

Clinical Mathematical Psychology

Matthew J. Shanahan,¹
James T. Townsend,² and
Richard W. J. Neufeld¹

¹Western University, Canada and ²Indiana University,
U.S.A.

Clinical mathematical psychology uses analytical (formula) derivations to describe behavioral, cognitive behavioral, or psychophysiological abnormalities associated with clinical disorders. The quality of being “analytical” differentiates mathematical modeling from statistical modeling, and computational modeling, or computer simulation. An analytical approach involves specifying the operation of the component variables in algebraic terms within formula structures representing the processes at work. Among the most valuable results of mathematical (analytical) modeling is the capacity for precise prediction of expected empirical response properties.

Examples of predictable empirical data properties include the categories into which responses fall (e.g., correct, incorrect; stated judgments, or choice selections), response latency, and intensity or expressed confidence in one’s registered response. A particular focus has been the measurement of *symptom-significant cognitive functioning*: cognitive activity that directly relates to pathological symptoms. These characteristic deviations in cognitive functioning are measured with approaches known as “cognitive psychometrics,” “cognitive modeling,” and “quantitative clinical cognitive science and assessment.”

Methods, Approaches, and Matters of Study

Essential parts of a psychological mathematical model typically encompass the explanatory

parameters (theoretical variables) it invokes, and its structure, or organization of the parameters, in terms of their functioning in the proposed theoretical response-generating process. As such, the development and application of a clinical mathematical model is typically done to fit the particular dynamics of a particular disorder, even a particular symptom or behavior proper to a given disorder. The model is usually “custom-built,” as it were.

On the other hand, much of psychological research makes use of analytical developments with an assumed normal (Gaussian, or bell curve) distribution when it comes to data analysis. Common Fisherian and Pearsonian statistical methods, and their contemporary extensions, have been developed through mathematical formulae using an assumed structure of the data. These approaches were developed in the early twentieth century, when the assumption of well-charted distributions facilitated the strength of statistical inference. Clinical mathematical psychology tends to select or develop relevant distributions that suit the phenomenon under study more closely.

An important feature of mathematical modeling is that it extends analytical developments from the data analysis enterprise out of reliance on verbal descriptions and into the realm of theoretical reasoning. The verbal theorizing combined with analytically developed statistical methods (e.g., analysis of variance, hierarchical linear modeling, chi-square) used in much of psychology research represents a “mixed deductive scientific system.” In such a system, the research question of interest is highly developed in verbal form in both the introduction and hypothesis statement. The mathematical test of the research question is often left to a stock method from a fairly short list of established, understood techniques. In contrast, the extension of mathematical formulations to theory and hypothesis generation makes for a “pure deductive scientific system.”

This kind of scientific system is characteristic of more established disciplines such as physics. The fundamental feature of a “pure deductive scientific system” is that its specification, testing, measurement, and vindication or falsification are conducted in a uniform language with unambiguous terms and clearly defined operations. Mathematical specification of research questions allows for this continuity of precise expression.

Clinical issues potentially subjected to mathematical modeling can range from very small-scale to very large-scale topics. At the level of an individual mind or brain, the study of cognitive functioning in clinical samples applies the rich body of information and measurement technology from the field of mathematical cognitive science to the analysis of cognitive abnormalities. At an interpersonal level, other avenues of modeling can take the form of, for example, quantitatively expressing clinically significant properties of continuous reciprocal influences between interacting parties, such as therapist–client interchange in a psychotherapeutic setting. At an even broader level, sources of vulnerability to stress and exacerbation of symptoms may be studied across a multiplicity of life settings with mathematical modeling approaches.

Clinical mathematical psychology generally implements stochastic rather than deterministic models. Stochastic models build a predictable amount of random variation into a predictive model. Models in hard sciences include this kind of variation routinely, where individual events are impossible to predict, but overall patterns are reliably described (e.g. weather patterns, molecular motion in a fluid). Doob (1953) has described a stochastic model as a “mathematical abstraction of an empirical process, whose development is governed by probabilistic laws” (page v). The stochastic aspect of model structure simulates naturally occurring random variation so as to expand deterministic model predictions, resulting in a distribution of empirical response properties (e.g., response latencies) over successive observations.

Statistical modeling concerns itself with theory of data structure for purposes of partitioning and analysis, and its mathematical foundations are not dependent on the research context. That is, the subject matter under scrutiny does not typically affect the analysis technique selected, beyond the selection of a well-known statistical test that suits the research design, rather than the research topic per se. Because hypotheses to which it is applied, by and large are nonformal (e.g., conjectured paths of influence in structure equation modeling, or moderation–mediation analysis), statistical modeling is part of a mixed deductive scientific system (a combination of verbal description coupled with a standard test of significance). As with any statistical analysis, focus is on treatment of the presenting empirical data. In contrast, the focus in mathematical modeling is on theoretical mechanisms held as responsible for bringing the data about in a direct fashion.

Computational models may be quite close to the procedures in analytic modeling but tend to emphasize incorporation of major assumptions into computer algorithms or Monte-Carlo software rather than through invention of explicit equations and the use of the latter in theorem and prediction development. Thus, in the case of computational modeling, specific rules of activation among its network of theoretical units (analogous, for example, to neurons, or neuronal modules; “neurodes”), are typically implemented through specific computer syntax and code. A network design and certain elements of its activation algorithm may correspond with selected properties of a mathematical model, such as its structure and parameters. Mathematical and computational models moreover may be mutually informative as to the mechanisms at play in a particular clinical process, or set of processes (Marr, 1982). The construction of the unit network, the strength of unit connections, and the unit-activation algorithm, nevertheless are not by and large themselves products specifically of mathematical derivations, or theorem-proof developments. It might be said

that though many such models can be crafted to generate predictive efficacy, as noted above, they are not explicitly mathematical in that they are typically constructed with “black box,” algorithm-based techniques rather than being designed with interpretable components by a human mind.

Procedurally, much of the work involved in clinical mathematical psychology resembles the method of titration in chemistry, where a reagent is introduced in careful measure to a substance to ascertain its composition. That is, many small increments or decrements must be made to the particular balance of the modeled quantities before the clinical model begins to reliably demonstrate the modeled behavior or cognitive phenomenon. The overall program involves theory building and theory correction in a dynamic interplay of modeling, clinical observation, and experimentation. Defensible models of normal functioning are modified to accommodate deviations occurring with psychopathology. Inferences are drawn from the titrated model: parts of the model that survive intact are taken to signify faculties that are spared; conversely, parts that are perturbed are triaged as signifying disorder-affected faculties.

As an example of clinical mathematical psychology applied to the study of schizophrenia-related cognitive deficits, the number of constituent cognitive operations or “subprocesses” has been shown to be involved in elongated encoding of presenting stimulation into a cognitive format facilitating further operations (e.g., in cognitively preparing and transforming a presented item for purposes of ascertaining its presence/absence among a set of earlier memorized items). Beyond the usual number of features encoded from the presented item (e.g., curves, lines, and intersections of an alphanumeric item), the individual with schizophrenia can spend an excessive amount of time tangentially transforming the presented stimulus. Increasing the number of subprocesses beyond a certain threshold in computer-assisted model simulations allows the model-generated data to closely approximate the response times obtained from

actual clinical participants. As such, the aspect of excess subprocessing is vindicated as an effective model of cognitive processing for this population.

To elaborate slightly, selected stochastic models addressing this deviation express mean process latency simply as the quantity k/v . Here, k is the number of constituent operations of the encoding process, and v is the rate of subprocess completion per unit time (“subprocess workload capacity”). Fitting predictions to schizophrenia data has occurred with elevation in k , but not a decrement in v (e.g., Neufeld, Boksman, Vollick, George & Carter, 2010).

Typically, it is parameter values, rather than model structure that need to be changed. In some cases, however, the architecture of the modeled cognitive behavioral performance mechanism itself must be modified, in order to accommodate observed performance deviations. The technique of theory building and adjustment aligns with the scientific strategy called “abductive reasoning,” whereby existing (mathematical) theory explanatorily is retrofitted to obtained data.

Prominent issues in clinical science and assessment, notably from the cognitive behavioral domain, surround the following: cognitive workload capacity, efficiency of capacity deployment, the presence and nature of stages of processing, serial versus parallel transaction of process operations, and so-called automatic versus controlled (effortful) processing. Some of these have been addressed by existing research, or are addressable via clinical mathematical psychology approaches. Symptom-significant deviations in clinical populations (cognitive patterns likely contributing to the pathology in question) can be approached through established methods for measuring information processing, decision and choice, memory processes, concept learning, perceptual organization, and psychological stress and coping.

Contemporary developments in mathematical psychology, that also have found a home in clinical science and assessment, have provided for parameter-free, distribution-general model

composition and empirical process diagnostics (e.g., Townsend & Nozawa, 1995; Townsend & Wenger, 2004). These theoretical formulations emanate from mathematical axioms that are universal (“distribution-free”) rather than based on an assumption of specific distributions such as the normal (Gaussian distribution). There exists associated measurement technology (known as Systems Factorial Technology) that discerns fundamental elements of cognition (e.g., serial versus alternate parallel-processing architectures of a hypothesized cognitive system, and its cognitive workload capacity).

Such developments are a unique contribution to the broader field of mathematical modeling, and their general robustness is an especially welcome feature in applied settings. Moreover in the field of psychology, they epitomize the ideal extolled by Meehl (1978), that theory testing, including the measures it uses, should emanate from the theory itself, again as seen in older scientific disciplines. In the science of particle physics, for example, the theory-driven prediction of the Higgs boson was so well supported from a foundation of mathematical inference that it justified massive resource investment in the Large Hadron Collider at CERN, Switzerland. Effective mathematical psychology theory should be able to generate novel, significant predictions and permit relevant hypotheses to be specified accurately and tested with precision.

Contributions of Informational Added Value

A presenting epistemic strategy or measurement technology will usually “carry its own credentials,” vindicating itself based on the informational “added value” it conveys. Such a payload delivered by clinical mathematical psychology is multifaceted. It includes construct-representation construct validity, the disentangling of processes conflated in generic data analysis, explanation and unification of seemingly disparate empirical observations,

and accessing otherwise intractable, or nonaddressable sources of clinical disturbance.

Construct-representation construct validity represents support for the interpretation of a measure in terms of the degree to which it incorporates mechanisms that are theoretically meaningful in their own right (Embretson, 1983). The theoretical infrastructure in mathematical modeling springs from the very subject matter under study. Analytical formulae of mathematical models have “working parts” that are in themselves substantively meaningful. In this way, construct representation is built into measures to which the modeling gives rise.

Mathematical theory, and the measurement methods it spawns, can be used to refine the treatment of research data so as to uniquely distinguish the operation of cognitive behavioral performance mechanisms among clinical groups. To illustrate, Riefer, Knapp, Batchelder, Bamber, and Manifold (2002) studied recall memory performance among individuals with schizophrenia and controls. The design was a “correlational experiment” whereby the differing groups are examined under varying conditions of theoretical interest. The conditions in this case were sequential trials of a recall paradigm, each consisting of an item-study phase, followed by a recall-test phase. Of considerable interest was the differential progression of performance over the successive trials.

Analysis of variance conducted on sheer proportions of items recalled nevertheless failed to yield a significant groups-by-trials interaction. Ordinarily, such nonfruitful traditional analyses would end with the study being “dumped.” However, careful mathematical modeling revealed important differences between the groups. In-depth analysis of storage and retrieval processes through a mathematically disciplined model of performance (known as multinomial processing tree modeling) exposed the epiphenomenal nature of the generic analysis. There was apparently an intrinsic offsetting whereby two existing effects “cancel out.” This pattern could only be

discerned, however, by the authors' application of mathematical modeling.

The use of model-driven measurement and statistical testing (part of what Riefer and colleagues term "cognitive psychometrics") revealed that the schizophrenia participants showed less improvement in storage accuracy specifically over the last three of the six study-retrieval trials. Contra this deficit, their improvement in storage accuracy actually exceeded that of controls during trials 2 and 3. Analysis of a model parameter distinguishing rate of improvement in retrieval accuracy, as set against the trial 1 baseline, showed a significantly slower rate throughout. The model-identified initial strength of storage accuracy evidently diluted later deficits, as well as those of a lower retrieval-improvement rate, to render superficially parallel the between-groups changes seen in the analysis of raw proportions of items recalled. As such, mathematical modeling suggested a more fine-grained re-examination of the data based on component processes, allowing the discrimination between a target clinical group and a control group that would have been, for all intents and purposes, invisible.

Accurately identified profiles in process strengths and weaknesses in principle can contribute to the navigation of clinical intervention strategies. They also can furnish important information about the "functional" side of functional neuroimaging measurement obtained during memory task performance.

As occurs in other applied mathematical sciences, informational added value, as bestowed by clinical mathematical psychology, can explain and unify enigmatically disparate empirical findings. Such unification can be illustrated with reference to observations on sensitivity to threat-valenced stimulation among anxiety-prone individuals.

Greater attention to threatening stimulus content (e.g., words such as "suffering" or "loss") among higher anxiety-prone (HA) in contrast to lower anxiety-prone (LA) individuals has been found across multiple experimental paradigms such as the Stroop

task, dichotic-listening tasks, and the dot-probe task. In the latter, HA individuals detect the probe more readily when in the vicinity of a threatening versus neutral visual stimulus.

The consistency of significant HA-LA group differences, however, has generally been tied to presentation of threat items *alongside* non-threat items. Such differences disappear when threat and non-threat items are present *singly*. Statistical significance of the HA-LA by threat-non-threat item interaction (significant group-item second-order difference, or, two-way interaction) is by and large restricted to conditions of co-occurrence of the two types of stimuli. Such findings have given rise to relatively complex conjectures emphasizing competition between the two types of stimuli and associated "cognitive control operations." Differences between HA and LA groups have been attributed, for example, to inequalities in cognitive tagging of threat items, or to HA participant deficit in threat-item disengagement. Difficulties in registering significant second-order differences with single-item presentations has led to questioning the potential importance, or even existence, of HA individuals' elevated threat-stimulus engagement.

The resolution of this research conundrum is that greater threat-stimulus sensitivity arguably exists among HA individuals, but this may be more strongly drawn out with the co-presence of non-threat stimuli—for reasons that are relatively straightforward. The cognitive apparatus transacting the processing task stands to be one of limited attentional resources, in this case meaning that these resources are spread between threat and non-threat items, when co-present. Items presented together furthermore arguably are processed in parallel (concurrently) among both HA and LA participants. With relatively less salience of the threat item, a greater, and possibly equalizing amount of LA participants' attentional capacity theoretically is consumed by the non-threat item, reducing the between-item difference in processing latency. In contrast, the inter-item difference would be larger for

the HA participants, if the pre-potent salience of the threat item made for increased resistance to the non-threat item's encroachment on processing capacity.

This proposition lends itself to the following simple numerical illustration. Brought into play is a defensible processing apparatus, called an independent parallel, limited-capacity (IPLC) processing system, with exponentially distributed item-completion times. Its technical specifics notwithstanding, the workings of this system make for straightforward inferences regarding the present issue. Let the visual information-processing resources of such a system (its cognitive workload capacity) be expressed as a value of 10 arbitrary units (corresponding to the *rate* at which task elements are processed, per unit time), for both HA and LA participants. During a dot-probe task (above; particularly prominent in the present research domain), the solo presentation of a threat item fully engages the system resources of an HA participant, and possibly 90% thereof in the case of an LA participant. By the workings of this model, the mean processing latency of HA individuals is $1/10$ and that for LA individuals is $1/9$, for a difference of $-.0111$ (note that latency varies inversely with capacity, expressed as a processing-rate parameter). The solo presentation of the non-threat item, on the other hand, putatively engages 50% of the system's resources for both participants, which by the model translates into $1/5$ for both individuals, now making for a zero difference. By the IPLC model, the second-order difference in mean latency (or inequality of the differences in the means)—this being targeted in the above studies—then is $(1/10 - 1/9) - (1/5 - 1/5) = -.0111$.

For the simultaneous-item condition, 80% of the HA individual's system processing resources hypothetically remain with the threat-item location, but the LA individual's resources now stand to be evenly divided between the threat and non-threat items. By similar reasoning to the above, and retaining a capacity limitation of 10 arbitrary units, the second-order difference becomes

$(1/8 - 1/5) - (1/2 - 1/5) = -.375$. Statistical power obviously will increase with the effect size accompanying this larger second-order difference.

These developments exemplify clinical mathematical psychology's potential to explain and unite seemingly discordant sets of observations. Potential sources of discrepancy are uncovered, and disparate findings are synthesized by a common underlying process.

In addition to generating new measures, experimental paradigms, and formal-theoretical explanations, clinical mathematical psychology increasingly has been used to enrich the informational yield from tasks already routinely used in clinical science and assessment. A type of dynamical mathematical modeling, known as Expectancy Valence Learning, and its variants have been used to dissect and specify sources of performance deviation on tests such as the Wisconsin card sorting test, the Iowa gambling task, and the go/no-go task. Performance abnormalities are pinpointed to sources that are motivational (differential sensitivity to positive versus negative feedback to individual decisions), learning-related (relative influence of recent versus more remote choice outcomes), and response-related (entrenchment of responding in acquired information, versus impulsive dislodgement therefrom).

In some areas of investigation, the acquisition of clinically important information may be untenable apart from mathematical modeling. It has been suggested that analogous to selected methods in biochemistry, where a compound is considered to be understood if successfully synthesized, some aspects of psychopathology are grasped if reproduced experimentally among nonclinical samples. Certain proposed agents of performance decline, however, may defy experimental manipulation of independent variables as the sole means of study. Such may occur for ethical reasons, because of the intensity of experimental manipulations required, or because the theoretically tendered agent may elude mimicking through experimental induction (e.g., organismic endogenous

stress susceptibility put forth as producing task-impeding intrusive associations, or siphoning off cognitive workload capacity).

The direction taken by clinical mathematical psychology instead is one of introducing the suspected agent into model composition, and examining for increased conformity of predictions to deviations occurring with psychopathology. Additionally, a considerable economy of data-harvesting resources can be realized with well-designed modeling approaches. Moreover, some large-scale patterns may only be revealed in extensive simulation runs of well-tested models, where empirical data gathering would be prohibitive or impossible.

Clinical Mathematical Psychology and Clinical Neuroimaging

Clinical mathematical psychology can provide vital information on the functional aspects of clinical functional neuroimaging (functional magnetic resonance imaging, magnetic resonance spectroscopy, electroencephalography, and electro-magnetoencephalography). It can do so through stipulating precisely the cognitive events taking place during cognitive neuroimaging trials. Symptom significance is associated with monitored neurocircuitry by applying models of neuroimaged cognitive activities that intersect with symptomatology (e.g., the quantitative interlacing of formally modeled stimulus-encoding deficit in schizophrenia, with thought-content disorder; Neufeld et al., 2010).

A frequently used method of neuroimaging called event-related neuroimaging is an important method of neuroimaging. In this approach, the specific cognitive functions that are instigated by experimental manipulations of a cognitive performance paradigm are studied. Estimating the time course of such functions within a cognitive performance trial demands a tenable mathematical model of their stochastic time trajectory. Imaging signals which correspond to estimated epochs of a symptom-relevant process (e.g., encoding

a presented item, for purposes of further treatment, such as comparing it for a match to other items held in “working memory”) command special attention. Clinical mathematical modeling can specify such epochs. In doing so, it can supply “times of measurement interest” that complement “brain regions of interest,” thus rounding out the navigation of space–time coordinates of functional neuroimaging measurement (mathematical and neuroimaging specifics are presented in Neufeld et al., 2010).

Using modeled time periods of interest for measurement, symptom-relevant functions can be analysed close together with other functions to which they relate. For example, item encoding remains conjoined with, say, scanning for the item in a memory-held item-set, and possibly other processes, for which the encoding process exists. Retaining the integrity of the experimental context in which the targeted cognitive process operates preserves its functioning in situ, or its “ecological validity” as one component process among many required. As a result, ecological validity of findings may be enhanced, over and against those obtained by deconstructing the performance apparatus through experimentally extracting constituent processes, and attempting to study them in isolation.

An epistemic issue vexing clinical and other functional neuroimaging is that of reverse inference. Reverse inference occurs when the cognitive functions whose neurocircuitry is being charted are inferred from the monitored neurocircuitry itself. Stipulating a target function in quantitative terms definitively anchors the entity whose neurophysiological substrates are being probed at the cognitive behavioral level of analysis, rather than at the temporal–spatial co-occurrence of neurocircuitry signals.

Dynamical Monitoring of Individual Treatment

Monitoring cognitive behavioral task performance can be used to ascertain individual

functioning over a course of treatment. Estimation of model properties pertinent to information processing, memory, perceptual and other faculties of mentation can provide cognitive science principled assessment of progress.

Tracking treatment response is potentially expedited by uploading the performance model onto a Bayesian statistical platform. Doing so exploits tenable theoretical distributions of individual differences in model parameter values among demographic or diagnostic populations of clinical interest (known as parameter mixing distributions, hyperdistributions, or Bayesian priors). Expanding clinical mathematical modeling to a hierarchical design, which strategically provides for individual differences, brings into play several noteworthy advantages from a clinical perspective.

Importing pre-existing information lodged in Bayesian prior distributions of model parameter values allows for relatively precise parameter estimation for a specific individual, using only a modest specimen of his or her task performance (a statistical property known as “Bayesian shrinkage”). Modesty of task demands may be considered especially welcome when dealing with already distressed individuals. Integration of individual performance with prior information, through Bayes’ theorem, is analogous to bringing to bear on a small blood specimen rendered in a hematological clinic the full bank of pre-existing hematological assay knowledge.

Along with charting the individual’s parameter estimates as treatment proceeds, the person’s relative position with respect to varying symptomatic and asymptomatic groups can be tracked in a similar, dynamically adjusting fashion. An individual performance sample can be subjected through Bayes’ theorem to prior parameter distributions preliminarily crafted to benchmark groups of interest. This procedure is analogous to the actuarial clinical practice of referring scores from a multi-item psychometric test or inventory to selected standardization samples (e.g., normed

scores for age and gender on a scholastic achievement test help generate more informed expectations against which to test a recorded individual performance sample). In the present case, profiles of probabilities of belonging to the respective symptomatic and asymptomatic groups, given updated performance specimens—altogether yielding profiles of “Bayesian posterior probabilities”—thus can be probed at desired time points.

Similar dynamical assessment of treatment regimens also presents itself. The above cognitive and Bayesian statistical methodology can be expanded to assess whether a treatment program is edging its treated sample closer to healthier cognitive functioning. Such examination of treatment regimens becomes especially relevant to evaluation of central nervous system-related pharmacotherapy, as pharmaceutical companies increasingly have expressed interest in effects of such treatment agents specifically on cognitive efficiency (mathematical developments and computational specifics with empirical illustrations are presented in Neufeld et al., 2010).

Integration with Other Practices in Clinical Science and Assessment

Clinical mathematical modeling and multi-item psychometric measures can selectively complement each other. Empirical associations between model properties and scores from multi-item measures can augment one another’s nomothetic-span construct validity (Embretson, 1983). Correlations with model properties also may increase construct-representation construct validity of multi-item measures. Psychometric measures in turn could be used as proxy measures for model properties with which they sufficiently correlate, potentially resulting in savings of assessment resources (but cf. Bayesian estimation, above).

In addition, mathematical modeling can throw light on processes involved in responding to psychometric items. Specifically, modeling of item selection through item-response

theory can be complemented by modeling item-response time, viewed as a product of a stochastic dynamic process (Neufeld, 1998).

Clinical mathematical modeling also speaks to a longstanding issue of substantive inference, which is interlaced with measure-theoretic considerations. The problem is the so-called differential-deficit, psychometric-artifact confound. False inferences of differential cognitive deficit across alternate aspects of cognitive behavioral functioning are risked because differences across the addressed faculties, between clinical and control groups, are conflated with psychometric precision of instruments used to measure the faculties. More and less pronounced apparent differences actually may be attributable in good part to more and less group-discriminating psychometric measurement properties. The problem retains currency in clinical science. Recommended solutions using psychometric calibration of constituent measures (equating them for reliability and observed-score variance) have been challenged both on mathematical and empirical grounds. It has been argued instead that assigning components of psychometrically partitioned variance to mathematically modeled sources lends a model-based substantive interpretation to the components and renders the psychometric-artifact problem essentially obsolete. Partitioned variance now is fully prescribed according to a governing cognitive performance model. This model-partitioned variance includes classical measurement error variance (now across-trial, within-participant variance); within-group, interparticipant variance; and between-group variance (see Further Reading).

Future Directions and Challenges

Clinical mathematical psychology is increasingly represented in both clinical and mathematical psychology journals. Its application is arguably vital to expediting theoretical and empirical progress in clinical science and assessment, including the resolution of important, longstanding and intractable issues. It has

been stated that, as seen in other sciences, it is inevitable that psychological science will come to rely more and more heavily on mathematical modeling.

Psychology has witnessed repeated calls in its periodicals and newsletters for more quantitatively trained graduates. Traditional education in quantitative methods may or may not include mathematical psychology, but those trained in the latter almost always are versed in mathematical and psychological statistics. A strong case can be made for both an increasing role of mathematical psychology in the discipline at large, as well as the broader credentials carried by mathematical psychologists. For those lacking a formal background on the subject, mathematical psychology's inroads are accompanied by an ever-greater number of reference resources, tutorials, and workshops (see Further Reading).

The growing presence of mathematical psychology in clinical and nonclinical psychology seems in line with the esteem in which mathematical psychology is evidently held. Six of the sixteen U.S. Presidential Medals of Science awarded to psychologists have been given to mathematical psychologists, a figure far out of proportion to the representation of mathematical psychologists in the discipline. Regarding the two Nobel Prizes in Economics awarded for seminal psychological work, Herbert Simon (1978) and the late Amos Tversky (whose work on prospect theory with prizewinner Daniel Kahneman was recognized in 2002) were mathematical psychologists.

Ongoing challenges involve the productive integration of "cold and hot cognition." Roughly, cold cognition pertains to mechanisms of information processing and hot cognition pertains to inferences yielded, especially with respect to their semantic and affective attributes. Deviations in mechanisms of processing leading to dysfunctional representation of environmental events and personal significance can be of special clinical interest. Research done by Teresa Treat and colleagues on eating disorders and risk of sexual coercion exemplifies the integration of

symptom-meaningful hot and cold cognition (e.g., Treat & Viken, 2010).

Areas of investigation relatively untapped by mathematical modeling include mood and anxiety disorders. The documentation of symptom-relevant information processing in these families of disorders is poised to benefit from a quantitative charting of adverse changes in cognitive efficiency. Implications for intervention stand to follow, such as the disclosure of new, model-informed routes to treating and assessing symptom-relevant information processing (e.g., reduction in the ascendancy of personalized negative associations).

Clinical mathematical psychology also raises the prospect of substantively driven meta-analysis techniques. Substantively driven meta-analysis entails explanatorily retrofitting available (mathematical) theory to pre-existing data, a bona fide scientific methodology known as “abductive reasoning.” Extracting model-based substantive information lodged in the literature’s data, much of whose format derives from a non-modeling perspective, remains an ongoing but important challenge.

It has been said that evidence-based practice is not best practice if it is not based on the best evidence. Best evidence implies maximizing the armamentarium of investigative options, notably including mathematical psychology adapted to the clinical arena. The complex nature of psychopathology and its treatment beckons the increased use of mathematical approaches as a sturdy platform for launching reliable and cohesive research agendas for the study of psychological illness and distress.

SEE ALSO: Bayes’ Theorem; Bayesian Analysis; Construct Validity; Differential Deficit; Evidence-Based Assessment; Item Response Theory; Kuhnian Paradigms; Meehl, Paul E. (1920–2003)

References

- Doob, J. L. (1953). *Stochastic processes*. New York: John Wiley & Sons.
- Embretson, W. S. (1983). Construct validity: Construct representation versus nomothetic span. *Psychological Bulletin*, 93, 179–197.
- Marr, D. (1982). *Vision*. San Francisco, CA: Freeman.
- Meehl, P. E. (1978). Theoretical risks and tabular asterisks: Sir Karl, Sir Ronald, and the slow progress of soft psychology. *Journal of Consulting and Clinical Psychology*, 46, 806–843.
- Neufeld, R. W. J. (1998). Intersections and disjunctions in process-model applications. *Psychological Assessment*, 10, 396–398.
- Neufeld, R. W. J., Boksmen, K., Vollick, D., George, L., & Carter, J. (2010). Stochastic dynamics of stimulus encoding in schizophrenia: Theory, testing, and application. *Journal of Mathematical Psychology*, 54, 90–108.
- Riefer, D. M., Knapp, B. R., Batchelder, W. H., Bamber, D., & Manifold, V. (2002). Cognitive psychometrics: Assessing storage and retrieval deficits in special populations with multinomial processing tree models. *Psychological Assessment*, 14, 184–201.
- Townsend, J. T., & Nozawa, G. (1995). Spatio-temporal properties of elementary perception: An investigation of parallel, serial, and coactive theories. *Journal of Mathematical Psychology*, 39, 321–359.
- Townsend, J. T., & Wenger, M. J. (2004). The serial-parallel dilemma: A case study in a linkage of theory and method. *Psychonomic Bulletin & Review*, 11, 391–418.
- Treat, T. A., & Viken, R. J. (2010). Cognitive processing of weight and emotional information in disordered eating. *Current Directions in Psychological Science*, 19, 81–85.

Further Reading

- Bussemeyer, J. R., & Diedrich, A. (2010). *Cognitive modeling*. New York: Sage.
- Neufeld, R. W. J. (2007). Composition and uses of formal clinical cognitive science. In B. Shuart, W. Spaulding, & J. Poland (Eds.), *Modeling complex systems: Nebraska Symposium on Motivation* (Vol. 52, pp. 1–83). Lincoln, NE: University of Nebraska Press.
- Neufeld, R. W. J. (Ed.). (2007). *Advances in clinical cognitive science: Formal modeling and assessment of processes and symptoms*. Washington, DC: American Psychological Association.
- Townsend, J. T., & Ashby, F. G. (1983). *Stochastic modelling of elementary psychological processes*. Cambridge, U.K.: Cambridge University Press.