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Formal Innovations to Clinical Cognitive Science and Assessment

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Abstract

Mathematical modeling is increasingly driving progress in clinical cognitive science and assessment. Mathematical modeling is essential for detecting certain effects of psychopathology – mental disturbance--through comprehensive understanding of tell-tale cognitive variables such as workload capacity and efficiency in using capacity, and their contrast under quantitative measurement. The research paradigm guiding this formal clinical science is outlined. An example using a distinctive cognitive abnormality in schizophrenia – taking longer to cognitively represent encountered stimulation – provides an illustration of a quantitative framework for studying intricate mental health-impairing phenomena. Added benefits of formal developments, among others, include symptom description and prediction, new methods of cognitive- and statistical-science grounded clinical assessment over time, both for individuals and treatment regimens, and refinement of the cognitive-function side of clinical functional neuroimaging.

Key Words

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Formal Innovations to Clinical Cognitive Science and Assessment

Contemporary directions in translational clinical science have taken on a wave of mathematical modeling of symptom-related cognitive abnormalities. The basic research paradigm for this movement is illustrated in Figure 1. Mathematical models of cognitive performance among healthy individuals are adjusted to accommodate deviations from normal performance, among participants with selected forms of psychopathology—mental disturbance. Such deviations typically center around cognitive-performance speed and/or accuracy. Parts of the model remaining intact are considered to indicate cognitive functions that are spared, while those parts where performance deviations compel modification of the model are flagged as signifying disorder-affected functions. Minimal adjustment of the model is desired, in the interest of parsimony. In this way, models provide a formal framework to determine which cognitive processes do or do not differ between clinical groups and healthy controls.

Such formal theoretical developments can offer multiple advantages in explanation and measurement of psychopathology. A case study of developments and advantages is presented, involving symptom-related cognitive neuroscience of schizophrenia.

Rounding out the research paradigm depicted in Figure 1, the domain of clinical mathematical cognitive neuroscience stands to uniquely contribute to mainstream mathematical

cognitive neuroscience. It can do so according to model generalization testing (Busemeyer & Wang, 2000). Generalization testing evaluates the robustness of model performance with new experimental paradigms or populations. Clinical mathematical cognitive neuroscience provides key opportunities for generalization testing with respect to extreme individual differences, associated with psychopathology. Models that readily accommodate performance deviations are preferred to those that fail, or that are strained in doing so. That is, a model is supported when observed results comprising psychopathology-related abnormalities, can be well-predicted without major adjustment of the model's workings.

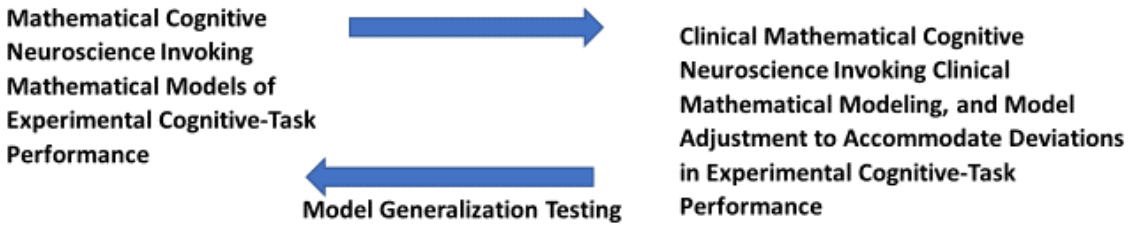

Figure 1. Relations between Mathematical Cognitive Neuroscience, and Clinical Mathematical Cognitive Neuroscience.

Note that clinical mathematical modeling, the subject of this paper, involves analytical theorems and proofs, and algebraic derivations expressing cognitive transactions relevant to

clinical disorders. It should be distinguished from another conspicuous, somewhat related type of modeling, known as "computational psychiatry". Computational psychiatry addresses how cognitive transactions might be realized at the level of neural organization and operations. By and large, computational psychiatry delegates disorder-related deviations of neural functioning to computer simulation of neural networks and neuro-dynamics (neuronal conductance properties). Accounts and examples of computational psychiatry are available in Montague, Dolan, Friston, et al (2012), Huys, Maia & Frank (2016), Wang and Krystal (2014) and Grossberg (1999). For a rigorous, comprehensive treatment of artificial neural networks, see Golden (1996). Computational psychiatry and clinical mathematical modeling potentially are complementary when it comes to quantitative accounts of clinical disorders (e.g., Carter & Neufeld, 1999; 2007). Extensive elaboration on distinctions among alternate forms of modeling clinical phenomena can be found in Neufeld (2007a).

Case Study: Cognitive Neuroscience of Stimulus Encoding in Schizophrenia

Schizophrenia affects approximately 0.5 percent of the North American population. Symptoms can take the form of delusions and hallucinations ("thought-content disorder"), incoherent speech, reduced cognitive efficiency, and impoverished motivation. Applying the research strategy depicted in Figure 1, a deviation in cognitive performance recurrently singled out in schizophrenia, across multiple experimental tasks, and levels of patient status (e.g., first-episode, never-treated, outpatient, inpatient) consists of delayed completion of stimulus encoding. Here, stimulus encoding refers to cognitively preparing and transforming cognitive-task stimuli into a format facilitating collateral processes. For instance, participants might be asked to memorize a set of novel stimuli (e.g., TZAM, CEYP). After a delay, they are presented with additional stimuli, some of which have been seen before and some of which have not, and

they are asked whether each stimulus was part of the original set. To do well in this task the participant must encode a presented stimulus into a cognitive format facilitating comparison to the previously memorized set of stimuli. For example, in the case of basic visual-template matching, it may be necessary to cognitively extract the presented stimulus' physical features, like curves, lines and intersections; or in the case of stimulus-name matching, it may be necessary to tag a presented digit or letter stimulus with its name, for comparison to names of the stimuli in the previously memorized set.

Mathematical modeling enables quantitative dissection of cognitive processes, to help pinpoint specific sources of cognitive performance deviation. With respect to encoding, for example, the process can be broken down, as follows. First, the process is made up of constituent encoding operations-- encoding subprocesses, such as registration of curves, lines, and intersections of a presented stimulus. Second, the encoding subprocesses themselves take place with a certain speed, known as subprocess-level cognitive workload capacity (e.g., Neufeld, Townsend & Jette, 2007; Wenger & Townsend, 2000). A recurrent result of the model-adjustment operation of Figure 1, has consisted of the following combination of spared and disorder-affected parts of the encoding process: subprocess-level cognitive-workload capacity remains intact, but the number of encoding subprocesses undertaken is elevated. Cognitive workload capacity escapes impairment, whereas efficiency of its implementation does not. This combination of spared and affected components of encoding performance is analogous to a race-horse striding at a normal pace, but running closer to the outside rail, thus increasing the requisite paces, and therefore time, for course completion. This combination also illustrates the nature of adjusting the model of healthy encoding, so that the altered model conforms to the specific pattern of empirical deviations among clinical (schizophrenia) participants

The developments described above defensibly have tracked the abnormality in the process of interest to a specific formalized property—the subprocess-number parameter of the stimulus-encoding process. Identification of such a property stands to open the way to potentially important advances in this domain of psychological clinical science. The abnormality arguably represents a critical deficit, compromising activities into which timely stimulus encoding routinely enters (e.g., daily self-maintenance functions and meeting environmental stresses and demands). The quantitative apparatus in which this property is embedded provides for certain methodological benefits, including theory-guided measurement and clinical assessment, and stipulation of the cognitive side of cognitive neurophysiology. It also can be shown to be potentially symptom related, notably with respect to thought-content disorder (delusions and thematic hallucinations). Such symptomatology is considered to emanate from failure to encode specifically context-related features of a stimulus complex, during episodes of information intake. With weakened influence of reality-grounding, objectifying cues, other information that successfully is taken in during an episode, is open to false interpretation (this symptom extension quantitatively is expanded upon in Neufeld, 2007b, and Neufeld, Boksman, Vollick, et al, 2010). The formal theoretical account of symptomatology consequences is in the spirit of meeting recent calls for "defining a mechanism of complex behaviors" and formulating "computationally-defined behaviors" (National Institutes of Mental Health Web Site, 2017; 2018). It also accords with the research-domain criterion initiative (e.g., Kozak & Cuthbert, 2016), inasmuch as the identified mechanism evidently extends to other forms of clinical disturbance (e.g., major depressive disorder; Taylor, Théberge, Williamson, et al, 2016), and non-clinical populations (Nicholson & Neufeld, 1993).

Note that model adjustment capturing changes in cognition associated with clinical disorder typically takes the form of altering model-parameter values ("scalar differences"), such as encoding-subprocess quantity, or rate of subprocess completion. Model architecture (non-scalar differences), meaning the number of model parameters involved, or their arrangement in relation to each other, ordinarily is common to clinical and non-clinical groups alike (see, e.g., Neufeld & Broga, 1981; Wallsten, Pleskac & Lejuez, 2005). In other words, the basic mental apparatus meeting a cognitive challenge is common across groups, but modification of one or more of its parts (parameters) accompanies clinical disorder.

Measurement and Clinical Assessment Guided by Formal Theory

Individual performance samples, for instance of encoding-intensive tasks, permit estimation of cognitive-process parameter values. Parameter-value estimation is available through established methods applied to performance durations of cognitive-task trials. Such methods include maximum-likelihood, distribution-moment matching (e.g., Evans, Hastings & Peacock, 2000), and Bayesian parameter estimation (e.g., Alexandrowicz & Gula, 2020, as used with a mathematical model of decision and choice, finding application with clinical disorders). Clinically relevant cognitive processing is concealed in raw data but can be revealed via mathematical modeling.

Often it may not be reasonable to assume that all individuals within a clinical group have (roughly) the same level of cognitive processing, as indexed for example by a fixed value for a model parameter. Fortunately, mathematical models can be expanded to account for this, notably through expansion as mixture models. Mixture models treat the overall performance of a group as a mixture of different levels of performance among individual group members (e.g., Carter, Neufeld & Benn, 1998; Cutler & Neufeld, 2017).

Mixing distributions can be important, not only in systematically accommodating individual differences, but also because the parameters that mathematically govern the random distributions of model properties (mixing-distribution hyper parameters) can be clinically meaningful in their own right. To illustrate, mixture-model hyper-parameters can convey a particular group's general level of facility with undertaking the elements of the cognitive process at hand (e.g., encoding-subprocesses); they can also be used to indicate susceptibility of such undertaking to the occurrence of psychological stress (exemplified with concrete examples in Neufeld, 2016).

Altogether, mixture-model expansions, illustrated in Figure 2, can increase the span of model explanation by incorporating individual differences. They additionally can tap clinically meaningful constructs, such as cognitive-task amenability, and performance vulnerability to psychological stress.

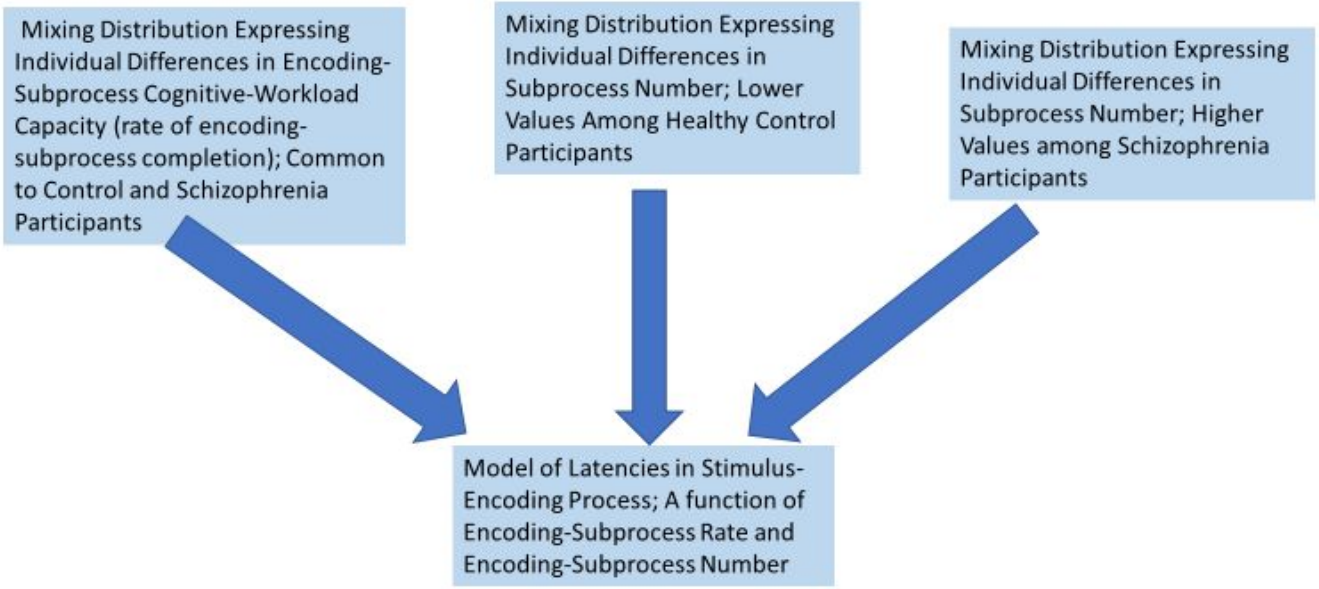

Figure 2. Design of Stimulus-Encoding Mixture Model Accommodating Individual Differences in Parameters of Encoding; The Rate at which Encoding Subprocesses Unfold is Shared by Control and Schizophrenia Participants, whereas the Number of Encoding Subprocesses is Greater among Schizophrenia than Control Participants.

Measuring better with Bayes

Mixture models allow for the likelihood that individuals systematically differ in properties of mathematically expressed cognitive performance. They go an important step further, in providing for efficient estimation of model properties for the individual. They do so by customizing the properties to the person, through Bayesian statistical methodology, as follows. Bayes' Theorem[1], appropriated to the present context, states that

$$\Pr(A \mid \{*\}) = \frac{Pr(A) Pr(\{*\}|A)}{\Pr(\{*\})}, \quad (1)$$

where, $\Pr(A \mid \{*\})$ is the Bayesian probability of an eligible value of a predicted entity A --such as a cognitive- process parameter (e.g., encoding-subprocess amount), given relevant observations {*} (e.g., a cognitive-performance specimen); $Pr(\{*\}|A)$ is the likelihood of the performance specimen, given the eligible value; $Pr(A)$ is the probability of the value under consideration, according to the relevant mixing distribution, entity-related observations {*} aside; and $Pr(\{*\})$ is the probability of the observations, all candidate values of the predicted entity considered (for accounts of Bayesian modeling generally, see classic works such as Berger, 1985, and O'Hagan & Forster, 2004).

[1] A landmark contribution to statistical science, by the Reverend Thomas Bayes of Tunbridge Wells, England, whose theorem was published in the Proceedings of the Royal Society in 1763.

With Bayes' theorem and a person's cognitive-performance specimen in hand, ushered in is a versatile cognitive- and statistical-science disciplined estimation of individual attributes of clinical interest. Predicted entities *A* actually can be as diverse as the parameter expressing model encoding-subprocess amount, or the symptomatology to which the mathematical model and estimated model parameter(s) relate (e.g., severity of thought-content disorder; elaborated upon, under *Dynamic assessment of treatment-regimen efficacy*, below). Importantly, Bayesian estimation makes for stabilization of estimated values, through the anchoring effects of mixing distributions, which act as "Bayesian priors" [e.g., Pr (A) , above]. Variance in estimates ("statistical inefficiency") thus is reduced through the formal device known as "Bayesian shrinkage". Altogether, estimation is solidified by feeding into its calculation specifically that information supplied by a pre-established referent, the Bayesian prior established by the mixing distribution.

The operation of mixing-distribution Bayesian priors can help alleviate the problem of "small-*N* mathematical modeling", ubiquitous in applied settings. The approach allows us to work with smaller sample sizes, which is particularly helpful when undertaking person-specific cognitive-performance modeling for assessment or research purposes. Valid mixing distributions supply a consolidating influence on estimates of model properties as they apply to the individual. They help compensate for small performance samples essentially by bringing into play performance-relevant information about the group to which the individual at hand belongs. Again, this information is conveyed by the mixing distribution(s) that quantifies the relative frequencies of the target(s) of prediction in a membership group. Such a scenario resembles that of a hematology laboratory, where a substantial extant bank of hematological information,

applicable collectively, is brought to bear individually on a modest blood sample from the person at hand.

Availed, moreover, is the dynamic assessment of changes in clinical condition, through undertaking the Bayesian estimation at designated times of clinical interest (e.g., after a selected bout of treatment). Specifically, equation (1) can be used to track changes in the status of symptom-related (e.g., thought-content symptomatology) cognitive-model parameters (e.g., stimulus-encoding-subprocess number, identified as inflated according to the model-adjustment operation of Figure 1). Changes can be monitored as they occur over the natural passage of time, over the course of treatment, or subsequent to an experimental manipulation. In these ways, the described formal methodology can be an important constituent in the arsenal of clinical assessment technology. Here, mixing-distribution Bayesian priors have replaced the usual actuarial standardization tables of multi-item psychometric inventories.

Bayesian individualization of model properties also allows for vetting of model performance at the person-specific level of model operation. Doing so ascertains model validity for an individual participant; it also affords strong tests of overall model performance. Fit of model predictions to empirical observations at both the group, and individual levels of data assembly, represents an added level of model evaluation. This unique form of model evaluation potentially bears on the currently prominent issue of robustness of findings in cognitive modeling (Neufeld & Cutler, 2019).

Dynamic assessment of treatment-regimen efficacy

With a modest expansion of equation (1), the present assessment methodology naturally extends beyond that of the individual; it can be applied to estimating the representation of

varying levels of symptom severity in a clinically treated cohort. With *A* of equation (1) now standing for cognition related symptom severity and given performance specimens from a random subsample of individuals in a treated cohort, changes in proportions of relative severity levels can be estimated and monitored repeatedly over time. The procedure loosely resembles one from mathematical ecology, where the stocks of various fish species are estimated according to netted samples, taken over the course of a fishing season. In the present case, the moving profile of symptom-severity proportions addresses the efficacy of the treatment regimen, in moving the treated cohort toward more healthy cognitive functioning. Note that inferences to the individual and treatment cohort levels are both centered on a cognitive, symptom-related mechanism (e.g., parameterized cognitive-encoding deviation). Such estimation is of special interest, for example where the administered treatment is a central-nervous-system directed drug treatment. Existing resources can be consulted for elaboration on mathematical and computational specifics, assumptions and methodological caveats of the assessment procedure described here (e.g., Neufeld, 2007a; Neufeld, Vollick, Boksman, et al, 2002; Neufeld, et al, 2010).

Implications for Clinical Functional Neurophysiology

Mathematical modeling of the cognitive side of vascular and electrophysiological cognitive neurophysiology conveys several methodological assets. Cognitive neurophysiology (aka functional, neurophysiology) comprises what technically are known as functional magnetic resonance (neuro)imaging (popularly, fMRI, for short), functional magnetic resonance spectroscopy (fMRS), magnetoencephalography (MEG), and electroencephalography (EEG). Certain of these divisions are returned to, and elaborated upon, below. Note for the present purposes that formally anchoring cognitive functions in a viable mathematical model represents

an antidote to a thorny problem in cognitive neurophysiology, known as reverse inference (Poldrack, 2011). This problem consists of circularly relying on measured neurophysiological signals to infer the cognitive functions whose very neuronal substrates purportedly are being charted. This inferential dilemma in principle can be overcome as follows. The cognitive functions at work while neurophysiological measurements are taken, are quantitatively stipulated in advance, anchored in a formal representation (e.g., Ahn, Krawitz, Kim, et al, 2011; White, Mumford & Poldrack, 2012). That is, cognitive functions whose neurophysiological substrates are being examined, are staked out in terms of a quantitative model—one that is *a priori* freestanding, independent of the examined neurophysiological activity itself.

Note, further, that dynamical models of cognitive operations treat the development of cognitive processes as stochastic functions of time (Townsend & Ashby, 1983). The unfolding of target processes, such as stimulus encoding, can be overlaid against monitored neuroimaging signals, producing neuroimaging times of measurement interest, complementing brain regions of measurement interest. (e.g., a region known as the encoding-intensive Dorsal Anterior Cingulate Cortex; Broadman Area 32). In this way, mathematical cognitive models can contribute to the calibration of space-time coordinates of neuroimaging measurement (illustrated in Neufeld, et al, 2010). Isolating critical times of target-process measurement has the advantage of allowing the target process (e.g., encoding of a presented stimulus) to function as it would alongside related processes involved in executing a cognitive task (e.g., comparing a presented stimulus to other stimuli held in memory). The approach, in other words, allows the target process to be examined as it operates *in situ*--as it were inside its cognitive ecological niche.

Estimating individual differences in model parameters, as described above, also can facilitate the formation of parametrically homogeneous groups. Reducing participant-group

heterogeneity potentially achieves greater statistical power for detecting subtle but key neurophysiological anomalies.

At a broader level, formal cognitive modeling can provide a cognitive-functional nexus for integrating observations from alternate domains of functional neuroimaging, investigative settings and experimental sessions. Ascertaining mathematically that the cognition at play remains stable across different sources of data lends assurance that neurophysiological results converge on a shared set of cognitive operations. For example, essentially a common mathematical model of stimulus encoding, as activated by a widely used cognitive task (called the Stroop Task), has been shown to cut across different levels of cognitive neurophysiological investigations. The investigative levels included first what is known as functional magnetic resonance spectroscopy (above), where neurochemical mechanisms accompanying cognitive performance are examined; and, second, vascular-signal functional magnetic resonance imaging, where the focus is on the specific neuronal circuits involved in performing the cognitive task (Taylor, Neufeld, Schaefer, et al, 2015; Taylor, et al, 2016, 2017).

All in all, by adopting the strategy portrayed in Figure 1, cognitive processing deviations can be identified and targeted, and the time course of the deviant processing during trials of an experimental task can be estimated. This time course then can be combined with measured activation of the brain region(s) apt to be involved in the suspected disorder-related cognitive process. The intended upshot consists of uncovering abnormality in neuronal operations paralleling abnormality in the targeted cognition. The combination of cognitive-functional and neurophysiological information on a disorder, in turn can profitably feed into clinical assessment and treatment activities.

Concluding Comments

Clinical mathematical psychology stands at the ready for further inroads into clinical science and assessment (see also Treat & Viken, 2010). Some may be put off by the requisite engagement in analytical developments, but advanced undergraduate and graduate statistics and design courses often place psychologists in a unique position to access available tutorials (see Recommended Readings). It is motivating to note that the history of science by and large is replete with exemplary advances hinging on decidedly formal theoretical developments ("necessary propositions"; e.g., Braithwaite, 1968; Harper, 2011). The transparency of predictions stemming from closed-form derivations moreover is intrinsically rewarding and self-vindicating, if rigorous, along with being self-indicting and potentially self-correcting if not.

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Declaration of Conflicting Interests

The authors declare that they have no conflict of interest with respect to authorships or the publication of this article.

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