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Neural Network Learning Theory Integrates Behavior Therapy and Behavior Genetics

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Open Forum

Visiting La La Land: A Reply to Weissberg and Owen

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Steve Hayes penned a thought-provoking piece titled "Resisting Biologism" that appeared in Volume 21 of tBT *(pp. 95-97). Weissberg and Owen's rebuttal appeared in Volume 22 of* tBT *(pp. 102-107). The following four submissions are part of an ongoing dialogue that stem from those two articles. Readers may wish to consult the original articles for a detailed understanding of the complexities associated with this important debate.* **The Editor**

Weissberg and Owen (1999) criticize my analysis of the threat of biologism to behavioral science. However, their criticism leaves my analysis untouched, and in fact provides further evidence of its legitimacy.

Weissberg and Owen defend behavioral genetics and its right thinking and see my article as an uninformed attack. In order to make this claim, however, they put words in my mouth. They complain that I "trash the entire enterprise" of behavioral genetics (p. 102), and that I "confuse behavioral genetics with biologism" (p. 107), when in fact I do not even mention behavioral genetics in the article.

As I will show, their other seemingly more substantive points never really rise above this level, which leads me to conclude that their core issue is merely one of protecting the old school flag, whether or not I even mentioned it. For example, they claim that I "seem to believe that if one concludes that the variability of a particular characteristic is primarily attributable to genetic variability, that implies that any intervention short of direct genetic manipulation is doomed to failure" (p. 102). "Seem to believe" is a weasel term used when the author being criticized has created a serious inconvenience for a critic, namely, not saying what is about to be criticized even though the critic dearly wishes the author would have done so. That is the case here, except that my point was the exact opposite, and I gave a concrete example of plastic surgery removing most of the documented negative behavioral effects for a particular characteristic that is primarily attributable to genetic variability, namely, physical attractiveness. Weissberg and Owen go so far as to say that my "underlying but unstated theme" (p. 102) is that genetic variation mandates a specific outcome. You know you are about to go from weasel words to la la land when what is unstated in a written article is supposedly incorrect, but this instance is particularly preposterous. In my article I repeatedly point out that when we are told that outcomes are due to genetic variation, we are "not being told that the functional processes involved are dominantly biological" (Hayes, 1998, p. 97) and that other "manipulable processes" (p. 97) may be involved.

Neural Network Learning Theory Integrates Behavior Therapy and Behavior Genetics

Warren W. Tryon, *Fordham University*

This article makes several preliminary points prior to presenting two examples of how Neural Network Learning Theory (NNLT) integrates behavior therapy and behavior genetics. I will argue that NNLT is both a cognitive and behavioral theory; that neither cognitive nor behavior theory informs us about biological, including genetic, factors; and that NNLT provides an understanding of how biological, including genetic, factors influence psychology and behavior. I use the term integration to mean that NNLT provides a single vocabulary and set of explanatory concepts consistent with the broad spectrum of behavioral and cognitive therapies that informs us as to how genetics can regulate both psychological and behavioral processes. My comments are intentionally general. I do not suggest that most or all of the desired information is at hand. Nevertheless, enough is known to conclude that NNLT is an extraordinarily promising theory that deserves close attention by all psychologists, especially behavior therapists.

NNLT is Both a Cognitive and Behavioral Theory

The vast majority of behavior therapists today are cognitive behavioral in orientation because behaviorism (operant and respondent conditioning) was, and is, viewed by many behavior therapists as theoretically insufficient to serve as a satisfactory basis for either empirical research or clinical practice. A difference of opinion regarding the validity of this statement is the cognitive behavioral debate. It is a corrosive theoretical schism that continues to divide behavior therapists and complicate training students. I have previously demonstrated that Neural Network Learning Theory (NNLT) resolves the cognitive behavioral debate because it is completely consistent with all positive values of behaviorism, including Skinner's radical behaviorism, and completely consistent with all positive values of cognitive theory (Tryon, 1995a, 1996). This is a genuine Hegelian synthesis constructed from thesis (behaviorism) and antithesis (cognitive theory) (or vice versa) and not mere eclecticism. Neither side need compromise any important theory construction value from the NNLT perspective.

NNLT is a cognitive theory because it was developed by cognitive psychologists to explore the "microstructure of cognition" (Rumelhart, McClelland, & Hinton, 1986, pp. 12-13). NNLT is a behavioral theory because it is highly compatible with behaviorism (Donahoe, 1998; Donahoe & Palmer, 1989; Tryon, 1995a, 1995b, 1996), and because many successful connectionist neural network (CNN) models of operant and respondent conditioning have been developed (Commons, Grossberg, & Staddon, 1991; Hawkins, 1989). Donahoe and Palmer have noted the compatibility of connectionism with the experimental analysis of behavior. Donahoe explains reinforcement in terms of selection, as does Tryon (1993), and also provides a CNN model of reinforcement that simulates acquisition, extinction, and reacquisition. NNLT is also a biological theory because it is based on fundamental principles of neural functioning (McLeod, Plunkett, & Rolls, 1998, pp. 11-15).

Resolution of the cognitive behavioral debate is a major contribution of NNLT. This achievement is important to the present discussion in that it demonstrates that we will be extending this cognitive

Weissberg and Owen agree that twin studies cannot tell us whether a behavioral outcome is due to genetic factors, and that "to say that genes influence behavior is to say nothing about the process by means of which that influence exerts itself" (p. 102). So far so good. They then reformulate the question and assert that twin studies do answer their preferred form ("Such studies can provide estimates of the proportion of the variance. that is attributable to differences among these individuals in their genetic endowment, their environmental experiences, and the interaction of the two." p. 102). Other than the substitution of "attributable to" instead of "due to" this is functionally identical to the approach I criticized. As I pointed out, twin studies simply cannot answer this question except within the data analytic model applied to studies in which words like "environmental experiences" assume meanings that correspond to little or nothing of practical or theoretical significance. In my artcle I gave examples of outcomes attributable to genetic variation that were in fact primarily or entirely due to universally available environmental contingencies that are reliably engaged by these differences (my point on physical attractiveness). In the ideal twin study, environmental experiences are reduced to gross differences across families across children as compared to differences between children within families. This is a definition of environmental experience that corresponds to almost none of the connotations of the term. The method is eminently logical but inherently misleading because almost no one developing a policy based on this method will understand how deeply the method hides what is being compared.

I was careful not to talk about behavioral genetics per se in my paper, in part out of respect for my friends and colleagues in that area, but since Weissberg and Owen have raised that issue I will address it. Many good behavioral geneticists understand the specific intellectual point I was making, and indeed the epigeneticist camp has championed these points and others related to them. Where the rub comes is that most feel there is still considerable value in these limited methods, despite the tendency for the results to be almost universally misunderstood by students, other professionals, policy makers, and the public. More than once I have had the experience of exploring this territory with world class behavioral geneticists, reaching grudging agreement, and then watching how moments later they are drawn back- bell curve fashion-into self-deceptive state-

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behavioral unification to include behavior genetics.

Isolation From Neuroscience

Neither behavioral theory nor cognitive theory makes serious theoretical contact with neuroscience. Cognitive theory is as much of a black-box approach to psychology and behavior when it comes to neuroscience as is Skinner's experimental analysis of behavior. Cognitive theory attributes psychological functions to the organism without any explanation as to how these functions are instantiated biologically. Learning and memory are presumed rather than explained. The boxes and arrows in contemporary cognitive theories indicate how various psychological states (cognitive and emotional), are related to behaviors without understanding what happens when learning occurs or how memories are formed and recalled. The lack of learning and memory mechanisms transforms cognitive models black-box and arrow models; diagrams that impute causal properties among cognitive processes that are not understood and cannot be explained.

Barlow (1997), Foa and Kozak (1997), and Wilson (1997) have called for theoretical integration with neuroscience. However, not all behavior therapists share this view. Hayes (1998b) argued that bio-

logical, psychological, sociological, anthropological, etc., explanations are each legitimate but distinct perspectives, thereby precluding the possibility of rendering one in terms of another, such as a biological (genetic) explanation of psychological and/or behavioral events. Rychlak (1993) previously argued for the independence, but complementarity of, four explanatory bases, which he termed physikos, bios, logos, and socius. Physikos appeals to physical properties, bios to biological properties, logos to logical and psychological properties, and socius to social factors. Rychlak asserted that no one perspective can be given in terms of another and therefore they must remain distinct but complementary. Tryon (1994) indicated that NNLT synthesizes the first three of these explanatory sources. Read and Miller (1998) indicated that all four explanatory bases may be integrated into a single theoretical perspective. This greater theoretical synthesis is another major contribution of NNLT. The unification of bios and logos is the focus of the remaining discussion.

Interaction Versus Biologism

Hayes (1998b) specifically, and I believe correctly, warned against a partic-

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son why such an error is inevitable and cited favorably his PKU example in which the real world consequences of this genetic anomaly are dramatically altered by the right environment. In citing our use of his PA example, Hayes accuses us of attributing to him the exact opposite of what he said. In re-reading what we wrote, we understand how our text could invite such an interpretation. But Hayes discussed PA in the context of his effort to demonstrate the "mistaken logic" of twin studies. Our comment was not intended to refer to what Hayes believes about the functional processes that affect PA but about what he believes behavioral geneticists believe about it. The main thrust of his argument is that when a twin study reveals behavioral variance that is significantly attributable to genetic variability, that fact compels a set of false and misleading conclusions. We do not deny that incorrect conclusions may be drawn; however, we do not believe that the logic of the method makes such conclusions inevitable. The logic of a twin study is defined by its design and accompanying statistical analyses. The substantive interpretations one draws from such studies are extrastatistical. We cited textbooks (Plomin, DeFries, & McClearn, 1990), articles (Plomin, Owen, & McGuffin, 1994), reviews (Rose, 1995), and examples (intelligence) in which the point was repeatedly made that to say that genes influence behavior is not to say: (a) that the phenotypic outcome is necessarily attributable to those genes; and (b) that the phenotypic outcome is predetermined. The exquisite irony of this exchange is that we agree on the limits of the implications that surround a finding of genetic influence; we disagree on whether certain designs ineluctably lead to false conclusions; and we disagree on whether reputable behavior geneticists consistently overinterpret their findings. Indeed, those who do have recently been called to task (cf. Turkheimer, 1998). More than this, we specifically made the point (p. 102) that it is behavioral genetic research that has highlighted the fact that environmental factors play an indispensable role in the etiology of all behaviors—including, most especially, behaviors that are strongly influenced by genetic factors. (Isn't that the point of the frequently cited finding that the concordance rate for schizophrenia among MZ twins is less than 100%?)

Hayes is also critical of human BG studies because they do not regularly attempt to identify the functional processes that underlie the outcomes they examine and because they lack specificity in their descriptions of environmental experiences. These are valid criticisms, and Hayes is right to call for more research that identifies the relationships among specific behaviors, specific environmental experiences, and specific genetic endowments. But it is an exaggeration to say that this issue is unaddressed in mainstream research. For example, while phenotypic intelligence, as indexed by IQ scores, is highly inheritable, there also is substantial evidence of its malleability. For example, Locurto (1990) estimates that children born into lower class families but adopted into higher class families experience an IQ gain of about 10 to 12 points. Similarly, in a follow up of the Minnesota Transracial Adoption Study, Weinberg, Scarr, and Waldman (1992) found that the positive effects of being reared by intelligent adoptive parents were maintained over a 10-year period. One might argue that these findings are provocative, but unhelpful, because the differences between lower class and middle class families were not identified with a level of specificity that would satisfy even a

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ular form of biological explanation he called "biologism," which he defined as "... the belief that the structure of the organism or its parts fully explains its contextually situated actions" (p. 95). He cited instances where findings that the inner ears of lesbians differ from those of heterosexual women are used to explain sexual orientation, and that smokers differ in a nucleotide sequence from nonsmokers, which is used to explain smoking status as examples of biologism. He drew attention to the interplay between biology and behavior and used the impact of physical features that influence attractiveness judgments on behavior as an example. Hayes (1998b) recommended that we ask "interactive questions," where the effect of environmental events on biological variables that influence behavior are posed and answered by empirical study. Recognizing current theoretical and methodological limitations, Hayes concluded that these are "... demanding questions that are mostly beyond our current reach..." (p. 96), indicating that contemporary behavioral, cognitive, and/or psychodynamic theories do not inform us about how biology influences psychology and behavior, nor how experience alters biology, as noted above. The absence of a truly psycho-biological and biopsychological theory leaves a schism between psychology and biology that distorts discussions about behavior genetics. This theoretical deficiency is part of a more general problem that Hayes (1998a) described as the theoretical emaciation of behavior therapy. Two special issues of *Behavior Therapy* (Vol. 28, Nos. 3 & 4) further document and discuss the theoretical problems behavior therapists face. NNLT eliminates this problem because it explains learning, memory, and the full range of psychological and behavioral phenomena that result from the developmental interaction of brain-like systems with environmental factors over time (Tryon, 1995b) and thereby provides a theoretical basis for understanding the difficult interactive question Hayes posed. This third major contribution of NNLT is illustrated below.

Behavior Genetics

Weissberg and Owen (1999) reminded us that behavior genetics identifies sources of variance across individuals and does not isolate biological from environmental causes of behavior. They emphasized the role of environmental factors by reminding us that genetic factors rarely account for more than 50% of behavioral variance. They reiterated that there is no gene for behavior. They correctly stated that genes regulate protein synthesis, which indirectly impacts behavior. They indicated that genetic does not mean fixed, in that both height and IQ are highly heritable but are increasing with time, faster than evolution can alter the gene pool. These comments may clarify the goals and objectives of behavior genetics but they do not address the interactive psychological and behavioral question posed by Hayes.

Weissberg and Owen's (1999) reply to Hayes (1998b) reflects the theoretical schism between psychology and biology. Weissberg and Owen defend against what they perceive to be an unfair characterization of behavior genetics. Proponents on opposite sides of theoretical schisms frequently feel misunderstood. Misunderstanding inevitably results when advocates of different theories, based on different assumptions and philosophical premises, using different methods of inquiry and analysis to address different questions, attempt to persuade each other on almost any point. Consequently, there

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Psychology and Biology: A Concluding Comment

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Weissberg and Owen sometimes seem to understand the point I was making, but moments later I am not so sure. For example, they say that the lack of identification of functional processes in twin studies is a "valid criticism," but they then add that this concern is addressed in mainstream research, saying, "For example, while phenotypic intelligence, as indexed by IQ scores, is highly heritable, there also is substantial evidence of its malleability." But that is simply not responsive. My point would stand with equal force regardless of the malleability of any trait, namely, that so called genetic effects can in theory be 100% due to psychosocial processes reliably engaged by structural biological differences, and the methods being used in human research are completely unable to detect that fact.

I think it is telling that Weissberg and Owen do not refute this core concern – nor have I ever had any behavior geneticist refute it. To me, this means the methods are inadequate and the resulting "answers" are inane. A true characterization of typical twin study results would be, "...we have found x amount of covariation between subject charachteristics and due to genetic differences, and oh, by the way, we do not know the actual processes that produced this covariation and whether or not these processes are biological or psychosocial." Excuse me? I should be excited by this research for exactly what reason?

Weissberg and Owen, and Tyron as well, seem to detect in this concern a certain lack of enthusiasm for the biological dimensions of behavior, but I am simply holding out for adequate methods focused on important questions. Here is the key question that lies at the interface of psychology and biology: "What specific biological features combine with what specific environmental characteristics (considered both historically and situationally) to produce what specific behavioral patterns, and how does this come about?" We already have methods that can begin to answer some aspects of this question in animal preparations. The Tang et al. (1999) study cited by Tryon provides an example. A gene with a known structural impact is injected into mouse ova. The mice that result preform better on standardized learning tasks, and some evidence of the exact neurological processes involved is obtained. This seems to me to be worthwhile research that begins to address the question above, but at present we have no such experimental methods available for humans. Twin studies (and adoption studies) are not even close, and it only creates confusion to pretend otherwise.

Connectionist models, such as those championed by John Donohoe or Warren Tryon, provide an interesting approach to the question of how psychological processes are reflected in structural changes in the organism, but we need to admit how far away these models are from saying much of anything to human psychological researchers, never mind behavior thera-

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can be no consensus regarding what the main conceptual and empirical questions are and no agreement regarding what empirical facts need to be collected. The predictable outcome is for behavior geneticists to continue to ask the same questions they have always asked using the same or similar procedures and for behavior therapists to continue to address different questions using different procedures in mutual isolation. Staats (1983) documented the corrosive consequences of such theoretical schisms.

Successful communication requires a certain degree of common theoretical ground; shared assumptions and/or elaborated theory. No amount of mutual criticism will substitute for the absence of a shared theoretical perspective. Nor do critical exchanges across a theoretical rift help develop the necessary common ground. A positive contribution is required. NNLT is a behavioral, cognitive, and biological theory and thereby provides the necessary interdisciplinary scope to effectively incorporate behavior genetics into a cognitive behavioral understanding of behavior disorder and its remediation via behavior therapy. NNLT provides the required common theoretical and empirical basis that will enable behavior geneticists and behavior therapists to work together.

Integration via NNLT

Architecture

The first way in which NNLT integrates biological, including genetic, factors into explanations of behavior and mediating psychological states is its use of brain-like, brain-inspired, structures to implement learning and memory. Genetics governs brain structure as it does other anatomical structures. NNLT simulates simple first approximations of real brain structures. A common neural architecture has three layers. The sensory or input layer frequently distributes representation of perceptual features of stimuli across all neurons in this layer using a binary code of on = 1, off = 0. A second layer of neurons is used to form concepts (latent constructs) based on sensory input in a manner that is similar to structural equation models (SEMs). This middle layer, also called the hidden layer, almost always contains fewer neurons than the input layer, just as SEMs contain fewer latent constructs than indicators. Because multiple neurons compose the middle layer, neural networks form a multifactor understanding of the microfeature information encoded by the sensory neurons. Unlike SEMs, where the investigator defines each latent variable by the indicators he or she associates with it, each input neuron is connected to every middle layer neuron. The network defines its own latent concepts by weighting the connection each middle layer neuron has with each sensory layer neuron. Connections given strong positive or negative weights are deemed important. Connections given small weights are minimized and those given zero weights are excluded. The third or output layer has one or more neurons that are each connected to every middle layer neuron. These are analogous to second order SEM factors. Again, the network forms its own latent constructs by differentially weighting input from each first order factor constructed by the middle layer to each second order output factor. A second difference between SEMs and CNNs is that SEMs are limited to linear functions whereas CNNs frequently employ nonlinear functions that also have a linear segment. These CNNs are closely related to multivariate discriminate analysis. CNN responses can therefore be understood as based on second order constructs (nonlinpists. The problem is this: connectionism unconstrained by actual biology is nothing more than a mathematical modeling technique. An infinite number of connectionist. models may produce functional systems with the same given properties. In the absence of actual detailed evidence about the organism's structure, we have no way to weed out various competing models except by taking on the burden of particular assumptions (e.g., that only networks organized within certain structural constraints, such as specific patterns of nodal connections, will be considered; or that the network with the simplest structure that adequately models the functions desired shall be declared the best solution). Any assumption can be incorrect (nature is not always parsimonious, for example, so the simplest solution may not be the actual one) so ultimately we have to examine actual physical structures (usually, the brain) to determine the accuracy of specific models. Connectionist models, in principle, can help the biologist or neuroscientist in that search but in the meantime they leave the psychologist with formulaic metaphors and little else. Brain researchers are far, far away from methods that would confirm or deny specific connectionist models in all but the simplest organisms.

But suppose we completely solved that problem and knew exactly how the organism was organized. Would we then resolve the cognitive behavioral debate, as Tryone claims? Would psychological theories be mere way stations for biological ones?

The answers are "no." Psychology is a separate level of analysis, examining how whole, individual organisms interact in and with a context, considered both historically and situationally. This level of analysis is informed by biology, but can never be supplanted by it. No amount of information on the structural properties of the organism (which is entirely the focus of NNLT and all similar models) will place the context inside the organism, unless one makes mechanical assumptions that contextual features are isomorphically reflected in the structure of the organism. But such assumptions do not resolve the cognitive behavioral debate – they *are* the cognitive behavioral debate (or at least, the part that is most difficult to resolve). The debate is not about whether behavioral principles can deal with cognitive behavior — that is merely an empirical question (one admittedly made far more interesting by the emergence of a behavioral psychology of cognition). The intractable core of the cognitive behavioral debate is the conflict between mechanism and contextualism (Hayes, Hayes, & Reese, 1988). In Tyron's hands, NNLT is a mechanistic theory, particularly because he seems to believe that information about neural processes, in principle, somehow supplants or supercedes a more complex unit of analysis. As such, this theory will appeal far more to most cognitivists than to most contextualistically oriented behaviorists.

Connectionism has been around for a while and though it can be good intellectual fun, I see few pragmatic reasons for behavior therapists to attend to it. I can think of no new interventions or assessment methods in behavior therapy that connectionism has produced. But then, that is the kind of deflatingly pragmatic thing a contextualist would say.

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ear multivariate discriminant functions) created by the three layers of neurons and two layers of synapses that derive from genetic factors in real biological systems.

The above discussion concerns structural issues associated with neural architecture. This discussion is warranted because genetics clearly governs brain structure. Individual differences in neural architecture can influence functional network properties and therefore modify psychology (middle representational layer) and behavior (output layer). Empirical support for this assertion is provided by lesion studies where CNNs are intentionally damaged resulting in systematic functional impairment (cf. McLeod, et al., 1998). However, structural considerations are only a partial and necessarily incomplete consideration of behavior genetics from the NNLT perspective. We now turn to functional issues.

First functional example

Hebb (1949) postulated that learning entails synaptic modification; synapses connecting simultaneously active neurons are strengthened. Neuroscience has since confirmed this view (e.g., Kandel 1989, 1991). Reinforcers modify behavior because they alter synaptic properties, which answers the age-old question why reinforcers are reinforcing. NNLT explains cognition and behavior in terms of environmentally induced changes in synaptic excitation and inhibition (cf. Elman et al., 1996; McLeod, Plunkett, & Rolls, 1998; Rolls & Treves, 1998). Genetic effects on cognition and behavior are similarly understood in terms of their impact on synaptic excitation and inhibition. The following long-term memory formation example is especially instructive because it pertains to all long-term cumulative learning and because it demonstrates how environmental events trigger genetic expression through a gene cascade, that synthesizes new proteins.

Genes have a coding region and a regulatory region. RNA is transcribed from template DNA in the coding region. Transcription is the process whereby DNA genetic sequences are used to construct RNA molecules. Translation entails protein synthesis; it moves from the language of nucleotides to the language of amino acids (Curtis & Barnes, 1989, p. 312). Repeated transmitter stimulation at the synapse causes a substance (cAMP) to phosphorylate (add a phosphate group and thereby energize) one or more transcription proteins that bind to regulatory regions of genes thereby stimulating transcription and the synthesis of new proteins responsible for long-term memory formation. Compounds that inhibit transcription and translation also inhibit phosphorylation and restrict memory formation. Thus, genetic expression mediates long-term memory formation. All efforts by behavior therapists to restructure cognitions and teach new behaviors requires the formation of new long-term memories and therefore entails the above mentioned gene cascade. This mediational role enables genes to have various effects on psychology and behavior that are relevant to understanding psychopathology and the effects of behavior therapies.

Second functional example. Hebb's rule indicates that learning takes place when two neurons are active as noted above. The N-methyl-D-aspartate (NMDA) receptor detects such events because it allows calcium ions to flow only when it simultaneously receives input from two neurons. A temporal window exists within which the two signals must be received in order to open the calcium ion channel. The longer the duration of this window the greater the probability of detecting associated events. NMDA receptors are constructed from NR2A and

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Dattilio, F. M. (1998). (Ed.), *Case Studies in Couple and Family Therapy: Systemic and Cognitive Perspectives.* New York: Guilford Press.

Reviewed by: Richard E. Watts, *Kent State University*

Examining the table of contents of *Case Studies in Couple and Family Therapy* is analogous to looking at the program of an all-star game. The editor, Frank Dattilio, assembled an impressive collection of couple-, and family therapy heavy-hitters to author chapters and to dialogue with him regarding the integration of cognitive and systems-oriented approaches in behavior therapy. Chapters are sandwiched between an opening that introduces cognitive behavioral therapy with couples and families and the epilogue, both written by Dattilo.

The chapters clearly present each author's theoretical position in the excellent case studies and accompanying commentary. The case studies and commentary alone make the book a valuable resource. The format of the book is unique in that it contains a continuing dialogue between the authors and the editor. Interspersed throughout the case studies, Dattilio identifies similarities between cognitive behavioral therapy and the various systemic perspectives. Additionally, he offers recommendations for how specific cognitive techniques and interventions might be employed at various points in the case studies. Each chapter concludes with a response by the author(s) to Dattilio's previous comments. In the final chapter, Dattilio briefly replies to all the authors' responses and provides some conclusions regarding the integrative focus of the book.

According to Dattilio, the fundamental goal of *Case Studies in Couple and Family Therapy* is to create a dialogue to assist readers in drawing their own conclusions regarding the integration of cognitive behavioral strategies with the systems-oriented approaches presented in the book.

In the dialogue between the authors and the editor, one gets the feeling of being privy to a frank discussion between very strong personalities, all of whom are well informed and able to justify and critique their own positions. Many authors find considerable common ground between their approach and cognitive-behavioral therapy (e.g., James Keim), whereas others (e.g., Susan Johnson), appear less interested in discovering points for integration. Nevertheless, I was impressed by the tenor of the dialogue between the authors and the editor. Whether in agreement or not, the author/editor discussions are candid, respectful, and cooperative. The authors and editor provide good examples of how to Aðdisagree agreeably.

Regarding the potential limitations of the book, the structure of various case studies may fall short; they provide excellent information but are inconsistent in their presentation. Some focus on early sessions of the case, whereas others address the case from initial assessment, to termination, and beyond. My personal preference would be for the case studies to be presented in a format that makes it easier for readers to compare and contrast the therapeutic process of each approach and the editor's comments for the same. A second reservation has to do with tracing the lineage of cognitive and systemic integration. Frankly, I was surprised that there was not even cursory mention of the first theory to integrate cognitive and systemic perspectives: Adlerian therapy.

The few limitations do not, however, change my strongly favorable opinion about this book. Practitioners, therapy educators, and students who are open to integrative dialogues will find this text useful. It clearly delineates areas of similarity and difference between cognitive and systemic perspectives in a unique and interesting format. This text may help readers increase their conceptual flexibility via the discussions of how similar clinical phenomena and interventions are framed from differing theoretical perspectives. In addition, the book demonstrates the facility and utility of integrating various ideas, interventions, and strategies from other approaches without abandoning one's primary theory of choice; i.e., being theoretically consistent and technically eclectic. *Case Studies in Couple and Family Therapy* is an excellent resource. Dattilio and his contributors are to be commend.

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ments about the role of biology based on the over-generalization of terms and findings and their enthusiasm for the long term success of their enterprise.

Far from "trashing the entire enterprise" in my column, I stated clearly that it is important to know "what specific environmental events combine with what specific genetic or organismic events to produce what specific behavioral patterns" (p. 96). Some behavioral geneticists who work with animal models are asking and answering forms of this question right now, and I explicitly held out hope in my column that the human genome project might eventually set up the proper question in the human area. But most of the methods available to behavioral geneticists in this area give inherently misleading answers to inherently unimportant or even wrong-headed questions. Rather than risk almost certain confusion, despite the terrible consequences that confusion can create, I wish more behavioral geneticists would either develop the needed methods or work in the more limited areas of their discipline where methods are reasonably adequate. The alternative is to write reports from la la land, and we have enough of those already.

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NR2B subunits. NR2B subunits have a longer time window and predominate in younger organisms while NR2A units have a shorter time window and predominate in older organisms. Staying open longer allows more calcium ions to pass, which makes the NR2B response more vigorous than the NR2A response. Both properties are consistent with better learning in younger than older organisms. The construction of the NMDA subunits and their differential construction rate over the life span is a genetically controlled aging process. Tang et al. (1999) created transgenic mice by linking a copy of the mouse NR2B gene to a promoter that is only active in the forebrain (hippocampus and amygdala) and injecting it into fertilized mouse ova. These additional genes, along with the mouse's

At AABT

Behavior Therapy Introduces

A New Feature

J. Gayle Beck, Editor

B*ehavior Therapy* may look like that familiar green journal that arrives in your mail four times yearly, but there's considerably more than meets the eye. For the past 6 months, the journal has been trying out a new feature...

Based on an idea provided by member Ann Steffen at last year's "Meet the Editors" session at the Annual Convention, we are experimenting with a Student Editorial Board (SEB). The SEB is an effort to actively involve student members in the peer-review process, with the hope that we can familiarize young researchers with the process. This feature will have many radiating effects:

Behavior Therapy will benefit from student input, we'll cultivate a pool of potential reviewers early in their careers; and the journal will become more familiar and attractive as a potential publication outlet for student authors.

SEB members will gain direct reviewing experience under the mentorship of one of the Editorial Board members; they'll learn how to shape potential manuscripts towards publication-quality products, and they'll contribute in an important way to the scientific mission of AABT.

How is the SEB organized, you may ask.

Each Editorial Board member is encouraged to include students in the review process. Upon receipt of a manuscript from the Editor or Associate Editor, the Editorial Board member serves as a guide and a mentor, working with the student to review the paper. Once a student has helped with a review, he or she is automatically an SEB member. These individuals will be acknowledged in the final issue of each volume and will have an impressive entry to add to their vitaes.

We will be test driving this feature for another 6 months before deciding whether to institutionalize the SEB into *Behavior Therapy*.

In the meantime, I'd love to hear your feedback about this new facet of the journal. Write: **J. Gayle Beck, Ph.D., SUNY at Buffalo, Department of Psychology, Park Hall, Buffalo, NY 14260** or Email me at: **jgbeck@acsu.buffalo.edu**

own NR2B-producing gene, caused an over expression of NR2B subunits in the forebrain of the resulting mice. These experimental subjects excelled on six behavioral tests of learning compared to control subjects: a novel-object recognition task, a retention test, contextual and cued fear conditioning tasks, a fear-extinction task, and a hidden-platform water maze. Additional evidence was presented showing enhanced NMDA receptor mediated current and consequently enhanced long-term potentiation (LTP), which is strongly implicated in the formation of long-term memories, and strengthens the cause and effect conclusions that can be drawn from this study. Learning and memory are fundamentally related in that cumulative learning presumes memory. The genetic enhancement described here improved both learning and memory. Genetic alteration was the independent variable with learning and memory measures as dependent variables. Such experimental evidence of the causal influence of genetics on behavior directly addresses the interactive issue posed by Hayes and completely avoids the problem of biologism.

Concluding Comments

The above examples might seem to be more about biology than behavior and psychology in that they emphasize genetic effects on synaptic properties. NNLT bridges the explanatory gap to psychology and behavior by explaining how psychological and behavioral phenomena emerge from connectionist models of simple neural systems where synaptic properties, degrees of excitation and inhibition, play a central explanatory role (cf. Elman et al., 1996; McLeod, et al., 1998; Rolls & Treves, 1998). Hence, NNLT provides the missing theoretically link from synapse to psychology and behavior. The examples presented above begin to provide answers to the interactive questions Hayes requests that we ask. The pitfall of biologism is avoided by describing a sequence of causal events that begins with environmental events, is mediated genetically in the first functional example, and begins with genetic enhancement that facilitates learning and memory in the second functional example. The neural architecture example illustrates a different causal sequence. These examples show how NNLT allows us to replace contemporary black-box models and arrow diagrams with computational models of brain-behavior relationships.

The interactions between biology and experience that generate psychology and behavior are sufficiently complex that computer simulation is required to generate specific empirically testable predictions (cf. McLeod, et al., 1998; Plunkett & Elman, 1997). Software to conduct these simulations is increasingly available. Tryon (1995b) published a list of vendors. Statistical Package for the Social Sciences (SPSS) has recently introduced CNN software. McLeod, et al. (1998) provide a computer program called tlearn on a disk as part of their book. CNN models typically begin with small random synaptic weights that produce ineffective behavior. Learning through experience modifies connection, synaptic, weights towards optimal settings. Functionality increases as a result of these weight changes. This process bears meaningful resemblance to an investigator who collects data one subject at a time and recalculates a regression equation after each subject has been tested. More subjects enables better prediction. Neural networks exhibit more effective behavior given greater experience.

The ability to construct models of how nature and nurture interact to produce perception, cognition, and behavior should enable us to drop one-sided arguments defending the importance of either environment or genetics by having models that explicitly describe how these processes interact. It should also enable us to move beyond calculating controversial percentages due to genetic and environmental influences to a description of interactive process. It should also help us

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avoid biologism by prompting us to ask questions about the biological mechanisms implied by reports of how the inner ears of lesbians differ from those of heterosexual women or that smokers differ in a nucleotide sequence from nonsmokers. We will now want to know how these differences explain changes in synaptic properties that regulate psychology and behavior. Or ask if these differences are markers for other biological events that impact synaptic function.

The benefits of the theoretical integration described above extend beyond academic scientists to clinicians who work in medical settings, to clinicians who consult with physicians, and to clinicians who are interested in a thorough understanding of the clients they serve. Kuhn (1970) concluded that a new theoretical system is adopted because it provides new ways to think and talk about formerly intractable scientific problems rather than because it currently answers all questions and/or is conclusively supported by definitive empirical proof of superiority. NNLT provides exciting new ways to discuss behavior genetics in addition to cognitive behavioral phenomena and should therefore be evaluated on the basis of how it integrates cognitive, behavioral, and neurobiological data. Criticism directed at what NNLT has yet to accomplish is inappropriate at this time because it fails to understand that the only way to know what can be done is to work at it and that, in the beginning, must largely be motivated by future promise.

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