detracting intrusive associations and/or diminished cognitive-workload capacity). Tenability of such conjectures nevertheless may be tested by examining improvement in model fit to data from afflicted individuals, through quantitatively inserting the otherwise inaccessible constructs into the model composition.

Current challenges can be expected to spawn vigorous research activity in several future directions. Among others, included are: (a) capitalizing on modeling opportunities in multiple areas of clinical science, poised for modeling applications (e.g., stress, anxiety disorders, and

depression); (b) surmounting constraints intrinsic to the clinical setting that pose special barriers to the valid extraction of modeling-based information (e.g., estimating and providing for potentially confounding effects of medication on cognitive-behavioral task performance); (c) developing sound methods for bridging mathematical and computational clinical science to clinical assessment and intervention; and, (d), creating methods of tapping model-disciplined information from currently available published data-- in the service of model-informed, substantively meaningful, meta analyses.

It may be said that "evidence-based practice is best practice only if it is based on best evidence". Best evidence goes hand in hand with maximizing methodological options, which compels the candidacy of those that stem from decidedly quantitative theorizing.

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Textbox 1. Abductive Reasoning in Clinical Cognitive Science.

Scientific rigor does not demand that theoretical explanation for empirical findings be restricted to a specific account, from a set of those bearing on the study, singled out before the study takes place. In fact, the value of certain formal developments to understanding obtained data configurations may become apparent only after the latter present themselves. It is epistemologically acceptable to explanatorily retrofit extant (formal) theory to empirical data (e.g., in the text, changing the clinical sample's value of *k*' versus *v*), a method known as "abductive reasoning" (Haig, 2008). Abductive reasoning not only has a bona-fide place in science, but is economical, in its applicability to already published data (Novotney, 2009), and/or proffered conjectures about clinically significant phenomena.

On the note of rigorous economy, the clinical scientist can play with model properties to explore the interactions of model-identified, or other proposed sources, say of pathocognition, with various clinically significant variables, such as psychological stress. For example, it has been conjectured that some depressive disorders can be understood in terms of "highly practiced", automatic negative thoughts supplanting otherwise viable competitors; and also that psychological stress enhances ascendancy of the former on the depressed individual's cognitive landscape (Hartlage, Alloy, Vasquez & Dykman, 1993). Translating these contentions into terms established in mainstream quantitative cognitive science, however, discloses that psychological stress instead *reduces* the ascendancy of well-practiced negative thoughts, at least within this highly-defensible assumptive framework (Neufeld, 1996; Townsend & Neufeld, 2004).

The quantitative translation begins with expressing the dominance of well-practiced (so-called automatic) negative, versus less practiced (so-called effortful) non-negative thought

content, as higher average completion rates for the former. With these rate properties in tow, the well-practiced and less-practiced thought content then enter a formally modeled “horse race”, where the faster rates for negative-thought generation evince higher winning probabilities, for all race durations. Note that although these derivations result from basic computations in integral calculus, they nevertheless yield precise predictions, and lay bare their associated assumptions.

Differentiating the above hoarse-race expression of the probability of “negative-thought” victory, with respect to a parameter conveying the effects of stress on processing capacity, leads to a negative derivative. Formalized in this way, then, the result shows psychological stress actually to handicap the negative thought contender.

It is conceivable that reduction in the ascendancy of well-practiced negative thoughts in the face of stressing environmental demands and pressures, in favour of less-practiced, but more adaptive cognitive processes, conveys a certain protective function. In all events, this example illustrates the hazards of depending on unaided verbal reasoning in attempting to deal with complex inter-variable relations (including stress effects on psychopathololgy), and exemplifies the disclosure through available formal modeling, of subtleties that are both plausible and clinically significant-- if initially counter-intuitive (Staddon, 1984; Townsend, 1984).

Textbox 2. A Novel Statistical Test for Model-parameter Differences.

As indicated in the text, mathematical modeling can prescribe its own measures, experimentation and tests. Sometimes proposed tests can transcend the specific domain from which they emerge. This is the case for a statistical test for inequality of model properties between groups, devised by Chechile (2007; 2010).

Considerations begin with a "hoarse race" model of cognitive processes occurring in parallel (Townsend & Ashby, 1983, e.g., p. 249). At any time point since the start of the race t', the probability density function of the first process of the pair winning is its density function $f_1(t')$ times the probability of the second function remaining incomplete $S_2(t')$, or $f_1(t')\ S_2(t')$. Integrating this expression from $t' = 0$ to $t' = t$ gives the probability of the first completion being earlier than the second, as evaluated to time t. Integrating across the entire range of values ($t' = 0$ to $t' = \infty$) gives the unconditional probability of the first process having a shorter time than the second. Chechile has adapted this reasoning to the construction of a statistical test expressing the probability that a model- parameter value under one condition of cognitive-behavioral performance is less than its value under a comparison condition $Pr(\theta_1<\theta_2)$.

The method uses Bayesian posterior distributions of θ_1 and θ_2 (with qualifications described and illustrated empirically in Chechile, 2007). Slicing the range of θ into small segments, the proportion of the distribution of θ_1 within a particular segment, times the proportion of the θ_2 distribution beyond that segment, gives the probability of θ_1 lying within the segment, and that of θ_2 lying beyond, analogous to $f_1(t')$ and $S_2(t')$. Summing the products over the entire range of θ (if θ_1 and θ_2 themselves are probabilities, then θ ranges from 0 to 1.0)

directly gives the unconditional probability of θ_1 being lower than θ_2. Such a probability of .95, for instance, corresponds to a 95% level of confidence that $\theta_1 < \theta_2$.

Glossary Textbox.

analytical construct validity: support for the interpretation of a model parameter that expresses an aspect of individual differences in cognitive performance, according to the parameter's effects on model predictions.

base distribution: distribution of a cognitive-behavioral performance variable (e.g., speed or accuracy) specified by a task-performance model.

construct-representation construct validity: support for the interpretation of a measure in terms of the degree to which it comprises mechanisms that are theoretically meaningful in their own right.

exponential distribution: a stochastic distribution of process latencies whose probability density function is ve^{-vt}, where t is time (arbitrary units), and v is a parameter; latencies are loaded to the left (i.e., toward $t = 0$, their maximum probability density being at $t = 0$), and the distribution has a long tail.

hyper-distribution: a stochastic distribution governing the probabilities, or probability densities, of properties (e.g., parameters) of a base distribution (see *Bayesian parameter estimation* in the text).

likelihood function: a mathematical expression conveying the probability or probability density of a set of observed data, as a function of the cognitive-behavioral task-performance model (see *Bayesian parameter estimation* in the text).

mixing distribution: see hyper-distribution.

mixture model: a model of task performance, expressed in terms a base distribution of response values, whose base-distribution properties (e.g., parameters) are randomly distributed according to a mixing distribution (see *Bayesian parameter estimation* in the text).

multiple dissociation: cognitive-behavioral performance patterns, or estimated neuro-circuitry, occurring for a clinical group under selected experimental conditions, with contrasting patterns occurring under other conditions. The profile obtained for the clinical group is absent, or opposite, for control or other clinical groups.

nomothetic-span construct validity: support for the interpretation of a measure according to its network of associations with variables to which theoretically it is related.

normalizing factor: overall probability or probability density of data or evidence, all model-prescribed conditions (e.g., parameter values) considered (see *Bayesian parameter estimation*, in the text).

overdispersion: greater variability in task-performance data than would be expected if a fixed set of model parameters were operative across all participants.

prior distribution: in the Bayesian framework, the probability distribution before data are collected (see hyper-distribution).

posterior distribution: in the Bayesian framework, the distribution corresponding to the prior distribution after the data are collected. Bayes' theorem is used to update the prior distribution, following data acquisition. The key agent of this updating is the likelihood function (see *Bayesian parameter estimation* in the text).

Footnotes

1. See also Chechile (2010) for the introduction of a novel procedure for individualizing MPTM parameter values-- especially pertinent to clinical assessment technology. See also Smith & Batchelder (2010) on computational methods for dealing with the clinical-science issue of group-data variability, owing to individual differences in parameter values (the problem of "overdispersion").
2. Still others (Ouimet, Gawronski & Dozois, 2009) have tendered extensive verbally conveyed schemata and flow diagrams [cf. McFall, Townsend & Viken (1995) for a poignant demarcation of models qua models] entailing dual-system cognition (extrapolation of "automatic" and "controlled" processing properties; Schneider & Shiffrin, 1977; Shiffrin & Schneider, 1977), and threat-stimulus disengagement deficit [possibly suggesting serial item processing; but cf. Neufeld & McCarty (1994); Neufeld, Townsend & Jette (2007); White, et al, 2010a, p. 674].
3. An independent parallel, limited-capacity processing system, with exponentially-distributed processing latencies, potentially characterizing the processing architectures of both HA and LA individuals at least during early processing (Neufeld & McCarty, 1994; Neufeld, Townsend & Jette, 2007): (a), parsimoniously can be shown to cohere with reaction-time findings on HA threat bias, including those taken to indicate threat stimulus disengagement deficit; and, (b), also can be shown to cohere with collateral oculomotor data (e.g., Mogg, Millar & Bradley, 2000; Neufeld, Mather, Merskey & Russell, 1995; see also, Townsend & Ashby, 1983, chapter 5).
4. Note that encoding and response processes typically have been labeled "base processes", and often have been relegated to "wastebasket" status. They may embody important differences, however, between clinical samples and controls. By examining "boundary separation", a response-criterion parameter of the Ratcliff diffusion model, for example,

White, Ratcliff, Vasey and McKoon (2010b) have discovered that HA, but not LA participants increase their level of caution in responding after making an error (an otherwise undetected "alarm reaction"). See also Neufeld, Boksman, Vollick, George and Carter (2010), for analyses of potentially symptom significant elongation of stimulus encoding-- a process subserving collateral cognitive operations-- among individuals with a diagnosis of schizophrenia.

5. Extensions to mathematically prescribed signatures of variants on the cognitive elements described above, recently have included statistical significance tests, and charting of statistical properties (e.g., "unbiasedness" and "statistical consistency" of signature estimates; Houpt & Townsend, 2010; 2012); and measure theoretic and methodological unification of concepts in the literature related to cognitive-workload capacity (known as "Grice" and "Miller inequalities"), all under qualitative properties of SFT's quantitative diagnostics (Townsend & Eidels, 2011). Bayesian extensions also currently are in progress. A description of software for SFT, including a tutorial on its use, has been presented by Houpt, Blaha, McIntire, Havig and Townsend (in press).
6. The challenge of requisite data magnitude may be overblown. Meaningful results at the individual level of analysis, for example, can be obtained with as few as 160 trials per experimental condition--if the method of analysis is mathematical-theory mandated (see Johnson, et al, 2010). Furthermore, clinicians who may balk at the apparent demands of repeated-trial cognitive paradigms nevertheless may have no hesitation in asking patients to respond to 567 separate items of a multi-item inventory.
7. Of late, cognitive science has witnessed a burgeoning interest in mixture models, dubbed "hierarchical Bayesian analysis" (e.g., Lee, 2011). The use of such models in cognitive science nevertheless can be traced back at least two and one half decades (e.g., Morrison,

1979; Schweickert, 1982). Mixture models of various structures (infinite, and finite, continuous and discrete), along with their Bayesian assets, moreover have enjoyed a certain history of addressing prominent issues in clinical cognitive science (e.g., Batchelder, 1998; Carter, et al, 1998; Neufeld, 2007b; Neufeld, Vollic,k, Carter, Boksman & Jette, 2002; Neufeld & Williamson, 1996), some of which are stated in the text.

Figure 1. Relations between clinical and non-clinical mathematical and computational psychology.