PAUL E. MEEHL AND B. F. SKINNER: AUTITAXIA, AUTITYPY, AND AUTISM

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ABSTRACT: Paul E. Meehl and B. F. Skinner, two of the foremost psychological theorists of the 20th century, overlapped at the University of Minnesota in the early 1940s when Skinner was a faculty member and Meehl was a graduate student. Though Skinner was well aware of, and influenced by, early 20th century physiology, he eschewed reductionism, developing his analysis of behavior without reference to concepts at another level of analysis. Meehl’s theoretical approach transcended levels of analysis, drawing upon data and concepts from genetics, neuroscience, and psychology. In this paper the functional components of Meehl’s (1990) “Toward an Integrated Theory of Schizotaxia, Schizotypy, and Schizophrenia” paper are re-formulated substituting autism as the condition of interest. Skinner’s and Meehl’s theoretical frameworks are integrated with recent findings in genetics and neuroscience in an attempt to better understand the reasons why Intensive Early Behavior Therapy (IEBT) provided to children with Autism Spectrum Disorders produces enduring improvements in social, language, and cognitive functioning.

AUTHOR’S NOTE: I will remember Paul Meehl (1920-2003) for his intellectual guidance, professional support, and wise counsel over my graduate school and faculty years and the personal contributions he made to my life. This paper is dedicated to his memory. Burrhus F. Skinner (1904-1990) will be remembered for many reasons. History may show that one of his most enduring contributions was providing the theoretical foundation that made it possible for O. Ivar Lovaas to transform his principles into effective treatments for young children with autism. Not many can say that their work has profoundly improved the lives of many thousands of children who would otherwise have faced dire futures. I'm most grateful to my colleague Dr. Leslie Yonce, who shared her personal life as Paul Meehl’s wife and professional life as his intellectual co-worker, for kindly reading the manuscript for accuracy and readability. She applied her considerable editorial skills in clarifying the author’s intent and making the paper more understandable. My thanks to Dr. Ford Ebner, a distinguished neuroscientist colleague in the Department of Psychology at Vanderbilt University, for suggesting alternative mechanisms by which neuroplasticity could be impaired in autism that could be responsive to activity-dependent treatments. Dr. Gail Peterson and Dr. Armando Machado kindly invited me to collect my thoughts regarding the contributions of Paul E. Meehl and B. F. Skinner and to add a few ideas of my own. I’m grateful to them for providing this forum for doing so. Please address all correspondence to Travis Thompson, Ph.D., Center for Neurobehavioral Development, 420 Delaware Street S.E., MMC 507, University of Minnesota Medical Center, Minneapolis, MN 55455; Email: tithompson@comcast.net
B. F. Skinner at Minnesota

Paul E. Meehl, one of psychology’s most influential theorists during the latter half of the 20th century, and B. F. Skinner, founder of the field of behavior analysis, rubbed intellectual elbows at the University of Minnesota in the early 1940s. In 1936 Skinner had joined the faculty at the University of Minnesota, having recently moved from Harvard University where he had been influenced by the distinguished physiologist L. J. Henderson, president of Harvard’s Society of Fellows (Skinner, 1985). As a graduate student, in addition to studying psychology Skinner had been exposed to early 20th century physiology emerging from Claude Bernard’s *Experimental Medicine*, including Charles Sherrington’s *Reflex Arc*. Sherrington (1906) had proposed the reflex arc as the unit of analysis in nervous system function. Skinner aspired to identify a comparable fundamental unit of analysis in psychology. In his dissertation, which was reproduced in edited form in *The Behavior of Organisms* (*BoFo*), Skinner argued that the reflex (without the hypothetical baggage of the reflex arc) was such a unit. He later dropped the term reflex, substituting respondent and operant behavior, which he had originally described as two types of reflexes. His fellow graduate students, Fred Keller and Willard Quine, appeared to have an impact on Skinner, though Keller later wrote that it would be some time before he understood the importance of Skinner’s ideas. Skinner and the philosopher Willard Quine were both selected as junior members of the Society of Fellows the same year (1933) and shared an interest in language and verbal behavior, a second of Skinner’s interests that matured with the publication of *Verbal Behavior* in 1957.

In Skinner’s early years at the University of Minnesota he was surrounded by an extraordinary group of graduate students: Kenneth MacCorquodale1, Frank Barron, William K. Estes, George Collier, Keller and Marian Breland, Norman Guttman, Howard F. Hunt, and Paul E. Meehl. Paul Meehl entered graduate school in clinical psychology at the University of Minnesota in 1941 and completed his training the year Skinner moved to Indiana (1945). In his autobiography Skinner

1 Kenneth MacCorquodale and Paul Meehl shaped much of my intellectual life as a psychologist. As was the custom when I was in graduate school, students were expected to enroll in courses taught by the entire senior faculty in the course of their training, so I had the good fortune of studying with both men. MacCorquodale, who was my undergraduate advisor and informal mentor during graduate school, was the cynosure of incisive, analytic clarity and rigor. I jointly taught a seminar on behavior analytic theory with him for many years prior to his retirement, which was a great pleasure (Thompson, 1987). One came away from a discussion with him with a clearer understanding of what it means “to know.” MacCorquodale and Meehl were life-long colleagues and friends who held one another in high esteem, though in many respects their approaches to psychological theory could not have been more different. Meehl masterfully promulgated his unique approach to synthetic reasoning throughout his voluminous theoretical contributions. He had the ability to find order in psychological phenomena that appeared chaotic, to disentangle threads of thought and evidence that enabled him to deconstruct, then re-weave a novel theoretical fabric, revealing important relationships that had eluded nearly everyone.
MEEHL AND SKINNER: AUTITAXIA, AUTITYPY, AND AUTISM

(1985) remarked, “...I stole Estes from engineering and Norman Guttman from philosophy. My teaching has never again been so richly reinforced.” A good deal has been written about Skinner’s and Quine’s mutual influence upon one another. John Malone has written, “However, according to Paul Meehl (personal communication, August 6, 2001), Quine had absolutely no influence on Skinner. In Meehl’s view, no one influenced Skinner, though Skinner influenced others” (Malone, 2001). Skinner certainly had an impact on Paul Meehl, Kenneth MacCorquodale, and the other bright young psychologists receiving training at the University of Minnesota in the early 1940s.

As a graduate student at the University of Minnesota and over subsequent years on the faculty, I frequently discussed Skinner’s writings with Paul Meehl. He regarded Skinner’s theoretical analysis of behavior as a major contribution to psychology that he incorporated into his own thinking. Meehl’s influential article with MacCorquodale (1948), “On a Distinction between Hypothetical Constructs and Intervening Variables,” provided grist for numerous discussions of Skinner’s views. Meehl considered Skinner’s adherence to pure intervening variables as misguided, arguing that in practice such constructs were rare except in certain fields of physics. Moreover, in Meehl’s view some putative explanatory variables that are not securely anchored to the observation level (what philosophers call “open concepts”) could at times provide important hypotheses to be empirically tested.

Perhaps Meehl’s greatest departure from Skinner was over the latter’s handling of behavioral variability (within and across individuals) and Skinner’s limited consideration of the organization of complex heterogeneous response classes and their stability over time, what personality theorists call traits (see discussion in Lubinski & Thompson, 1986). In BofO Skinner implied that behavioral variability amounted to an experimental nuisance to be dealt with by identifying the controlling environmental variables that could eliminate variability. Emotion and motivation were among the sources of variability that Skinner argued could be controlled with appropriate manipulation of variables such as hours of deprivation or changes in reinforcement history.

Meehl’s theoretical appetite was as encompassing as Skinner’s was focused. Meehl’s intellectual peregrinations at times seemed contradictory. In his autobiography Meehl (1989) reported that as a clinical psychology trainee he was fascinated by neurophysiology and found going on rounds with the Minnesota neurologist A. B. Baker intellectually captivating. For a time he delved into psychoanalysis, and with MacCorquodale he wrote an influential interpretation of Tolman’s expectancy theory (MacCorquodale & Meehl, 1954). He developed the K scale for the MMPI, a measure of subtle defensiveness. He wrote on psychology and politics, philosophy of science, and behavior genetics, but perhaps his book, Clinical versus Statistical Prediction (1954) had the broadest lasting impact. In that book he presented a compelling argument (and evidence) that actuarial combinations of data are as good or better than clinical judgments. The book was pure atheoretical Dust-Bowl Empiricism. Epistemology and its application to the
philosophy of science was one of Paul Meehl’s enduring intellectual passions, which was interwoven throughout his theoretical writing.

**Skinner, Meehl, and Reductionism**

In my 50th anniversary appreciation of *BofO* (Thompson, 1988) I expressed perplexity over Skinner’s rejection of his roots in physiology, which were the methodological foundations upon which much of the field of behavior analysis was constructed (Thompson, 1984). Claude Bernard and his followers had introduced the reversal experimental design (ABA) for demonstrating the effects of physiological variables within individual subjects. Bernard’s goal was to understand—by which he meant to be able to predict and control—factors affecting physiological functioning of individual people, not the average person. Bernard renounced hypothetical constructs that purported to explain empirical phenomena, processes that he claimed were better accounted for by testing effects of observable independent variables on measurable physiological outcomes (Bernard, 1865 [trans. 1927]).

Perhaps Skinner abandoned physiology in part because of his antipathy for slapdash reasoning that masqueraded as serious psychological theory at the time. When Skinner wrote his most influential early work, hypothetical constructs ran amok within psychology, including unabashedly speculative notions about brain structure and functions and the role of genetics in behavior. Little of it was based on empirical evidence. But at times Skinner’s aversion to what he called *explanatory fictions* failed to distinguish between conjecture regarding hypotheses that were in principle not falsifiable and other inferences that were not currently testable due to lack of technology. At times Skinner distinguished among these types of hypotheses, but he concluded that theories involving reference to anatomy, physiology, or biochemistry in relation to behavior would add little to the field of behavioral analysis, even if verified. Skinner was so strongly committed to a version of psychology that could stand on its own feet without reference to terms or concepts at any other level of analysis, that he dismissed the possibility that the explanatory reach of his science could be enhanced by incorporating empirical evidence and concepts from related disciplines. Skinner’s metatheoretical position was antithetical to Paul Meehl’s perspective, which freely drew upon empirically observable phenomena across levels of analysis in arriving at an understanding of psychological and behavioral phenomena.

The historian of science, Ernst Mayr (1982) pointed out that one can subscribe to *constitutive reductionism* (sometimes referred to as ontological reductionism) without adopting *explanatory* or *theoretical reductionism*. He contended that all biologists subscribed to constitutive reductionism. Mayr wrote that explanatory reductionism “claims that one cannot understand a whole until one has dissected it into its components, and again these components into theirs, down to the lowest hierarchical level of integration. In biological phenomena it would mean reducing the study of all phenomena to the molecular level (i.e., “Molecular biology is all of biology”). Constitutive Reductionism “posits that none of the events and processes
encountered in the world of living organisms is in any conflict with the physico-
chemical phenomena at the level of atoms and molecules. . . . The difference
between inorganic matter and living organisms does not consist in the substance of
which they are composed but in the organization of biological systems” (Mayr,
1982, pp. 60-62) [this author’s underlining]. On the other hand, theoretical
“reductionism postulates that the theories and laws formulated in one field of
science (usually a more complex field or one higher in the hierarchy) can be shown
to be special cases of theories and laws formulated in some other branch of
science. If this is done successfully, one branch of science has been ‘reduced’ to
the other one. . .” (Mayr, 1982, pp. 60-62).

Skinner was surely right that describing molecular events within a given
neuron will not help us understand such phenomena as a person’s aspirations or
what cultural identity means. However, Skinner wrote little about several topics of
central importance in psychology. He neglected the multiplicity of sources of
behavioral variability within and across individuals (some of which have genetic
and neurobiological origins), and he did not advance adequate strategies for
dealing with unpredictable, emergent phenomena. Mayr (1982) considered the
latter to be among the more distinguishing features of scientific explanation in
biology, a topic that Meehl and his philosopher colleague Wilfred Sellars (1956)
had explored in relation to psychology under the auspices of the Minnesota Center
for the Study of the Philosophy of Science.

Schizophrenia: A Case in Point

Meehl contended that understanding the types and sources of behavioral
variability (Meehl, 1992) and taxonomy (Meehl, 2004) were at the heart of the task
before psychology. In his paper “Toward an Integrated Theory of Schizotaxia,
Schizotypy, and Schizophrenia,” published in 1990, Meehl developed his most
extended analysis of the relation among genes, brain function, and
psychopathology (Figure 1). He had introduced the basic idea behind this paper
many years earlier (Meehl, 1962), but it was not until 1990 that he presented his
theory in its elaborated form. He argued that in schizophrenia a gene defect
produces generalized dysfunction throughout the brain (hypokrisia), which in turn
leads to more specific cellular dysfunction (schizotaxia), which when acted upon
by epigenetic factors (social and/or physiological) produced degrees of the
schizotypy. Meehl suggested it is likely that a mutation or other gene defect causes
a widespread neurochemical deficiency producing physiological consequences
throughout the brain (e.g., an integrative failure at the sub-cellular level). He called
this underlying neurochemical mechanism hypokrisia, from the Greek words
meaning an insufficiency of separation, differentiation, or discrimination. He
hypothesized that hypokrisia produces schizotaxia, a genetically determined
physiological integrative defect causing the person to be socially avoidant and
misperceive social relationships. This, in turn, predisposed the individual to
schizophrenia; however, only a minority of persons with the CNS defect
decompensated to the point of being diagnosable by DSM criteria. Meehl
suggested that the degree to which schizotaxia manifests itself as a schizotype depends on polygenic potentiators such as personality traits and social learning history as well as unpredictable life events like traumatic experiences. He hypothesized that only a small percentage of people with the genetic predisposition (schizotype) exhibit the extreme form of regressed psychotic schizophrenia (presumably due to the confluence of polygenic traits, learning history, and unpredictable events). Meehl also suggested that a combination of polygenic potentiators alone were capable of producing a combination of some of the features resembling true schizophrenia but lacking the core underlying genetic schizotaxia. He referred to these latter individuals as displaying genophenocopies.

Figure 1. Meehl’s (1990) conjectured causal pathways determining schizotaxic schizophrenia and non-schizotaxic genophenocopies.
Autism: A Functional Companion to Meehl’s Theory of Schizophrenia

The theoretical elements of Meehl’s theory of schizophrenia apply equally to other complex disorders including autism. Autism Spectrum Disorders (ASDs) provide a model similar to schizophrenia for beginning to understand the relations among genetics, brain structure and function, and behavior, and how they relate to behavior analysis. ASDs are the only severe behavioral disorder in which many of the signs and symptoms can be permanently ameliorated by psychological intervention early in life. Empirical evidence suggests that the most effective treatment methods emerged from principles of applied behavior analysis, an outgrowth of Skinner’s experimental analysis of behavior (Lord & McGee, 2001). Because of this unique relationship between signs and symptoms of autism and their lasting amelioration by intensive early behavior therapy (IEBT), questions arise about the nature of autism and why behavioral treatment produces lasting effects. It will be suggested here that lasting effects of behavioral treatment of young children with ASDs are closely tied to underlying neural mechanisms, and that ASDs are a special case of the theoretical argument that Meehl (1990) presented for schizophrenia (Figure 2).

Genetic Defects

It is proposed that underlying gene defects produce widespread effects on the developing brain via one or more common mechanisms. As we will discuss shortly, the genetic defect would be expected to be manifest selectively in some brain structures more than others (as revealed by structure-specific gene expression studies) and lead to autitaxia, the neurobiological mechanism leading to autism susceptibility.

Selective Hyposynapsia

The proposed genetic defect or defects are likely to be expressed in processes involved in synapse formation and neuroplasticity. It is a defect whose consequences are partly reversible through intensive behavioral intervention between 2 and 5 years of age. The underlying defect that leads to dysfunctional synaptogenesis could involve cytoarchitectural or neurochemical errors (see “Autitypy” section on p. 109). It seems unlikely that other mechanisms such as neuronal migration or axonal guidance would be susceptible to amelioration by experience-dependent intervention, though they cannot be ruled out a priori. The synaptogenesis defect is likely to be expressed differentially in some structures but not others, though there may be some other neurodevelopmental abnormalities that follow no consistent pattern.
Figure 2. Hypothesized causal pathways determining autotaxic autism and non-autotaxic autism phenocopies. Note potentiators include other genetic disorders, developmental disabilities, possible neurotoxin and autoimmune factors as well as social learning variables.
**Autitaxia-Dysfunctional Synaptic Processing**

Synaptic processing of information is the basis for cognitive functioning, learning, memory, and understanding and expressing emotions, all of which are problematic in autism. Various steps in synapse formation and functioning could be defective in autism. Synapse formation involves integrating transport, assembly, and regulation of protein components produced by specific genes. Presynaptically, several factors play a role in setting the stage for synaptic function. Among them, the first are the transport mechanisms permitting synaptic components to be delivered to the synaptic region. Once these components reach the appropriate site, processes required to assemble synaptic components could be defective (Cai, Su, Gerwin, et. al., 2004).

Neuronal information processing involves transduction of chemical signals into short- or long-term changes in the cell’s membrane potential. A chemical neurotransmitter is released by presynaptic cells which bind to receptors on the postsynaptic cell. Receptors are either *ionotropic* (ion-channel) receptors or *metabotropic* receptors. Ionotropic receptors directly and rapidly regulate membrane potential and activity of ion channels that generate nerve impulses. Ionic receptor effects are rapid and persist for only milliseconds; however, *activating a metabotropic receptor produces effects that may persist indefinitely.* The capacity for synapses to change in neurotransmission efficiency, and formation of new synapses, are two mechanisms by which long-term changes in synaptic transmission persist in the brain. These processes appear to be responsible for permanent changes in brain structure during learning. The neuroplasticity defect in specific brain structures (autitaxia) is functionally equivalent to what Meehl called *schizotaxia*, but in this case the predisposition to autism rather than predisposition to schizophrenia.

**Autitypy**

It is proposed that the degree to which *autitaxia* (the proposed neuroplasticity defect) manifests itself as degrees of ASD (e.g., Pervasive Disorder Not Otherwise Specified, Asperger’s Disorder, High Functioning Autism, or severe Autistic Syndrome) depends on environmental and polygenic potentiators (epigenetic factors) interacting with the genetic predisposition to produce degrees of *autitypy* (the counterpart of schizotypy). As with other genetic syndromes (e.g., Prader Willi, Trisomy 21), combinations of genes may interact to produce wide variation in the clinical phenotype. Interaction of the gene defect with other epigenetic factors such as neurotoxins, autoimmune abnormalities, or consequences of other concurrent neurogenetic syndromes introduces great phenotypic variability. This variability makes identifying autism subtypes difficult. A more effective strategy calls for identifying autism *endophenotypes*, people displaying clusters of physical, behavioral, or neurocognitive features that can be linked to subsets of autism-related genes.
Finally, some polygenic and environmental potentiators are capable of producing components of the Autistic Syndrome by themselves, but not the full form that typically meets all of the DSM or International Statistical Classification of Diseases and Related Health Problems (commonly known by the abbreviation ICD) criteria for Autistic Disorder. Such potentiators might include damage to some of the relevant brain structures by other neurodevelopmental disorders (e.g., Fragile X syndrome, Prader Willi syndrome), other genetic mental health or developmental conditions overlapping with ASDs (e.g., Obsessive Compulsive Disorder [OCD] or language disabilities), or exposure to infections or neurotoxins (e.g., Fetal Alcohol Syndrome or Effects) during gestation, and would invariably include vast differences in social learning history. Children born to parents who are themselves inflexible, socially aloof, affectively constricted, and provide poor language models may be especially prone to developing autism-like symptoms if they also inherit genes for OCD, language disability, or ADHD. Some individuals with heavy dosages of such social potentiators may be erroneously diagnosed with “high functioning autism” or “Pervasive Developmental Disorders—Not Otherwise Specified” in clinical settings and even within some autism study samples. Having misdiagnosed them, well-meaning clinicians may refer such children for autism-specific interventions such as IEBT, which at times may be inappropriate. Other children with less specific brain insults may suffer damage to some of the same structures that are implicated in autism, and they may display autism features sufficient to lead to a DSM diagnosis of ASD. They will tend to have microcephaly, dysmorphic features, low IQ, and be less responsive to most treatments, including intensive early intervention (see Miles, Takahashi, Bagby, et. al., 2005). In the following sections, evidence and the reasoning behind this proposal are examined.

Autism Background

Autism was first described in detail by the French physician Jean Itard in 1801. A naked boy estimated to be around 12 years of age had been brought to him after he was found running naked near farmhouses in Aveyron, France where he had been seen scavenging food. The boy, who he named Victor, was non-verbal, did not respond appropriately to people, and had fixed routines such as rocking and hand-flapping. When his preferred routines were interrupted he had tantrums and at times became aggressive. Victor made guttural vocalizations and howled, making sounds resembling wolves in the forest, where he had apparently lived. Itard took Victor into his home and attempted to cure him. Itard taught Victor to communicate using pieces of wood cut into shapes that represented things he might want, such as milk or going outside into the courtyard to play. When Victor handed Itard or his housekeeper the appropriate icon he was rewarded with access to what he wanted. Victor’s outbursts diminished as he learned to use icons for communication. Victor learned most self-care and daily living skills, and although
his behavior problems improved he continued to exhibit bizarre behavior. Eventually Itard concluded he could not cure Victor and transferred him to a school for deaf children.

When Leo Kanner first described autism as a clinical syndrome in the medical literature in 1943 he focused on three behaviorally defined features: (1) lack of social awareness, (2) lack of understanding of social communication (language and verbal behavior), and (3) repetitive routines and interests. He noted the onset was early (usually before 3 years of age), and he speculated that it was a congenital brain disorder. He noted that parents of children with disabilities often had some of the same behavioral characteristics as their children, such as social anxiety and aloofness and inflexibility and insistence on sameness. Around the same time, Hans Asperger (1944) described a similar, but less severe, neurodevelopmental disorder that he had observed in his clinic in Austria. The children were similar to those described by Kanner, though they appeared to develop language at about the same age as their peers. However, often the language sounded stilted, was very literal, and was socially naïve. For the next 20 years autism and Asperger’s Syndrome (AS) were considered to be untreatable. Aside from Bettelheim’s (1967) failed attempt to treat children with autism employing psychoanalytic concepts, children diagnosed with autism generally attended segregated special education classrooms and were often placed in institutional settings as adults. Approximately 80% of these children were said to test in the moderate to profound range of intellectual disability. Most professionals assumed there was no effective treatment for autism.

Though Ivar Lovaas at UCLA had conducted limited studies using operant techniques with children with autism as early as 1964, he first described significant positive outcomes of behavior therapy with several 7- to 9-year-old children with autism in 1966. His findings were viewed with skepticism among experts in the field. Several times per week Lovaas used operant reinforcement and stimulus control techniques that were limited to a clinical setting. The targets of intervention were also limited (e.g., task compliance, speaking single words and phrases). Though there were improvements among most children treated, the outcomes lacked practically significant consequences. The children still displayed extreme rigidity, tantrums, occasionally self-injured, engaged in rocking and hand-flapping, were minimally verbal, and lacked social appropriateness. Lovaas concluded that his efforts had been too limited and that he had begun too late in the children’s development. The UCLA Young Autism Project that emerged dramatically changed our understanding of what is possible with behavioral treatment of children with autism.

Lovaas’s widely cited 1987 outcome study of the results of intensive, individual, home-based early behavior therapy as compared with much less intensive center-based therapy was controversial. He reported that when the children entered elementary school, roughly half of the children who had received intensive home-based behavior therapy were mainstreamed in elementary school with IQs testing in the typical range. None of the children in the less intensive center-based therapy program achieved these performance levels after three years.
of treatment. Critics found many reasons to question Lovaas’s findings, but none of them could point to an instance in the psychology, psychiatry, or education literature in which any treatment had produced similar outcomes for children with autism, regardless of cognitive ability at baseline. There were shortcomings in Lovaas’s participant matching and assessment procedures, but the magnitude of effects obtained were so unprecedented that it appeared that the critics were quibbling. Even if there were methodological weaknesses, the results were striking. Over the next decade numerous replications of basic components of Lovaas’s intervention generally yielded similar results (e.g., Harris & Handleman, 2000; Sallows & Graupner, 2005; Smith, Groen, & Wynn, 2000). Lovaas and colleagues conducted a longer-term follow-up study of the initial group of children who had received intensive behavior therapy and found that several graduated from high school and some had entered college (Lovaas & Smith, 1998; McEachin, Smith, & Lovaas, 1993; Smith, Eikeseth, Klevstrand, & Lovaas, 1997).

While some clinicians and many parents claim that children undergoing IEBT recover from autism (Madore Family’s Autism Resource Site; Maurice, 2001; Saffron, 2005), such statements appear to inadequately characterize children’s outcomes. Many children with autism who achieved optimal outcomes of intensive early behavioral intervention continue to display subtle psychological, language, and some behavioral differences on close inspection (e.g., Sallows & Graupner, 2005). However, as a practical matter, many children who would never have spoken or had been largely echolalic and who led segregated lives in classrooms for severely disabled children due to very limited skills and serious problem behavior are now participating members of their families and are attending regular education classrooms. In the past these individuals grew up to live in sequestered residential settings and often developed progressively disturbing behavior problems such as repetitive stereotyped movements, self-injury, and aggression. Today many individuals with autism who have similar baseline characteristics are living successfully with minimal supports as adults in typical community settings.

**New Developments in Genetics and Neuroscience: What is Now Proved Was Once Only Imagined**

Between the time Skinner staked out his position opposing explanatory linkages between genetics, brain science, and behavioral analysis and the present there has been an extraordinary growth of new technologies with which to study brain development, brain function, and genetics, which he could not have anticipated. It has become possible to obtain images of the brain at work as an individual with autism reads and interprets words or reacts to images of facial expression. Techniques permitting measurement of events within nerve cells as learning occurs and measures of the expression of as many as 30,000 human genes are possible. It has now become fruitful to explore the relations among genetic, neurobiological, and behavioral variables in ways that were unthinkable only a few decades ago (Kennedy, Caruso, & Thompson, 2001; Moore, 2002). Many of what
Skinner considered “explanatory fictions” have been transformed into objectively measurable variables that can be examined in relation to behavioral phenomena. Some within the field of behavior analysis continue to question the explanatory gain by integrating knowledge from different levels of analysis, and they suggest that a reinforcement contingency and stimulus control analysis are sufficient to provide an explanatory account of autism etiology (e.g., Drash & Tudor, 2004). Some even imply that functional brain imaging measures are fanciful reconstructions that fail to accurately represent reality (Faux, 2002; Uttal, 2004). The dramatic success of IEBT in engendering enduring improvements in cognitive, language, and social functioning in half of children treated raises the question why the other 25-50% of children treated profit far less from what appears to be the same treatment. Lovaas (1987), Bibby and colleagues (2002), Goldstein (2002), Lord and Paul (1997), and, recently, Sallows and Graupner (2005) reported that children with IQs below 50 who displayed low social responsiveness and who do not exhibit motor or verbal imitation at the outset of treatment are among those who profit least. They may be the same type of children that Miles et. al. (2005) describe as their dysmorphic children. In their study, Miles et. al. (2005) found those children were more likely to exhibit brain MRI abnormalities and other dysmorphia. ASDs did not recur in families of children with dysmorphic autism, while recurrence was more common among non-dysmorphic children with autism, suggesting a genetic component to their etiology. The onset of autism was more likely to be regressive in dysmorphic children with autism. The ratio of males to females within the dysmorphic group were similar, while in the non-dysmorphic group the ratio was approximately 3:1 males to females. As nearly as can be determined, the type and intensity of intervention for the non-dysmorphic rapid learners and dysmorphic slow learners diagnosed with autism appear to be equivalent, suggesting that differences in learning during the course of IEBT for the two types of children cannot be attributed to differences in the behavior analytic procedures employed.

As Skinner (1945) pointed out over six decades ago, the skin is not an important barrier. Events occurring within the skin can have the same functional properties as those occurring distally. Neurochemical receptor events can have discriminative and reinforcing properties (see Kelly, Stoops, Perry, et. al., 2003; Lelas, Spealman, & Rowlett, 2000; Preston & Bigelow, 1991; Thompson & Pickens, 1971), and as we have suggested elsewhere (Thompson, Moore, & Symons, 2006), psychopathological states such as depression and anxiety can function as establishing operations (Michael, 2000), much like serotonin depletion during sleep deprivation (Kennedy & Meyer, 1996; Kennedy, Meyer, Werts, et. al., 2000) or physiological changes during the menstrual cycle (Carr, Smith, Giacin, et. al., 2000). Adopting a theoretical stance consistent with constitutive reductionism provides opportunities for people within the field of behavior analysis to extend their explanatory reach without sacrificing anything of importance. Mayr (1982) pointed out that it is the interaction among systems that distinguishes biological systems, not the reduction of the terms and concepts of one to the other. The antecedents and consequences of behavior can include events occurring within the
brain that increase our ability to describe the necessary and sufficient events that lead to enduring behavioral changes among young children with autism. Those neurobiological events may also predict which behavioral interventions lead to optimal outcomes. Intellectual reclusiveness has not served the field of behavior analysis well, and it is unlikely to do so in the future.

Linkages among genes, brain development, and psychological and behavioral outcomes as a result of intensive social intervention will be the focus of the following discussion. The goal is to explore the relation between the metatheoretical argument presented in Paul Meehl’s 1990 theory of schizophrenia and the present examination of autism, with special reference to the effects of IEBT.

**Autism Genetics**

There have been countless attempts to identify chromosomes uniquely associated with autism, and in some cases to pinpoint an autism gene or genes (e.g., Lauritsen & Ewald, 2001). Genes are segments of DNA that provide the transcription instructions to create proteins, which in turn may be required to construct the cytoarchitecture of nerve cells such as the actin of axonal growth cones that propel axons on their way from point A to point B in the brain. Other proteins may be used as components of metaboreceptors, devices that transduce neurochemical information into permanent changes in synaptic conductivity. Some gene-produced proteins may become components of enzymes that are necessary to fabricate neurochemical transmitter molecules gathered together in vesicles, slowly migrating down an axon and collecting in the pre-synaptic space, waiting to be called upon when needed. One or some combination of these genes is likely at the root of the cause of autism endophenotypes. Synapses are created and consolidated through experience and the neurons are spared—or if potential synapses are not established and used those cells regress and are lost. A genetic defect that leads to failure to form and consolidate synapses is proposed to be the counterpart of what Paul Meehl called hypokrisia in his theory of schizophrenia.

It is unlikely that a single autism gene will be identified (see the Online Mendelian Inheritance in Man website; OMIM 209850; 12-21-05). As with schizophrenia, it is more likely that understanding of gene-brain-behavior causal sequences in autism will arise from studying endophenotypes, not cases with the myriad of variations seen in clinically encountered phenotypes. In some cases, people suspected of having an ASD are likely to be phenocopies that lack the genetic precursors. Their features arise from some combination of social and/or polygenic potentiators, not the genetic predisposition to autism. Gottesman and Shields (1967) argued that simpler clues to genetic underpinnings than the disorder syndrome itself can be found which can result in more successful genetic analysis. An endophenotype may be neurophysiological, biochemical, endocrinological, neuroanatomical, cognitive, or neuropsychological. However, to be most useful, endophenotypes must meet certain criteria, including association with a candidate gene or gene region, heritability that is inferred from relative risk for the disorder.
As noted earlier, Miles and colleagues (2005) present persuasive evidence consistent with an endophenotypic approach to studying autism. They argue that heterogeneity within the autism diagnosis obscures genetic bases of the disorder and impedes development of more effective treatments. They compared children with autism with and without microcephaly and dysmorphology. Complex Autism consists of individuals who appear to have experienced an abnormality of early morphogenesis, manifested by either significant dysmorphology or microcephaly. Individuals with complex autism comprised 20% of the total autism group. Individuals with complex autism have lower IQs, more seizures, more abnormal EEGs, and more brain abnormalities by MRI. Everyone with an identifiable syndrome (i.e., polygenic potentiators) was in the complex group. Essential Autism comprised the more heritable group with higher sibling recurrence, with more relatives with autism (Miles et. al., 2005).

Despite compelling indications of multiple factorial etiology of autism, there is also strong evidence supporting genetic contributions to autism spectrum disorders. Folstein and Rutter (1977) reported that about 2% of siblings are affected (far higher than chance) and that speech delay is common in the sibships containing autistic children. In a study of 21 same-sex twins pairs, 11 monozygotic (MZ) and 10 dizygotic (DZ), in which at least one twin had autism, Folstein and Rutter (1977) found 36% concordance among the MZ twins and no concordance among the DZ twins. The concordance for cognitive abnormalities was 82% for MZ pairs and 10% for DZ pairs.

Several chromosomes have been linked to autism within families using linkage analysis and molecular genetic techniques. According to the OMIM database website operated by the National Institutes of Health (OMIM, 12-21-05), one form of autism (AUAT4) has been mapped to chromosome 15q. Other loci have been mapped to 7q (AUT1), 3q25-27 (AUT2), 13q14 (AUT3), 2q, 17q21. Susceptibility to autism has also been linked to glyoxalase I gene (GLO1). Three X-linked forms of autism (AUTSX1, AUTSX2, AUTSX3) are caused by mutations in the NLGN3, NLGN4, and MECP2 genes, respectively. Each of these forms of autism has distinctive features and no doubt somewhat different brain abnormalities.

The GENSAT database maintained by the NIH National Center for Biotechnology Information maintains an atlas of mouse genes (homologues to human genes) expressed in the brain, ranking them by their degree of expression within specific brain structures (see http://www.ncbi.nlm.nih.gov/projects/gensat/). Many of the same genes have been found to be strongly expressed in structures that are commonly dysfunctional in autism and differing in degree of expression from those in other brain structures (e.g., amygdala, basal frontal cortex, hippocampus, and cerebellum). Some genes common to these brain structures listed in the NCBI database are specifically involved in processes related to synaptogenesis or receptor function, such as acetylcholine, dopamine, GABA, and
serotonin receptor genes. Others, such as synaptosomal-related protein, synaptogyrin, and synaptotagmin and ubiquitin protein ligase are important in brain development. If one or some combination of these shared genes were deleted or inactivated it is plausible that they would result in substantial differences in brain development in autism.

One mechanism underlying the plasticity failure may involve inadequate retraction of exuberant synapses, which are activity-dependent (Boulanger & Shatz, 2004). Non-functional dendritic spines are excessively long and do not convey synaptic currents from the head of the spine to the dendrite (and cell body) to which the extra-long spine neck is attached. In normal brains dendritic spines change continuously through typical use and disuse. The spines contract with mechanisms similar to muscle actin and myosin. Early disuse could reduce expression of the molecules needed for spine contraction, which is required to make the spine functional.

The task is to identify the genes that produce the proper amount of substrate required for a specific plasticity-related function (e.g., dendritic spine contractions) and are only turned on by experience. The Major Histocompatibility Complex (MHC) plays a major role in immunity and has also been reported to be related to autism. Increased frequency of a common abnormal chromosomal locus has been reported among people with autism, their mothers, or both (40%) as compared to controls (2%; Daniels, Warren, Odell, et. al., 1995; Warren, Singh, Cole, et. al., 1992). Corriveau, Huh, and Shatz (1998) found that a class of genes involved in the MHC complex plays a role in activity-dependent neuroplasticity as well as in immunity. They are involved in neuronal differentiation and synapse plasticity, and they are sensitive to the effects of neuron activation (i.e., experience). It appears that MHC 1 molecules may translate neuronal activity (e.g., due to experience) into lasting synaptic connectivity. That raises the question whether the MHC 1 genes, per se, are faulty in autism (producing too little or dysfunctional proteins), or whether normal genes are being prevented from turning on due to lack of experiential input. IEBT could have the effect of compensating for the lack of necessary protein required for synapse formation or insufficient experiential input.

“Big Symptoms” and Endophenotypes

In his 1990 paper Meehl hypothesized a single underlying anatomical, physiological, or neurochemical defect he called hypokrisia that (1) influenced nearly all brain organizational levels somewhat, (2) influenced some levels more than others, and (3) was clinically, quantitatively “fuzzy” due to other individual difference factors and specific experiences (e.g., traumas). Meehl proposed focusing attention on “big symptoms,” by which he meant strong statistical evidence that they reliably distinguished the schizotype or schizophrenic from people in general as well as from people with other mental illnesses. Meehl argued that these variables are less dependent on social learning history and life experiences and are therefore more likely to reflect underlying schizotaxia.
Some autism indicators found in the DSM and ICD are approximations of what Meehl called “big symptoms” and are objectively measurable. Common clinical practice, which is often highly subjective, is distinguished from cognitive and behavioral assessments done by trained professionals using standardized scoring criteria. Electrophysiological and brain imaging measures are more akin to similar variables in Meehl’s categories of anatomical, neurophysiological, perceptual, and language differences.

Brain Development in Autism

Around the time Lovaas (1987) reported his initial follow-up findings of the effects of IEBT with children who had ASDs, research on brain structure and function in autism—and our understanding of brain development more generally—was emerging rapidly. Consistent abnormalities have been found in brain anatomy and functioning in several specific structures that will be briefly reviewed here: amygdala, fusiform gyrus, orbitofrontal cortex, and hippocampus. Less consistent (but frequent) findings have implicated the cerebellum and two language areas, Wernicke’s and Broca’s areas.

Bauman and colleagues reported structural abnormalities in specific areas in brain tissue from young children with autism. Light microscopic brain tissue studies from individuals with autism revealed a paucity of Purkinje cells and granular cells in parts of the cerebellar cortex and smaller than normal, more tightly packed cells in some cerebellar nuclei and limbic structures, including the amygdala and hippocampus. These cells exhibited poor dendritic arborization and far less synapse formation (summarized in Bauman & Kemper, 1994). Using magnetic resonance imaging (MRI), Schumann and colleagues (2004) studied localized brain volume differences among children and adolescents with autism compared with same-age groups with intellectual disabilities and typical controls. They found that 7.5- to 12.5-year-old youths with autism had larger amygdala volumes than control children. However, there were no differences in amygdala volumes between the adolescent groups (12.75-18.5 years of age). The amygdala in typically developing children increases substantially in volume from 7.5 to 18.5 years of age. Thus, the amygdala in children with autism was initially larger but did not undergo the age-related increase observed in typically developing children, suggesting that cell loss must have occurred. Other researchers have shown similar amygdala volume differences as well as abnormalities in the relative sizes of Broca’s and Wernicke’s areas, which are involved in speech recognition and production (Sparks, Friedman, Shaw, et. al., 2002; Ziegler, Makis, Fillipek, et. al., 2004).

Subsequent functional brain imaging studies (fMRI) revealed reliable differences in activation in several brain areas of children and adolescents with autism. The amygdala plays and important role in recognizing emotions expressed by others and appropriately expressing emotion. Brothers (1990) proposed a theory of the “social brain,” hypothesizing that a network involving the orbito-frontal cortex (OFC), superior temporal gyrus (STG), and amygdala were central to the
cognitive and emotional deficit seen in autism. Baron-Cohen and colleagues (1999) tested Brothers’ theory by examining both typical subjects as well as individuals with high-functioning autism or AS. The task involved judging what another person might be thinking or feeling based on photographs of the expressions of another person’s eyes. During this task Baron-Cohen and colleagues found typical participants experienced increased activation in the superior temporal gyrus, amygdala, and some areas of the prefrontal cortex. In contrast, individuals with autism or AS did not activate the amygdala when attempting to draw inferences from images of eyes. Kawashima and colleagues (1999) determined the brain areas involved in gaze monitoring by typical subjects using functional neuroimaging. When asked to discriminate the direction of gaze, a region in the left amygdala was activated. However, a region in the right amygdala was specifically activated only when performing the task while maintaining eye contact. This suggests that the left amygdala plays a role in interpreting eye gaze direction and that the activity of the right amygdala increases when another individual’s gaze is directed toward her or him. These and other related findings suggest that the human amygdala plays a role in reading social signals from the face, which is specifically deficient in autism (Hooker, Paller, Gitelman, et. al., 2003).

In comparing brain structure of boys with autism with matched controls, Herbert and colleagues (2002) found significant posterior temporal fusiform gyrus asymmetry (more left-sided in autism as compared with controls), whereas adjacent fusiform gyrus and temporo-occipital inferior temporal gyrus both approached significance (more right-sided in autism than controls). These inferior temporal regions are involved in interpreting faces and facial expression. Schultz (2005) suggests that early developmental failure in autism begins with the amygdala, with a cascading influence on the development of cortical areas that mediate visual social perception, specifically the fusiform “face area” of the ventral temporal lobe (Schultz, 2005). People with autism generally attend less to faces than matched controls (Dawson, Meltzoff, Osterling, & Brown, 1998; Klin, Jones, Schultz, et. al., 2002). Several investigators have found lack of activation of the fusiform gyrus during face recognition tasks in brain imaging studies with individuals with autism. Other studies have shown that the degree of fusiform gyrus activation is related to the amount of experience people have with a particular category of visual stimuli (e.g., cars, faces, tools). Since people with autism seldom look at faces, they have little opportunity to enhance connectivity in the fusiform gyrus when looking at faces (i.e., they are not “experts” in faces as are most people who devote a great deal of time visually studying people’s facial expression; Grelotti, Gauthier, & Schultz, 2002; Schulz, 2005).

Areas of the frontal cortex have been repeatedly found to fail to activate among individuals with autism when confronted with specific provocative visual tasks, in particular the orbitofrontal cortex. Luna, Minshew, Garver, et. al. (2002) found that autistic subjects demonstrated significantly less task-related activation in the dorsolateral prefrontal cortex (Brodmann area [BA] 9/46) and the posterior cingulate cortex (BA 23) in comparison with typical subjects during a spatial
working memory task. Courchesne and colleagues (2005) reported underdevelopment of the cerebellum and hypothesized that microstructural maldevelopment results in reduction in frontal-posterior reciprocal connectivity. These connections normally mediate the complex social, emotional, speech, language, attention, and cognitive functions that are dysfunctional in autism. They point out that cellular and growth pathologies are reduced or nonexistent in other structures in autism (e.g., occipital cortex) and they argue that this altered circuitry impairs frontal cortex from integrating information from emotional, sensory, autonomic, memory, and other systems.

**Language Cortical Areas**

Several reports have suggested anatomical and functional differences in brain areas important in language in autism. Herbert and colleagues (2003) used morphometric MRI to examine cortical asymmetries in children with autism aged 7 to 11 years with a nonverbal IQ greater than 80, and 15 age- and MA-matched controls. Boys with autism had significant asymmetry reversal in the frontal language-related cortex: 27% larger on the right in autism and 17% larger on the left in controls. DeFosse and colleagues (2004) studied brain volumes using MRI in boys with specific language disabilities alone and those with autism with and without language impairment. They found that language-impaired boys with autism and specific language disabilities both had significant reversal of asymmetry in the frontal language-related cortex: larger on the right side in both groups of language-impaired boys and larger on the left in both unimpaired language groups. In another report Just and colleagues (2004) compared brain activation of a group of participants with high-functioning autism and a verbal IQ-matched control group using fMRI during sentence comprehension. The groups differed in the distribution of activation in two of the key language areas. The autism group produced reliably more activation than the control group in Wernicke’s (left laterosuperior temporal) area and reliably less activation than the control group in Broca’s (left inferior frontal gyrus) area. Furthermore, the degree of synchronization of the activation between the language cortical areas was lower for the autistic than the control participants. This suggests a lower degree of information integration and synchronization across the language cortical network.

Prosody refers to the ability to express and interpret emotions through variations of the human speech such as pitch contour, intensity, and duration (Besson, Magne, & Schon, 2002). Speakers with ASDs display difficulties with prosody (Paul, Augustyn, Klin, & Volkmar, 2005). Since the 1980s it has been known that the ability to interpret the emotional meaning of speech depends on structures in the right temporal lobe (Ross, Harney, deLacoste-Utamsing, et. al., 1981). People with injury to this brain area are often able to repeat words that are spoken verbatim and state their literal meaning, but if asked to report whether the speaker was angry, sad, or happy they are unable to do so. They may also fail to understand metaphors and idiomatic speech. More recent studies using brain imaging methods have isolated several other brain areas involved in prosodic
processing, including the anterior cingulate and the prefrontal cortex (Barrett, Pike, & Paus, 2004; Buchanan et. al., 2000). There is also evidence of differences in prosodic processing in females and males. Schirmer and colleagues (2002) reported differences between men and women in emotional prosodic processing. Women base their linguistic expectations on emotional prosody whereas men process word meaning independently of emotional prosody.

Baron-Cohen, Knickmeyer, and Belmonte (2005) point out that female infants and toddlers make use of non-verbal cues in reacting to the content of speech, whereas male youngsters react primarily to speech semantics. Baron-Cohen has referred to autism as a case of the “extreme male brain,” meaning that children with autism exhibit typical male characteristics in an exaggerated form. Individuals with autism usually have difficulty expressing emotions verbally, including producing prosody that matches their emotional state. Clinicians have reported that girls with autism are more attuned to the affective aspects of speech and tone of voice than boys, but these differences have not been clearly corroborated in well-controlled studies. Other work indicates that fetal testosterone levels are negatively correlated with quality of social relationships and positively correlated with restricted interests in boys at 4 years of age (Knickmeyer, Baron-Cohen, Raggatt, & Taylor, 2005). Thus, sex hormones may play an interactive role (e.g., gating G proteins) in facilitating or inhibiting development of social skills, including some language functions.

**IEBT and Neuroplasticity**

Whatever leads to the histopathological, volumetric MRI and microanatomical deficiencies in specific brain structures and associated lack of activation by provocative stimulation tasks as revealed by electrophysiological, fMRI, and PET analyses discussed previously should be amenable to amelioration by experience. Possible mechanisms should be consistent with IEBT evidence. It is unlikely that experience would lead to new layering of neurons in the cortex (i.e., cell migration up microglia) in 2- to 5-year-old children. Similarly, it seems improbable that experience would induce de novo axonal growth, which would involve axons finding their way to distant brain areas to form new connections. It is more likely that the underlying defect (hypokrisia in Meehl’s 1990 paper) is related to neuroplasticity.

The latter period of maximal neuroplasticity coincides with the period in which intensive behavior therapy appears to have its greatest effect (see Figure 3). IEBT is typically initiated between 2 and 4 years of age, toward the latter part of maximal neuroplasticity. Beyond 24 months of intensive therapy, improvement in a child’s cognitive and language functioning appears to gradually diminish with continued treatment (Sallows & Graupner, 2005). Synaptogenesis normally increases rapidly from 4 months to around one year of age, declining fairly rapidly until around 4 years of age. The rate of synapse formation more gradually diminishes from that point until around 11 years of age. The rapid decline in new synapses is thought to be due to reductions in the number of dendritic spines per
nerve cell over that interval rather than cell pruning (Huttenlocher, 1984), which suggests that IEBT promotes neuroplasticity from around 2 to 4 or 5 years then reaches a point of diminishing returns. Neuronal regression of cells that are not used occurs from around 8 months to 11 years of age (Huttenlocher & de Courten, 1987). IEBT may promote new synapses and consolidate established synapses rapidly from 2 to 5 years of age if they are autotaxic, which would have the effect of preventing cell loss as neuronal regression occurs. If the child does not have the underlying genetic condition causing hyposynapsia but suffers damage to the same structures by a different mechanism (e.g., mislayering of the cortex, erroneous axonal guidance, or reduced neuronal numbers), IEBT would be expected to have more limited effects.

Figure 3. Synaptic density in layer I and layer II/III of striate cortex. Open circles = Layer I; Closed circles = Layer II/III (Huttenlocher & de Courten, 1987, Figure 3)

**Brain Imaging and Selecting Targets of IEBT**

Experience required to produce these changes must be specific, intensive over a prolonged period of time, and involve active responding to relevant stimuli in the child’s environment. Passively listening to, or visually observing, computer-generated stimuli, for example, would likely produce results comparable to attempting to learn a foreign language by listening to instructional tapes while
asleep. Presenting the young child with autism with an array of visual and auditory stimulation (e.g., sensory integration therapy) does not produce lasting behavioral changes comparable to those from IEBT (see Lord & McGee, 2001). The child’s experience must enlist activity in the specific brain structures that are empirically demonstrated to be minimally responsive in brain imaging analyses and synaptically underdeveloped.

Selecting Optimal Interventions

Learning experiences provided in the course of IEBT are usually selected based on baseline behavioral assessments of entry-level cognitive, language, and social skills. A standardized assessment tool, such as the Assessment of Basic Language and Learning Skills (ABLLS) developed by Partington and Sundberg (1998), is a reasonable beginning point but may not be sufficient alone. The ABLLS is a prescriptive assessment tool broken into 25 face valid domains, each with a series of concrete sub-skills that are scored based on the child’s ability to perform the skill and with what degree of independence (See Table 1).

Table 1: Assessment of Basic Learning and Language Skills
(Partington & Sundberg, 1998)

<table>
<thead>
<tr>
<th>ABLLS Scale</th>
<th>Skill</th>
<th>No. of Items</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Cooperation and Reinforcer Effectiveness</td>
<td>11</td>
</tr>
<tr>
<td>B</td>
<td>Visual Performance</td>
<td>21</td>
</tr>
<tr>
<td>C</td>
<td>Receptive Language</td>
<td>52</td>
</tr>
<tr>
<td>D</td>
<td>Imitation</td>
<td>13</td>
</tr>
<tr>
<td>E</td>
<td>Vocal Imitation</td>
<td>9</td>
</tr>
<tr>
<td>F</td>
<td>Requests</td>
<td>27</td>
</tr>
<tr>
<td>G</td>
<td>Labeling</td>
<td>42</td>
</tr>
<tr>
<td>H</td>
<td>Intraverbals</td>
<td>42</td>
</tr>
<tr>
<td>I</td>
<td>Spontaneous Vocalizations</td>
<td>9</td>
</tr>
<tr>
<td>J</td>
<td>Syntax and Grammar</td>
<td>20</td>
</tr>
<tr>
<td>K</td>
<td>Play and Leisure</td>
<td>10</td>
</tr>
<tr>
<td>L</td>
<td>Social Interaction</td>
<td>22</td>
</tr>
<tr>
<td>M</td>
<td>Group Instruction</td>
<td>12</td>
</tr>
<tr>
<td>N</td>
<td>Classroom Routines</td>
<td>10</td>
</tr>
<tr>
<td>P</td>
<td>Generalized Responding</td>
<td>6</td>
</tr>
<tr>
<td>Q</td>
<td>Reading</td>
<td>15</td>
</tr>
<tr>
<td>R</td>
<td>Math</td>
<td>42</td>
</tr>
<tr>
<td>S</td>
<td>Writing</td>
<td>9</td>
</tr>
<tr>
<td>T</td>
<td>Spelling</td>
<td>6</td>
</tr>
<tr>
<td>U</td>
<td>Dressing</td>
<td>16</td>
</tr>
<tr>
<td>V</td>
<td>Eating</td>
<td>10</td>
</tr>
<tr>
<td>W</td>
<td>Grooming</td>
<td>7</td>
</tr>
<tr>
<td>X</td>
<td>Toileting</td>
<td>10</td>
</tr>
<tr>
<td>Y</td>
<td>Gross Motor</td>
<td>28</td>
</tr>
<tr>
<td>Z</td>
<td>Fine Motor</td>
<td>28</td>
</tr>
</tbody>
</table>

The number of sub-scale items making up each scale is indicated in parentheses following the name of the scale (Partington & Sundberg, 1998). Scales Q-Z are non-specific skills generally useful for most children with developmental delays.
Of the 25 scales, which have from 6 to 52 sub-scales, there is little to guide the clinician in selecting which to emphasize. The specific skill sets within the ABLLS (or any other psychological or behavioral assessment tool) that would most profitably be primary targets of behavior therapy intervention would be those requiring use of the specific brain areas in which there is empirical evidence of dysfunction. Most evidence comes from fMRI activation studies that indicate the loci of minimal activation during provocative stimulation tasks.

Table 2 presents the ABLLS scales, sub-scales, and the associated brain areas that would likely be required to perform the specific learning tasks listed. The ABLLS minimally addresses some signs and symptoms of autism such as discrimination of emotions from facial expression, receptive prosody, or flexibility in repairing failed communicative attempts. To develop a comprehensive intervention that will address all of the major autism features, additional objectives and intervention procedures would likely be required.

Table 2: Structures commonly dysfunctional among children with autism in brain imaging studies (column 1), functions of those structures (column 2) and ABLLS scales and subscale items that would require the use of those structures to achieve the goals specified in the curricular manual (Partington & Sundberg, 1998)

<table>
<thead>
<tr>
<th>Brain Structure</th>
<th>Function</th>
<th>ABLLS Scales</th>
</tr>
</thead>
<tbody>
<tr>
<td>Superior Temporal Sulcus</td>
<td>Gaze detection</td>
<td>L9 Eye Contact</td>
</tr>
<tr>
<td></td>
<td></td>
<td>L12 Looks at Others to Start Social Interaction</td>
</tr>
<tr>
<td>Amygdala</td>
<td>Affect Recognition and Affect Expression</td>
<td>None Currently Developed</td>
</tr>
<tr>
<td>Fusiform Gyrus; Inferior temporal gyrus</td>
<td>Face Recognition</td>
<td>L9 Eye contact</td>
</tr>
<tr>
<td></td>
<td></td>
<td>L12 Looks others to start social interaction</td>
</tr>
<tr>
<td></td>
<td></td>
<td>M3 Attends to teacher in group</td>
</tr>
<tr>
<td></td>
<td></td>
<td>M4 Attends students in group</td>
</tr>
<tr>
<td>Orbito-Frontal Cortex</td>
<td>Social Judgment &amp; Planning</td>
<td>B5 Sort non-identical items into categories</td>
</tr>
<tr>
<td></td>
<td></td>
<td>B6 Block designs on picture cars</td>
</tr>
<tr>
<td></td>
<td></td>
<td>B7 Block designs from pictures</td>
</tr>
<tr>
<td></td>
<td></td>
<td>B8 Sequence pattern to match visual model</td>
</tr>
<tr>
<td></td>
<td></td>
<td>B15 Delayed replication of a sequence</td>
</tr>
<tr>
<td></td>
<td></td>
<td>B16 Delayed finding a sample</td>
</tr>
<tr>
<td></td>
<td></td>
<td>B.18. Replicate simple 3 dimensional object</td>
</tr>
<tr>
<td></td>
<td></td>
<td>B19 Seriation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>B20 Picture sequences</td>
</tr>
<tr>
<td></td>
<td></td>
<td>N Classroom Routines</td>
</tr>
</tbody>
</table>

Table continued on next page
<table>
<thead>
<tr>
<th>Brain Structure</th>
<th>Function</th>
<th>ABLLS Scales</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medial Temporal Gyrus</td>
<td>Process phonemes &amp; ph. combinations; needed for reading</td>
<td>C. Receptive Language (C1-52)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>L5 Listener-Receptive</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Q. Reading</td>
</tr>
<tr>
<td>Wernicke’s Area; Left posterior superior temporal gyrus</td>
<td>Receptive language</td>
<td>C. Receptive Language (C1-52)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>J. Syntax &amp; Grammar</td>
</tr>
<tr>
<td>Broca’s Area; Inferior frontal gyrus of frontal lobe</td>
<td>Expressive language; Lesions → expr motor aphasia &amp; non-fluent aphasia</td>
<td>E. Vocal Imitation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>F. Requests</td>
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<tr>
<td></td>
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<td>G. Labeling</td>
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<td>H. Intraverbals</td>
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<td></td>
<td></td>
<td>I. Spontaneous Verbalization</td>
</tr>
<tr>
<td></td>
<td></td>
<td>J. Syntax &amp; Grammar</td>
</tr>
</tbody>
</table>

It is hypothesized that repeatedly practicing the skills in Column 3 should promote synaptogenesis in structures in Column 1, thereby improving the cognitive communicative and emotional functions in Column 2.

**Therapy Timing and Dose**

Beginning intensive therapy prior to 4 years of age appears to produce more substantial lasting cognitive and language gains than later therapy onset (e.g., Fenske, Zalenski, et. al., 1985; Lord & McGee, 2001). Capitalizing upon maximal plasticity before neuronal regression leads to substantial cell loss is presumed to optimize child outcomes. Much as the quantity of a neurochemical transmitter released and binding to a receptor determines neuronal firing rate, it appears that the *dose of behavioral intervention* determines whether the behavioral changes are transitory or permanent. To produce lasting behavioral, cognitive, and language changes it appears from most reviews of this literature that children with autism require 20-25 hours per week or more of intervention for several years. Consolidating the synaptic changes that underlie the behavioral changes appears to require extensive repetition over prolonged periods. Behavioral intervention must be focused, with one therapist per child for much of the therapy time rather than providing instruction to the child with autism within a large group of typical children or others with disabilities. In the latter arrangement the child with ASD has insufficient opportunities to practice essential skills with adequate frequency over time. When IEBT was introduced some developmental psychologists and other professionals were critical of the approach on the grounds that such intensive, repetitive instruction does not appear to be developmentally appropriate (e.g., Greenspan, Wieder, & Simons, 1998). Developmental appropriateness is important in child interventions, as emphasized by Lord and McGee (2001), but that must be understood in the context of the nature of the disability. As it is sometimes posed, this criticism overlooks the fact that teaching a child with autism who has substantial deficiencies in brain connectivity in specific areas is a very different matter from teaching a typical same-age toddler without that disadvantage.
Which Behavior Therapy Approach is Most Effective?

Though there are strong differences of opinion among practitioners in the field, there is no convincing evidence that one intensive behavior therapy method produces superior results than another. The UCLA Young Autism Program and its replications are the only therapy methods that have been subjected to long-term follow-up, so it is impossible to know whether other IEBT methods yield similar outcomes. Some IEBT approaches are quite different from Lovaas’s original method; some emphasize incidental teaching within natural environments (Vincent Carbone, Ed.D., http://www.drcarbone.net/) and teaching broader problem solving, independence, and social skills (McClannahan & Krantz, 1999) that serve common functions in the child’s day-to-day life (e.g., Koegel & Koegel, 2005). Some approaches have greater emphasis on the array of social functions of language (Sundberg & Partington, 1998). However, all of these methods are based on the same behavior analytic principles and emphasize consistency and repetition, with interpolated free play and other periods for socializing with peers.

A common misconception regarding the various IEBT approaches is that they ignore the importance of social relationships. Most IEBT approaches begin with 20 to 30 hours over a week or more of therapists observing, playing with, and becoming familiar with the child and the child becoming comfortable with the therapists. The child’s initial wariness of strangers gives way to their looking forward to the therapists’ arrival to play with them—at least that seems to be the way the children see it. A critical factor in enlisting the child’s willing participation is their learning that the therapists will never ask them to do anything that is frightening, unduly frustrating, or upsetting to them. That is the reason why therapists must thoroughly understand the child’s beginning skill levels in each domain and begin therapy at, or slightly below, their entry-level performance. The child’s success at each step makes their participating in therapy something they look forward to each day. In most instances, within a week or so of regular exposure, as soon as the therapists enter the child’s home for a therapy session, the toddler runs up to the therapists to be held, tickled, or hugged. The child must trust the therapists and see them as a source of enjoyment and acceptance in order for subsequent therapy to be effective. In behavior analytic parlance, the therapists’ smiles, hugs, and encouraging positive remarks become generalized conditioned positive reinforcers for the child’s performance of new skills. For their part, the therapists’ continued attempts to help the child develop new skills are maintained by the child’s obvious pleasure at being able to make their wishes understood without having to scream and cry. Those momentary joys, combined with graphic evidence of the progressive growth of essential communicative and social skills, keeps the therapists coming back to work with a child who has ASD.

Discussion and Conclusions

In his parables, the 18th century poet William Blake wrote, “What is now proved was once only imagin’d.” Skinner can be excused for not imagining the
profound changes that would occur in genetics and neuroscience that could increase the reach of his science. Providing a more complete account of the processes involved in reversing some of the features of autism begins to change the explanatory landscape. Moreover, we are now in a position to better predict which specific behavior analytic interventions will produce optimal developmental outcomes for children with autism. This provides a more complete understanding of the reasons IEBT methods are effective and predict which specific methods should be used, and it begins to suggest where to look to for the neurogenetic and neurobiological etiologies of autism.

Reading Meehl’s exuberant prose is intellectually exhilarating as well as challenging. His expository style was to the intellect what John Coltrane’s improvisation was to jazz (Nisenson, 1993). Meehl’s prolix, discursive approach to composition appears to have closely followed his intellectual processes rather than being the product of carefully edited syllogistic afterthoughts. The result is a complex network of exploratory tangential allusions, intentional explorations of blind alleys, and brilliant insights. The richness of Meehl’s relational vocabulary attests to its importance to emergent creative behavior. When he used a word or phrase in a sentence, that set the occasion for a half dozen other words, phrases, concepts, or images to rise to strength, from which he selected one or two to incorporate into the remainder of the sentence or to be inserted as a parenthetical comment. In the end, the reader understands how Meehl got from point A to point Z and why he rejected numerous appealing alternatives along the way.

The basic structure of Meehl’s 1990 schizophrenia argument provides a primer in clear thinking about the complex linkages among genes, brain, and behavior. It reminds us that, as Poincare (1905) wrote, “Science is facts; just as houses are made of stones, so is science made of facts; but a pile of stones is not a house and a collection of facts is not necessarily science.” Meehl and Skinner differed about the most effective strategy for making psychology into something more than a pile of stones, each strategy having merit in its own way. Skinner prized internal consistency and powerful demonstrations of prediction and control of limited phenomena, at times at the expense of comprehensiveness. Paul Meehl’s metatheoretical preference for scientific theory transcending levels of analysis is an apt model not only for the scientific study of schizophrenia—it applies to other complex conditions like autism. The present paper suggests that there are times when Paul Meehl’s multi-layered, Mobius strip approach can be especially effective, including helping us understand the importance of behavior analytic principles and procedures.

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