

ADAPTIVE WILL: THE EVOLUTION OF ATTENTION DEFICIT DISORDER

ANDREW LAKOFF

The increasing prevalence of attention-deficit disorder among American school children was a source of significant controversy in the 1990s. This paper looks at the social and historical contexts in which ADD evolved in order to understand its emergence as a coherent and widespread entity. Changes in expert models of child behavior interacted with the formation of new identities around disability to shape a milieu in which the disorder could thrive.

The pattern of affect control, of what must and what must not be restrained, regulated, and transformed, is certainly not the same in this stage as in the preceding one of court aristocracy. In keeping with its different interdependencies, bourgeois society applies stronger restrictions to certain impulses, while in the case of others aristocratic restrictions are simply continued and transformed to suit the changed situation (Elias, 1994, p. 125).
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In *The Civilizing Process*, Norbert Elias traced the history of European manners to show how changes in social and political structures have been accompanied by new restrictions on the expression of bodily impulses. Throwing into relief the contingency of modern social regulations, his study leads us to reflect on the contemporary work of the civilizing process. While we now take for granted rules against relieving ourselves in public or using our neighbor's eating utensils, these were accomplished painfully through centuries of work. In the flash of the present, how can we detect emerging behavioral regulations linked to social status? If Elias used etiquette manuals to trace the changing consciousness of proper social form, what sources might we analyze to seek out the changes in our midst? I propose to look at the sciences of child observation as a site where one may witness the continued unfolding of the civilizing process. In this essay, I examine expert knowledge of children—specifically, at a newly emergent pathology of social development—as a source of insight into the changing norms that are embodied in healthy behavior. I begin with a discussion of late nineteenth century pathologies of the will in order to frame what may be distinctive about the contemporary situation.

THE EVOLUTION OF INHIBITION

George Still, a physician at King's College Hospital at the turn of the century, owes his continued celebrity to an observation that certain pathologies of moral control in children might be due to an organic cause. In his 1902 lectures to the Royal College of Physicians, Still presented a series of cases of children with normal intellect, but with "defects of inhibitory volition." One prominent problem they had was the inability to sit still, but there were other defects: stealing, lying, violence, and sexual chicanery apparently came naturally to these children. According to Still, this deficit could be understood as a severe lack of reserve signalled by persistent self-gratification, shamelessness, immodesty, and passionateness. This

ANDREW LAKOFF is a doctoral candidate in the Anthropology department at the University of California, Berkeley, and is the Marian E. Koshland Fellow at the Townsend Center for the Humanities. His dissertation is entitled, "Pharmaceutical Reason: Subject and Psychotrope in Buenos Aires."

“disorder of the will” was integrated with his larger picture of the evolutionary development of inhibitory volition. For him, volitional inhibition was the cornerstone of civilized behavior, a prerequisite to becoming a moral adult. The absence of such volition was a lapse that could potentially be correlated with other “stigmata of degeneration,” such as anomalous phrenological characteristics.¹ Thus, Still particularly was interested in the disproportionately large head circumference of these deviant children, and he took family histories to determine their forebears’ degree of moral degeneracy (Still, 1902, p. 1164).

Searching for the source of antisocial behavior with skull and tape measure was, of course, not unique to Still, nor does it explain his present renown. Rather, what would rescue him from obscurity in the annals of Victorian medicine was an aside in which he observed that “a notable feature in many of these cases of moral defect without general impairment of intellect is a quite abnormal incapacity for sustained attention” (Still, 1902, p. 1166). Not only were they unable to sit still, but these children also seemed unable to focus for long on any one thing: They were easily distractible, an inattentiveness Still associated with a lack of volitional control. To cement this link, he cited William James, who had written that “effort of the attention is the essential phenomenon of will.” James’s work had been inspired by the evolutionary psychologist, Théodule Ribot, who wrote the classic treatise on the psychology of attention in 1890. Named to the first chair of experimental psychology at the Collège de France, Ribot was a product of the first flood of evolutionism into theories of human behavior and was one of Spencer’s chief exponents in France. He distinguished between the faculty of spontaneous attention, an inborn trait, and voluntary or sustained attention, the product of education and training. While the former was present in children, animals, and savages, the latter could be won only through civilization:

The same progressive movement that in the order of moral events has caused the individual to pass from the control of instincts to that of interest and duty; in the social order, from primitive savagery to the state of organization; in the political order, from almost absolute individualism to the constitution of a government: this same onward movement, in the intellectual world, has also effected the transition from spontaneous attention to the dominance of voluntary attention. The latter is both effect and cause of civilization (Ribot, 1890, p. 42).²

Still’s children were reminders of the arduous process of civilization that, ideally, instilled moral self-restraint over the course of childhood. As evolutionary social science declined in the early decades of the twentieth century, the relation between attention, will, and moral behavior faded as a major problem. In fact, the very category of volition lost status as a structuring category in psychological research. Two contemporary historians of psychiatry have ascribed the decline of volition more to disciplinary politics than to intellectual necessity (Berrios & Gili, 1995). Proclaiming the continued relevance of will to the discipline, they have called for a return to the use of the category of volition in psychiatry. Such a demand may, however, be redundant, as the concept had already been revived in connection with a renewal of interest in George Still (Barkley, 1990; Hallowell & Ratey, 1994). This event was due, in turn, to the curious fact that, beginning seventy years after his observations, there was an outbreak of the disorder Still had described. Or at least something quite similar to it.

1. See Pick, 1989, for the cultural context of late nineteenth century fascination with the problem of degeneracy.
 2. Ribot also had written a tract on volition, called *Diseases of the Will*.

A NEW DISORDER OF THE WILL

The most commonly diagnosed mental disability among American schoolchildren in the 1990s, attention deficit disorder (ADD), sometimes with hyperkinesis (ADHD), was also the most recently recognized. Whereas Still's cases had been rarities, ADD was estimated to afflict at least 5% of the school population, predominantly boys. The disorder was defined as a behavioral syndrome characterized by impulsivity, disruptiveness, and difficulty in sustaining vigilant attention. There were two treatments recommended by experts: either behavioral retraining—an intensive process centered around reinforcement techniques such as timers and lists—and the more convenient remedy of stimulant medication (Barkley, 1990). In special schools and camps devoted to treating the disorder, the two often were used in combination. A variety of alternative treatments also appeared, testifying to parents' worries over administering brain-directed medications to their children (Garber, Garber, & Pizman, 1996).

Such reluctance was not surprising despite efforts by some child psychiatrists to frame psychostimulant treatment as akin to providing eyeglasses to the nearsighted or insulin to the diabetic (Hallowell & Ratey, 1994).³ Critics pointed to the possible contradictions involved in prescribing chemical stimulants to children while waging strong public-information campaigns against youth drug use (Leland, 1995, p. 74).⁴ Although such concerns existed, children were being prescribed such medication in increasing numbers: according to a 1996 estimate, 1.5 million children—two-and-a-half times more than six years earlier—were taking Ritalin, the leading brand of stimulant medication (Kolata, 1996). In fact, the prevailing belief among doctors, teachers, and parent support groups was that the disorder was valid, and treatment with prescribed medication the best available approach.

According to supporters of the diagnosis, recognition of the existence of the disability was an important educational and health reform that promised to rehabilitate those who, in the past, had been written off as beyond help (Gladwell, 1999). From this perspective, ADD brought hope to the formerly hopeless, whose handicap carried high risk for later onset of more significant social liabilities, such as drug addiction and criminality. The writer of a 1996 article in *Newsweek* took this view:

These are the children who can't wait their turn. They blurt out answers before questions are asked. They can't stop wiggling their legs, tapping their pencils. They lose their bookbags, their homework, their tempers . . . not sometimes, but *constantly*. Decades ago "these children were the outcasts, the losers, the zoned-out kids" (Hancock, 1996).

In the final line of this passage, a child-behavior specialist explained why the disorder had not been recognized earlier. His hypothesis was that a certain percentage of the population always had shown the traits associated with ADD but that teachers and administrators formerly turned a cold shoulder to their condition. Experts continued to warn that those who remained untreated ran the risk of more serious sociopathologies.⁵ Scientific resources increasingly were devoted to the disorder, especially to understanding links with more worrisome behavioral problems, such as oppositional defiant disorder, conduct disorder, and

3. "The medication works like a pair of eyeglasses, helping the individual to focus" (Hallowell & Ratey, 1994, p. 20).

4. The two phenomena converged on 10 February 1997, in a CNN special report on Ritalin abuse following a teenager's death from an overdose of the stimulant.

5. The relation between ADD and conduct and oppositional defiant disorders remained a subject of some disagreement among child psychiatrists. For a review, see Hinshaw & Anderson, 1996.

antisocial personality disorder (American Psychiatric Association, 1994).⁶ In 1995, the National Institute of Mental Health (NIMH) made ADD the target of a significant multisite treatment research grant (Richters et al, 1995).⁷ In the mid 1990s, investigators began to report that the disorder did not necessarily go away upon adolescence, as it previously had been thought. Experts advised adults who showed symptoms to seek professional help and social support (Wender, 1995). The integration of ADD into such discourses of self-help, however, should be distinguished from the application of the category to children, for whom the diagnosis was not a matter of self-recognition but was normally initiated by parents or teachers.

Acceptance of the diagnosis was widespread enough that courts began to require school districts to provide special education for ADD children or else pay for the children to attend one of the schools focused around the disorder. This considerable cost brought on criticism of the diagnostic validity of the disorder by fiscal conservatives (Vatz, 1994). Such controversy was due to the sudden visibility of the disorder and to the much higher rate of diagnosis in North America compared with other industrialized countries.⁸ Pundits asked whether the American rate of diagnosis bore witness to the peculiarity of the nation's children or to that of social and medical systems in the United States (Gladwell, 1999). Even if the science of developmental psychopathology was more advanced in the United States, by the late 1990s the nature of the disorder still had not been established definitively, despite recent advances from the neurosciences.⁹

The internal history of the diagnosis, recounted in both popular and professional texts, does not account for the apparent surge in the prevalence. This history tells of maverick discovery, followed by gradual recognition. The story begins with the event traced above; around the turn of the century, George Still proposed that a number of children he had seen whose behavior was difficult to control might be suffering from organic brain damage, and further that such damage led to a crippling of the children's moral development (Barkley, 1990, p. 5). This observation was ignored until the contours of the disorder again were glimpsed, after an outbreak of St. Louis encephalitis between the world wars. Doctors noted that a number of the victims, although they seemed to have recovered from the disease, continued to have trouble restraining their motor impulses. Researchers hypothesized that the children's problems with impulse control had to do with encephalitic brain lesions in the region of the brain that governed volition (Ebaugh, 1923; Levy, 1959). In the late 1940s, the psychiatrist Alfred Strauss extended this lesion theory to explain a number of non-encephalitic patients with related motor-impulse problems. Even if the patients' organic lesions were not apparent, he reasoned, the cause of similar dysfunction should be due to similar organic conditions. Proposing that extremely overactive children be given such a diagnosis, Strauss coined the disorder "minimal brain damage" (Strauss & Lehtinen, 1947).

This remained a relatively obscure diagnosis for some time. In the 1957 edition of Leo

6. These were the DSM-IV categories for a progression of disorders potentially related to violence and criminality.

7. This research direction was promoted by a report of the Institute of Medicine, 1990.

8. Prominent child psychiatry researchers attributed this difference to variations in diagnostic criteria, a problem that will be addressed below: "Across multiple revisions of the official DSM and ICD [International Classification of Diseases] criteria, differences in the symptom lists and decision rules for diagnosing ADHD and HKD [hyperkinetic disorder] have produced confusing, cross-national differences in administrative prevalence, despite lack of differences in epidemiological prevalence when similar operational definitions are used for diagnosis" (Swanson, Castellanos, Murias, Lohoste, & Kennedy, 1998).

9. NIMH investigators reported that they had confirmed the hypothesis of a "dysfunction of right sided pre-frontal striated systems in ADHD" with MRI. See Castellanos et al., 1996.

Kanner's standard text, *Child Psychiatry*, there was no mention of it. It did appear by the next edition, published in 1972, tucked away as the final entry under "organic behavior disorders" (Kanner, 1972). Kanner's definition replaced the term "damage" with "dysfunction," for by that time there was some doubt over Strauss' speculative organic etiology: The number of children being diagnosed with MBD seemed to exceed those likely to have actual damage. As early as 1960, psychiatrist Stella Chess had argued that there was no necessary connection between the observed behavior—hyperactivity—and brain damage (Chess, 1960). In the 1968 edition of the American Psychiatric Association's Diagnostic and Statistical Manual (DSM-II), the question of the etiology of the syndrome was sidestepped by naming it according to its most visible property, excessive movement. Hyperkinesis, the immediate precursor to ADD, was "characterized by overactivity, restlessness, distractibility, and short attention span" (American Psychiatric Association, 1968, p. 50). It occurred mostly in boys and tended to disappear by adolescence.

Hyperkinesis could be treated with a stimulant-based medication that for unknown reasons had calming effects—a phenomenon that had first been observed in 1937 (Bradley, 1937; Levy, 1959). Charles Bradley, who experimented in the 1930s with benzedrine in a Rhode Island home for children with behavioral problems, speculated that underactive centers of inhibition in the brain were stimulated by the drug, which "reduced activity through increased voluntary control." He reflected on the impressive effects of the drug in the educational setting:

To see a single dose of benzedrine produce a greater improvement in school performance than the combined efforts of a capable staff working in a most favorable setting, would have been all but demoralizing to the teachers, had not the improvement been so gratifying from a practical viewpoint (Bradley, 1937, p. 582).

By the 1960s, knowledge of these effects had spread, and the most commonly prescribed stimulant medication was methylphenidate (trade name Ritalin), developed by the Swiss Ciba corporation. Still, the actual cause of the problem remained a mystery. In 1977, a British study in the Isle of Wight showed no correlation between hyperactivity and the presence of brain lesions (Rutter, 1977). As environmental pollution and nutrition became subjects of social concern, new theories emerged to explain the rise in the number of hyperactive children. Some blamed food additives, excess sugar, or allergies for the seeming rise in behavioral problems (Feingold, 1975). Psychodynamic theorists analyzed ego processes to explain the condition, seeking the source of maladjustment in early childhood social relations (Leventhal, 1968).¹⁰ Meanwhile, estimates of the prevalence of the disorder still varied widely. Yet these professional controversies subsided within a period of only a few years; behavioral tests were standardized and competing models swept away as the discipline underwent a sea change that enabled the construction of a more unified view of the disorder. After the third edition of the Diagnostic and Statistical Manual (American Psychiatric Association, 1980) renamed the disorder Attention Deficit Disorder, a research program coalesced that focused not only on treatment strategies, but also searched for evidence of genetic predisposition and attempted to locate a site of neural causality.

10. A reviewer of an important 1966 book on learning disorders summarized this position: "The editors are convinced that most failures to acquire knowledge in school, associated with anti-social behavior, psychosomatic disorders, and psychotic disorders, may be viewed as failures in the learning processes with parents or parent figures early in life. These early parental experiences result in aversions to learning which at times make this process impossible" (Fried, 1968, p. 562).

FROM THE CASE STUDY OF THE POPULATION

Given the unsettled status of the syndrome, “discovery” is not the appropriate term to describe the emergence of attention deficit. Rather, to understand the vitality of the disorder in the decade of the 1990s, one must look at how shifting notions of individual rights, social risk, and personality became entangled in an ecology of knowledge in which the disorder could thrive. A brief history of the profession will help to map the terrain, for the strengthening of the disorder coincided with the rejuvenation of child psychiatry as a discipline. The specialty of child psychiatry first emerged from a combination of groups that had, beginning at the end of the nineteenth century, organized around the problem of social adjustment. Progressive reformers codified the distinction between childhood and adulthood and urged governments to take a protective interest in the moral development of future citizens. During the 1920s, the mental-hygiene and child-guidance movements came together in philanthropically sponsored clinics in which social workers, teachers, and doctors could consult together in matters of juvenile health (Horn, 1989).

The pathologies of mental development became a subject of scrutiny in the 1930s at Johns Hopkins, where Leo Kanner was one of the first academic child psychiatrists. Kanner brought to the subfield the dynamic approach to personality espoused by his mentor, Adolph Meyer, whose theoretical perspective would remain dominant in the discipline through the 1970s. Meyer’s technique of life-history analysis emphasized the careful measurement of whole persons in the understanding of mental disorder (Leys, 1991). Kanner defined his mentor’s “dynamic attitude” as a biographic exploration in which origins of present troubles should be sought in happenings and experiences of the past; mental illness was to be viewed as the culmination of personal difficulties. While Meyer was suspicious of an emphasis on infantile sexuality and repression in the etiology of psychopathology, he shared with psychoanalysts a belief in the central role of life history in the explanation of mental illness. For this reason, research stemming from the Meyerian tradition centered on the case study.

In his 1957 guide to the practice of child psychiatry, Kanner derided the old, outmoded approach to diagnosis, which he presumed had been superseded by Meyer’s life-history technique. In the unenlightened earlier days of the field, he wrote, “who the patient was seemed less important than what the patient had. For this reason, the psychiatry of that day has been referred to as static, descriptive, nosographic (disease-describing) psychiatry” (Kanner, 1957, p. 7). Descriptive, nosographic: these are codewords for the diagnostic system originally espoused by turn-of-the-century German psychiatrist Emil Kraepelin. They are also terms that will come to the fore in the discussion of contemporary psychiatry below; moribund though it may have been at the time of Kanner’s writing, this marginalized style would return with a vengeance two decades later.

The resurgence of descriptive nosology in child psychiatry, which was coemergent with the rise of ADD, can be traced in professional journal articles, the concrete form in which scholarly research is presented. In 1959, when the various stands of child psychiatry merged into a group powerful enough to gain professional recognition, psychiatry as a whole was under the domination of neo-Freudian models. Thus, the first years of the *Journal of the American Academy of Child Psychiatry*, founded in 1962, had a distinctly analytic flavor. In discussing behavioral problems ranging from nail biting to suicide, the journal’s writers most often looked to the experiences of early childhood for etiological clues. The articles tended to present case studies rather than the statistical analyses of groups of patients that later would appear. Overprotective mothers, those reticent weaners responsible for a variety of cold war pathologies, were especially prominent in the journal.

This period, which lasted until the mid 1970s, is striking mainly for its utter difference from the way the journal appeared in the 1990s. The transformation reflected the decline of psychodynamic psychiatry during the interim period (Hale, 1995; Luhrmann, in press; Shorter, 1997). Discussions of analytic techniques, professional certification, and ego processes were banished in favor of multiple authors' presentations of statistics, charts, and longitudinal studies. Rather than citing hallowed founding theorists, the references guided readers to contemporary, related statistical studies. This new appearance indicated the changing disciplinary alliances of child psychiatry; rather than relating to education and social welfare, the leaders preferred to affiliate themselves with more scientifically prestigious fields such as neuropsychology, biology, and epidemiology.

In child-psychiatry research, the fate of dynamic models was sealed by 1976 when Melvin Lewis took over editorship of the *Journal of the American Academy of Child Psychiatry*. From its inception, the journal had been run by dynamic theorist Eveleen Rexford. Lewis explicitly opened the journal to the biological sciences and demanded programs of research based on standard definitions that would be replicable according to scientific methods. The form of articles shifted, indicating a transformation in what constituted reasonable medical knowledge in the field. Most concretely, the new form dictated the end of the individual case study. Scientific validity required testing on clearly defined populations made up of "normals"—right-handed males of at least average intelligence—and including a control group. The first article under the new editorship, "Genetic Factors in the Hyperkinetic Syndrome," signalled the new direction of the discipline (Cantwell, 1976).¹¹

TEMPERAMENT AND THE MODERN SYNTHESIS

The underlying spirit of the new approach appeared in the research program of the assistant editor of the journal under Lewis, Stella Chess. She and her husband, Thomas Alexander, had been heralds of the somatic turn in child-development theory, publishing since the late 1950s the results of longitudinal studies indicating that certain "behavioral styles" were continuous from infancy through childhood and were little affected by environmental factors. Such claims were a direct attack on dominant models that traced the origins of mental pathology to early mother–infant interactions. Thomas and Chess's position was directed explicitly to absolve parents of the guilt over children's personality problems that was fostered by dynamic models. In fact, by pointing out the existence of inborn "behavioral styles," they sought to improve parent–child relations; parents would have to learn to adjust their expectations to the inherent characteristics of their child. There would be no one best method for dealing with the arduous chores of child-rearing: weaning, toilet training, discipline, and socialization (Thomas, Chess, & Birch, 1968).¹²

Thomas and Chess focused on the child's ability to adapt to its social environment, an interest based on the emergence of the "modern evolutionary synthesis" in postwar biology and animal behavior. During the time that neo-Freudian ego psychology reigned in the study of human personality, ethologists had integrated natural selection with population genetics to forge a powerful engine for the interpretation of animal behavior. According to the modern synthesis, humans were characterized genetically by their adaptive flexibility. For a disen-

11. For the historical background to the approach based on clinical research, see Healy, 1997.

12. By citing the importance of temperament in the conceptualization of ADD, I do not mean to imply that this was a neo-Kraepelinian perspective per se. Rather, I see Thomas' and Chess's theories as complementary to the goals of the neo-Kraepelinians to describe psychiatric disorders phenomenologically and to ground them organically.

chanted minority of researchers in psychiatry, this work inspired a rebel program in psychiatry to supplant Freud with Darwin. Dobzhansky's genetics provided them with a bridge between animal behavior and human psychology.¹³ In 1988, Chess reflected on this history:

Our own studies of temperamental individuality and its significance for the child's development, initiated in 1956, were perhaps the first systematic applications in child psychiatry of Dobzhansky's (1966) formulation that 'what is biologically inherited are . . . the ways in which the body reacts to the environment' (Chess, 1988).¹⁴

The role of ethology in shaping the new child psychiatry is intriguing, for part of the mystery of small children lies in their animal-like inscrutability. We can begin to see ourselves in the curious figure of the infant but cannot quite grasp what it is to be in that position for the infant does not yet see him- or herself as an analyzable object. We rely on behavioral clues to imagine what the infant will become—inquisitive or stubborn, shy or social, demanding or playful—and we tell stories of the emergence of these traits. Historians have accustomed us to the notion that such narratives are transient, that there are tales to be told about the emergence of the stories themselves; we may thus locate the innocent child's origin in the emergence of bourgeois culture or look for the birth of the libidinal child at the end of the nineteenth century (Aries, 1962; Foucault, 1978; Stephens, 1995). In America, the malleable self of progressive reform cohered with both behaviorist and psychodynamic models of character development (Burnham, 1988). Thomas and Chess's early work on child behavior foreshadowed the decline of both of these models.

For Thomas and Chess, the implications of Dobzhansky's findings were that psychopathologies should be treated as maladaptive behaviors—which, they theorized, emerged from a mismatch of a child's inborn behavioral style with the parents' expectations. Borrowing from premodern humoral theory, they named the child's genetic endowment his or her "temperament" and formulated nine fundamental categories of behavior with which to measure it. Their categories, drawn from observation and inference, were based on physical description and held clear implications for the child's socialization process: activity level, rhythmicity, adaptability, approachability to or withdrawal from new stimuli, intensity of reaction of response, threshold of responsiveness, quality of mood, distractibility, and attention span/persistence (Thomas et al., 1968).¹⁵ After being scored on scales from high to low, the children were divided into groups of "easy," "difficult," and "slow to warm up." These qualities indicated the child's behavior in the face of new stimuli—his or her capacity to adapt.

Neither Thomas' first book detailing these results, published in 1963, nor his text coauthored with Chess and Herbert Birch in 1968, had much of an impact upon publication. Perhaps their audience—child psychiatrists and psychologists—was not yet ready for them. But their 1977 publication, which did not differ much from the earlier versions, created a sensation and became a classic among theorists of child behavior (Kagan, 1994, p. 30; Thomas & Chess, 1977). These temperament studies were the forerunner to later claims, popularly dis-

13. As Donna Haraway (1989, p. 213) writes, "With 'group integration' as the core scientific knowledge grounding the all-important 'sharing way of life,' 'behavior' would link the adaptationist physical anthropology and therapeutic medical psychiatry into a common research program and public discourse on modern crises."

14. Perhaps to leave room for the heritability of temperament, Chess edited Dobzhansky's actual words, which were: "What is biologically inherited are not body parts, not even traits, but the ways in which the body reacts to the environment" (Dobzhansky, 1966, p. 14).

15. Jerome Kagan (1994) criticized their quantitative categories as assuming that opposing traits constitute more or less of the same thing. He proposed qualitative categories as an alternative to "dimensions."

seminated, that the genetic basis of such traits as thrill-seeking and excessive worry had been found. Thomas and Chess's ideas were extended further in the work of evolutionary psychologists such as Robert Wright (1994).¹⁶

The advancement of the idea that inherited temperament should substitute for acquired personality in psychiatric epistemology was aided by the development of improved scales for evaluating and monitoring pathological behavior. These were questionnaires given to parents and teachers asking them to evaluate various aspects of a child's everyday behavior. An example is Thomas Achenbach's Child Behavior Checklist, which was influential in codifying the dimension "internalizing/externalizing" (Achenbach & Edelbrock, 1983). This category, related to Thomas and Chess's approachability/withdrawal trait, concerned the brain's ability to inhibit bodily impulses. George Still's notion of "inhibitory volition" might have found a place in this dimension. Psychiatrists related internalizing behavior, which indicated too much inhibition, to anxiety disorders such as obsession-compulsion. One instance of internalizing behavior, extreme shyness, was interpreted as an inherited temperamental trait by psychologist Jerome Kagan in his own longitudinal studies that began in the early 1960s.¹⁷ Achenbach's "externalizing" disorders, on the other hand, referred to the inability to inhibit one's impulses and included Tourette's syndrome, attention deficit disorder, and conduct disorder (Hinshaw, 1987).¹⁸ While 30 years earlier, the heritability of such traits would have been anathema to most child psychiatrists, by the 1990s it was nearly taken for granted in professional discourse.

THE ECOLOGY OF PSYCHIATRIC KNOWLEDGE

What happened between 1968 and 1977 to create an intellectual milieu ready to accept the notion of inherited temperament? It was not the force of the observations alone, stiking though they may have been; otherwise, presumably, Thomas and Chess's work would have been accepted earlier. For professionals to give up their hard-earned assumptions, experimental data alone is not enough. During the 1960s, dynamic personality theorists were secure intellectually and professionally in their positions, but by the mid 1970s a set of crises had created the need for a new theory, one that would work in the changing environment of health care. For one, the site of the child psychiatrist was shifting; the Urban Community Mental Health Centers, founded in the reformist, public spirit of the early 1960s, were no longer welcome homes for these professionals. Predominantly white and upper middle class, they were shut out from the empowerment movements that shifted the demographic landscape of inner-city social welfare agencies (Rafferty, 1975). The 1970s were also a time of tightening health budgets, when lengthy psychoanalytic treatments came under fire as wasteful and unproven; treatments, to be approved by third-party payers, would have to involve medical therapies that could be shown in clinical trials to be efficient and effective (Enzer, Philips & Cohen, 1984; Hale, 1995; Healy, 1997; Starr, 1982).

Meanwhile, the child-psychiatry profession suffered from low prestige in the medical world, and declining residency applications signalled a lack of professional promise (Silver, 1980). Psychiatry as a whole had trouble pointing to a record of progress in the understanding

16. Following in this tradition, Frank Sulloway (1996) wrote: "The topic of temperament is especially important in the context of why siblings are so different. Depending on differences in family niches, individual disparities in temperament have highly varied consequences for the rest of personality."

17. Kagan writes in *Galen's Prophecy* (1994) that he at first leaned to an interpretive melange of behaviorism and neo-Freudianism, but converted during the seventies to something approaching Thomas and Chess' position.

18. Chess and Thomas actually protest the ADD diagnosis, seeing it as an agglomeration of related temperamental traits. See their forward to Carey & McDevitt, 1995.

and treatment of mental disorders. The emergence of effective medication treatments threatened to narrow the scope of the discipline, practitioners of which already had ceded areas under their authority to the advances of biological medicine—in the case of syphilis early on, for example. When previously psychogenically conceived disorders like schizophrenia and manic depression showed positive responses to pharmacological treatment beginning in the 1950s, it became clear that to maintain legitimacy, psychiatry would have to take somatic models seriously. In fact, some members of the profession saw this development as an opportunity to reshape the discipline.

It is helpful to view the professional transformation of the 1970s not as the result of scientific or technological breakthroughs, but as a kind of adaptive strategy. Although the logic of organic causality had been substantiated by the effect of psychotropic drugs on psychotic patients since the introduction of psychopharmaceuticals in the early 1950s, this event in itself did not explain the transition. Dynamic therapists had been prescribing antidepressants, tranquilizers, and sedatives for over a decade without altering their overall model. Drugs were assimilable into a psychodynamic model; they could be understood as treating the symptoms rather than the underlying cause of pathology, whose true contours would emerge only through the self-knowledge acquired in the psychotherapeutic encounter. The introduction of psychopharmaceuticals did not need to spell the doom of talk therapy, as the case of France, the birthplace of the antipsychotic, demonstrates; there, Freudianism—albeit a very different version than that of the United States—had a first wave of popularity just as it was declining in North America.¹⁹ As much as drug efficacy shifted the locus of causality in mental disorder from childhood to the brain, so too did the importance of having organic explanations transform the interpretation of medication from palliative to therapeutic.

The American turn from the dynamic psyche to an organically grounded descriptive nosology was crystallized in 1980 with the publication and widespread dissemination of the third edition of the Diagnostic and Statistical Manual, DSM-III (American Psychiatric Association, 1980). The shift had been assured at least six years earlier when disaffected psychoanalyst Robert Spitzer was named to head the American Psychiatry Association DSM steering committee and was given free rein to determine the committee membership. With this move, the APA indicated that dynamic psychiatry was being superseded. Spitzer, influenced by the Washington University neo-Kraepelinians, sought like-minded theorists who would return diagnosis to the purely descriptive, objective categories Kanner had disparaged. Although this group professed to suspend the question of etiology, their work was premised on the belief that organic correlates eventually would be found to correspond with well-defined categories (Healy, 1997; Kirk & Kutchins, 1992; Wilson, 1993; Young, 1995).

For dynamic psychiatry, whether inspired by Freud or by Adolf Meyer, knowledge had been accrued and diagnoses proffered through example and analogy. The expert's charismatic authority relied on his or her exegetical prowess and technical skill in psychotherapy. Now the psychiatrist, using Spitzer's manual, was to be a measurer rather than an interpreter. Such a programmatic change obviously would affect the everyday practice of psychiatrists. But more importantly, the new manual had the potential to improve the status of the field as a science with respect to other medical disciplines. The movement toward making psychiatry a biomedical science was indicated by the increasing centrality of clinical research, which in turn required the establishment of agreed-upon diagnostic criteria and therapeutic protocols. By creating such standards, DSM-III provided a frame for psychiatric research that abandoned

19. For a sociological analysis of the French fascination with Jacques Lacan in the 1970s, see Turkle, 1978.

the case study in favor of the population forged via diagnostic classifications.²⁰ Unlike the individual case, the population had no particular history, no tale of relations with parents or rejections at school—it simply had a set of answers to given questions, and these answers placed patients together in a diagnostic category. With this standardization, the individual patient was integrated into a system of manipulable statistics consisting of correlatable factors, the combined presence of which weighed heavily on any psychiatrist unwilling to abide by the new categories.

DSM-III was to be a kind of diagnostic machine, the reliable performance of which would liberate psychiatry from the idiosyncracies of subjective judgment. As such, the manual was premised upon professional agreement about what was to be measured. Journals such as the *American Journal of Psychiatry* and the *Journal of the American Academy of Child Psychiatry* were sites for forging this consensus where new mental disorders could come to fruition. For the manual to work properly, everyone who measured such things would have to use it. As a result, the process of honing the categories of disorder was fraught with professional struggle (Healy, 1997; Young, 1995).

Though the epistemology of the new psychiatry was positivistic—disorders were out there in the world to be found—the categories were honed according to pragmatic principles; the pathological could best be defined by the dysfunctional. Scales were refined to measure the norm of functionality, which was characterized as “adaptiveness.” An interesting word—not only did it bring psychiatry into the realm of biology, but also it indicated that the brain worked in a directed way to bring the organism into harmony with its environment. Another popular term for this was “coping.” The inability to cope with what seemed to be reasonable expectations could then define pathology.

Mostly, questionnaires provided the clues as to the patient’s particular category of pathology. Once sanctioned, these devices allowed the act of diagnosis to be a medical act, and therefore to lead to a specific treatment protocol. If the act were legitimately medical, the person then would become a patient, her or his identity transformed. The patient could now be prescribed medications or undergo a therapeutic regimen designed to modify her or his behavior. This medical act separated the patient from her/his pathology, and what might have been an inauthentic, artificial personality change became a technical adjustment, a correction—a restoration rather than a transformation of self. Meanwhile, having gone from interpreter of the past to compiler of questionnaires, the reborn psychiatrist was shorn of hermeneutic responsibilities.

THE GENESIS OF A PSYCHIATRIC FACT

The DSM-III Child Disorders Committee appointed by Spitzer included several proponents of biogenic models. Many of these experts had written about hyperactivity, which had not been a major focus of dynamic theorists’ work. Stella Chess, in an important 1960 article, had sought to remove blame from parents for hyperactivity, framing the behavior as a temperamental condition, while at the same time distinguishing it from brain damage (Chess, 1960). Also included on the panel were hyperactivity expert Paul Wender, who later wrote the

20. In his important history of the pharmacological era in psychiatry, David Healy (1997, p. 237) writes, “The new system, DSM-III, also appealed to the pharmaceutical industry and to the FDA, because it introduced much greater clarity into the regulatory process and indeed meant that studies which had been conducted anywhere in the world that adhered to DSM-III criteria could be used as part of a regulatory submission.” For the perspective of a child psychiatry researcher on this process, see Castellanos, 1997.

first major professional text on attention deficit disorder in adults (Wender, 1995), and Dennis Cantwell of UCLA, who had edited an important volume on hyperactivity (Cantwell, 1975). Hyperactivity was emerging as a key disorder in child psychiatry, and the presence of such figures signalled that the DSM-II category of “hyperkinesis” would be subject to scrutiny.

Given the number of members who were invested in the problem, it is intriguing that the committee emerged in 1980 with a new name for hyperactivity. The disorder was rechristened attention deficit disorder, based on the work of McGill psychologist Virginia Douglas. From observation and experiments with children who had been diagnosed with DSM-II hyperkinesis, Douglas had shown some years earlier that motor problems were not, in fact, the salient characteristic of their disorder (Douglas, 1972). After administering batteries of questionnaires, observations, and machine-aided tests, she found that the children’s main problem was their inability to “stop, look, and listen.” They were unable to pay attention to a specific task for prolonged periods. Douglas was explicit that this incapacity to inhibit distracting impulses was not merely an excess of movement but should be considered a disorder of will.

In her seminal paper introducing the notion of the attention deficit, Douglas cited William James as an authoritative source for rejoining the faculties of attention and volition. Her reference was similar to the passage George Still had cited seventy years before: “The essential achievement of will is to attend to a difficult object and hold it fast before the mind,” James had written in *The Principles of Psychology* (Douglas, 1972; James, 1890). This, to Douglas, named the central problem she was seeing in “hyperkinetic” children. Thus Douglas had abstracted the diagnosis, moving it to a less palpable, but more fundamental explanation. At this point, the official history of the disorder emerged, legitimating it as an actual entity in the world; William James provided the philosophical foundation and George Still the clinical history.

The DSM Committee accepted Douglas’ redefinition and so expanded the purview of the disorder, for a child could be diagnosed with attention deficit disorder without being hyperactive at all. The essential features, according to DSM-III, were “signs of developmentally inappropriate inattention and impulsivity,” and possibly hyperactivity. In the description, the key future terrain of the disorder was highlighted: “In the classroom, attentional difficulties and impulsivity are evidenced by the child’s not staying with tasks and having difficulty organizing and completing work” (American Psychiatric Association, 1980, p. 41). As school was the site where the disorder appeared, teachers—in collaboration with parents and school psychologists—became the first line of defense. The somaticization of psychiatry in the wake of DSM-III did not involve necessarily the conversion of previously psychogenically conceived disorders to brain-based etiologies (as with schizophrenia and depression). Rather, ADD shows a different path—the expansion of an already-organic malady (hyperkinesis or minimal brain dysfunction) to cover a broader variety of cases.

In DSM-III, the criteria for each subcategory of attention deficit disorder—inattention, impulsivity, and hyperactivity—were listed separately. In order to diagnose the condition, the manual required evaluators to give affirmative answers on two or three of the possible symptoms under each subcategory. New scales emerged that were designed for the evaluation of children by either parents or teachers. A key element in standardizing evaluations was the acceptance of the Connors Scale, the most important of a series of scales for rating observed behavior. This scale first had been published as a guide for clinical drug research, and only later did it become part of the general diagnostic apparatus (Connors, 1969). After Connors and Douglas’ reports, which made it possible to confirm quantitatively the efficacy of stim-

ulant medication, there was a rapid increase in the use of medication for hyperactivity, which soon inspired a critique of drug treatment as social control from the perspective of the anti-psychiatry movement (Schrag & Divoky, 1975).

Meanwhile, technological improvements in detecting hyperactivity also had been made. Whereas, during the 1960s, researchers had pasted masking tape on the floor and counted the number of times children crossed over it in a given play period, now they were equipped with electronic devices such as the “pedometer,” a shoulder attachment that could measure movement quantitatively (Douglas, 1972; Hinshaw, 1997). There were other important new devices, such as the Continuous-Performance test, a primitive sort of video game that measured the capacity to sustain attention in the face of the most boring of tasks, and Jerome Kagan’s Matching Familiar Figures Test, which calculated the cognitive ability to resist tempting impulses.

According to DSM-III, a child could be diagnosed with ADD and prescribed medication if, for instance, he or she fulfilled the following criteria: *Inattention*: often fails to finish things he or she starts, easily distracted, has difficulty concentrating on schoolwork or other tasks requiring sustained attention; *Impulsivity*: often acts before thinking, has difficulty organizing work, frequently calls out in class; *Hyperactivity*: has difficulty sitting still or fidgets excessively, is always “on the go” or acts as if “driven by a motor.” These traits had to have begun by age seven and to have lasted for at least six months.²¹

Growing interest in the disorder among researchers after the publication of DSM-III can be charted in terms of the relative number of articles published on it in the Academy journal. Between 1971 and 1977, when symbolically charged inhibitory disorders like enuresis and encopresis (involuntary urination and defecation, respectively) were more popular, a total of three articles on hyperactivity appeared. A crescendo of interest began in 1980, and after 1984, when an entire issue was devoted to attention deficit disorder, from 10 to 20 articles on it appeared each year in the pages of the journal. Clinical practice at the same time became ever more focused around the disorder, which by 1996 accounted for at least 40% of child-psychiatry references (Castellanos et al, 1996). From the wildly varying estimated prevalence rates of two decades before, a consensus emerged holding the figure to be between 4 and 8% of the school-age population. The score on impulsivity scales enough above the mean to count as ADD-positive was a product of professional agreement rather than objective criteria. One member of the Child Disorders Committee of the American Psychiatric Association reported that the figure of 5% was agreed upon because it was seen as the maximum number of attention-disordered children schools would be able or willing to treat (Elliott, 1997).²²

Although it had been declared “in crisis” less than ten years earlier (Silver, 1980), child psychiatry was apparently in good shape by the late 1980s. In an address to the Child Psychiatry Association’s annual meeting, Stella Chess (1988) announced that the discipline had at last “come of age.” As with the rest of psychiatry, the field’s new vitality was linked to practitioners’ conversion to a medically viable model of diagnosis and treatment. In 1990, the NIMH mounted a campaign targeted at the more common of child disorders aimed especially at those that indicated risk for future antisocial behavior (National Advisory Mental

21. This is a hypothetical case rather than a comprehensive list of the DSM criteria. See American Psychiatric Association (1980), 41-43.

22. That a disorder is located at an extreme along a continuum is not unique to psychiatric pathologies such as ADD. Hypertension and hypoglycemia might be seen as comparable examples. Philosopher of science Georges Canguilhem argued that norms of health should not be defined quantitatively, but rather in terms of the individual’s adaptive, or norm-making capacity. See Canguilhem, 1989.

Health Council, 1990). ADD specialists benefitted from this research effort, as the disorder, as noted above, was linked to risk for later, more serious social pathologies (American Psychiatric Association, 1980, p. 48; Barkley, 1990, p. 83–84; Castellanos, 1997, p. 382).

Along with the emergence of reliable diagnostic populations under DSM-III, communities formed that consisted of people whose alliance was based upon a common mental disorder. Such groupings produced a kind of social identity representing a new form of governmentality—an emergent technique of regulation through self-definition.²³ The movement from diagnosis to self-identity worked through a new kind of interpretation to be performed by the patient rather than the psychiatrist. Previously incoherent signs took on biological meaning retrospectively when the correct diagnosis was made. In a review article written for pediatricians, an ADD specialist described his treatment of a 15-year-old female whose academic and school failure had led to depression and anxiety, which at first were treated unsuccessfully with psychotherapy and antidepressants:

Detecting the subtle but clear evidence of ADHD in her past and present functioning allowed her to benefit pharmacologically from the addition of a stimulant to her anti-depressant, and psychotherapeutically as a result of increased self-awareness. It also resulted in the discovery that the adolescent patient's mother had experienced similar symptoms for her entire life, for which she had coped as an adult by repeatedly refusing job promotions (Castellanos, 1997, p. 383).

Groups of patients and their families demanded legislative recognition of their rights and lobbied for research funding in order to codify the existence of the disorder with which they were identified. Along with attention deficit disorder, mental disorders that have spurred such mobilization include Alzheimer's disease, multiple personality disorder, posttraumatic stress disorder, and chronic fatigue syndrome (Fox, 1989; Hacking, 1995; Showalter, 1997; Young, 1995). One activity of the ADD support network, the work of which was publicized via mailings, websites, and meetings in hospitals and community centers, was to police media coverage, making sure that the disorder was cast in a sympathetic light.²⁴

The fight for legal recognition of the disorder as a disability was an important rallying point for the ADD community. In 1975, the U.S. Congress passed legislation, the Education for All Handicapped Children Act, which required states to provide "free and appropriate public education" to children with disabilities (Beitchman & Young, 1997). The 1997 Individuals with Disabilities in Education Act (IDEA) expanded this legislation to ensure that schools provide adequate facilities for students with a set of academic dysfunctions, prominently dyslexia and other developmental cognitive deficits. Parent-based ADD-support groups, with support from child psychiatry experts and the pharmaceutical industry, campaigned for the inclusion of ADD children in these regulations. Given that the population of ADD-diagnosed children had increased to as much as six percent of the school population (Swanson, Posner, et al. 1998), some districts were hesitant to comply.

In March 1999, ADD support-group leaders announced a triumph; under new IDEA

23. See Nikolas Rose's (1996) analysis of risk-based community under the political rationality of advanced liberalism. After the decline of the welfare state, he writes, "individuals are to be governed through their freedom, but neither as the isolated atoms of classical political economy, nor as citizens of society, but as members of heterogenous communities of allegiance, as 'community' emerges as a new way of conceptualizing and administering moral relations amongst persons." In his work on the repercussions of the human genome project, Paul Rabinow (1996) has located the emergence of communities of identity based on shared genetic traits, a process which he terms "biosociality."

24. See, for instance, the CHADD (Children and Adults with Attention Deficit Disorder) website: <http://www.chadd.org>.

regulations, ADD was listed under “other health impairments” that schools would be forced to recognize, next to asthma, diabetes, epilepsy, heart conditions, hemophilia, lead poisoning, leukemia, nephritis, rheumatic fever, and sickle cell anemia. The president of CHADD (Children and Adults with Attention Deficit/Hyperactivity Disorder) announced, “The inclusion of AD/HD in the new regulations is a triumph for every child and adult affected by AD/HD. This is a landmark in the history of this disorder because it affirms the right of every AD/HD student to succeed in school” (CHADD, 1999). ADD as an entity thus was legislated officially into existence after a series of disputes between parents and schools over the education of ADD-diagnosed children. A political discourse of rights had secured the place of ADD-identity by linking academic success to biological health. But just as the disorder solidified, the central characteristics of the syndrome threatened to mutate once again.

ADAPTIVE WILL

We have seen the disorder shift from Still’s cases of moral defect to imperceptible brain damage causing hyperactive movement to a temperamental deficit in attention. In the late 1990s, a new body of work reopened the question of what constituted the core deficit of this syndrome. A proposal emerged from the burgeoning field of cognitive neuroscience and gained adherents among many of the more prominent ADD researchers. The author of several authoritative texts on the disorder, Russell Barkley, was vocal in making a case for redefinition in a series of important articles (Barkley, 1997a, 1997b, 1998). His theory combined the results of lesion studies in nonhuman primates, cognitive studies of ADD-diagnosed children, and artificial-intelligence modeling of reasoning processes, all unified by the hypotheses of Jacob Bronowski regarding the evolution of man’s “executive function.” “We are finding,” he began.

that ADHD is not a disorder of attention per se, as had long been assumed. Rather it arises as a developmental failure in the brain circuitry that underlies inhibition and self-control. This loss of self-control in turn impairs other important brain functions crucial for maintaining attention, including the ability to defer immediate rewards for later, greater gain (Barkley, 1998, p. 67).

According to Barkley’s model, “the essential impairment in ADHD is a deficit involving response inhibition.” He and others argued that such a deficit explained the various sorts of problems found in ADD children, ranging from impulsivity to inattention to poor motor control.²⁵ This deficit, Barkley claimed, was the result of a disruption in a set of abilities known as executive functions, which were critical for planning and self-regulation. “These are the functions that provide for the human will and volition, as well as behavior that is characterized as intentional, purposive, future oriented, self-disciplined, and reasoned,” he wrote (1997a, p. 273).

At the organic level, the neural circuit between the evolutionarily advanced prefrontal cortex and the more primitive limbic system was implicated in the dysfunction of this system. The prefrontal cortex was the putative site of a crucial faculty known as working memory, in which subjective time was conceived as the active presence of impending possibilities. This faculty, which simultaneously maintained representations of intentions and programs for

25. As another expert put it, “Today we suspect that the primary problem in ADHD is one of cognition that primarily affects the considered output or behavioral inhibition and, hence, much of intellectual and adaptive function, with hyperactivity but an epiphenomenon of an immature and disorderly brain” (Werry, 1997).

action, was a kind of memory for the future. “It is in working memory that goals and intentions to act are retained and that action plans are formulated and used to guide the performance of the goal-directed responses” (Barkley, 1997a, p. 71).²⁶ Martha Denckla, a child psychiatry researcher at the NIMH, reached similar conclusions:

Research suggests that neither attention nor long-term memory are the critical cognitive correlates of ADHD or LDs [learning disabilities]. Rather, encoding processes, particularly working memory, are identified as deficiencies for children with these conditions. Furthermore, intention and inhibition appear to be particularly impaired in children with ADHD, who exhibit broader deficits in executive function (Denckla, 1994).

If the psychodynamic ego was the product of a life history as it engaged instinct, the neo-Kraepelinian locus of will might be found here, under the rubric of executive function. Child behavior specialists understood this new volitional matrix as both a cognitive process and an anatomical locus for prudent self-management. It was a self-regulatory mechanism that relied on functioning neural circuitry to work properly. The executive function operated to inhibit response to distracting, nonessential phenomena by focusing on longer-term goals to be attained—to be goal-oriented, yet flexible.²⁷ This subcortical system allocated cognitive resources, selecting among the variety of options for action presented by the body, the senses, the environment, and memory, tracking which goals needed to be prioritized, what actions to take in a given moment, which sets of knowledge and memory to access. It was able to evaluate new situations in order to pursue long-term goals. Researchers understood the brain as a self-organizing system, the higher functions of which, located in the frontal lobes, allowed the organism to plan for the future. The dysfunction of this system, a number of child psychiatrists agreed, best explained the deficits that had been found in children diagnosed with ADD.²⁸

What are we to make of this latest expert model of ADD? Given the emphasis of the theory on volition, it is tempting to return to George Still, who also understood his cases in terms of a disorder of will. Can we say that Still glimpsed the future of child psychiatry? Did his children have attention deficit disorder? Since in Still’s time the coalition of actors and technologies necessary for the existence of the disorder had not yet coalesced, it is a badly put question; the existence of the disorder as ADD depends upon the historical process we have traced.²⁹ But to assert that the disorder is contingent is not to dismiss it as false. It is

26. The notion of a neural location for the temporal representation of information to be used in the process of decision making was by no means limited to work in child psychiatry; rather, it was broadly accepted as a research hypothesis in cognitive neuroscience (Goldman-Rakic, 1992). “It seems that the dorsolateral frontal cortex is where past and future meet,” wrote Joaquin Fuster, a pioneer in research on working memory in lesioned monkeys (Fuster, 1995, p. 76). Other areas of neuroscience have formulated similar hypotheses about the planning area of the brain. Neurologist Antonio Damasio (1994, p. 196-198), for example, calls the prefrontal structures that coordinate possibilities for action the “generator of diversity.” Specifically, he refers to attention and working memory as essential components of the reasoning process, “during which possible outcomes are compared, rankings of results are established, and inferences are made.” This line of research provided hypotheses for simulations in artificial intelligence as well: “In mammals, prefrontal cortical circuits seem instrumental in generating goals, in maintaining representations of goal-relevant information, and in selecting these representations as a function of their expected value for the organism. Our simulations illustrate how specialized prefrontal circuits may implement these functions” (Dehaene & Changeux, 1991).

27. An account of ADD in the *New Yorker* adopted the concept of working memory: “An accurate sense of time is a function of a certain kind of memory—an ability to compare the duration of ongoing events with that of past events. . . . Time is about imposing order, about exercising control over one’s perceptions, and that’s something that people with attention deficit disorder have trouble with” (Gladwell, 1999, p. 82-83).

28. “The population most likely to show [executive dysfunction] in our research context was, of course, the one most commonly reported and clinically experienced to show EDF: children carrying the diagnosis of ADHD,” wrote Denckla (1996, p. 6).

29. In both professional and popular discourse, it is important that the disorder be understood as universal, and thus locatable both trans-historically and cross-culturally. See Barkley, 1998.

rather to ask why it took on a particular form and resonance at a given moment.³⁰ The question then becomes: What was this will that was disordered?

For Still and his contemporaries, volition was founded on the capacity to inhibit immoral or unnecessary desires (Smith, 1992). In Still's evolutionary framework, the badly behaving child was not yet properly civilized, was still incapable of the inhibitions necessary for social inclusion. Still was explicit in his understanding of will as a moral faculty linked to social virtue:

Volition, in so far as it is concerned with moral control, may be regarded as inhibitory; it is the overpowering of one stimulus to activity—which in this connexion is activity contrary to the good of all—by another stimulus which we might call the moral idea, the idea of the good of all (Still, 1902, p. 1008).

To late twentieth-century eyes, there was something archaic in Still's formulation of the ties between social virtue and behavioral inhibition. To what extent was the "good of all" at stake in the emergence of the attention deficit? The notion of collective interest is absent in the discourse around ADD. Pressure from society at large in ADD controversies militated against, rather than for, using stimulants to aid children's inhibition. In looking at the production of ADD identity and the growth of legislation around it, one is hard-pressed to call it a form of social control. Police and school authorities did not so much impose the diagnosis as parents and children insisted on the validity of the designation. The expert model based on executive dysfunction outlined above was formed in part to help in this advocacy:

The most vocal proponents of stimulant treatment for ADHD have been the parents of children with ADHD, but their arguments have been weakened by the imputation of self-interest and by the absence of a coherent explanation for the therapeutic utility of psychostimulants and an understanding of their limitations (Castellanos, 1997, p. 382).

Following Elias, by studying cultural forms such as hygiene manuals (or child psychiatry texts), one may witness the stages of the civilizing process as they are reproduced in the life of the individual human being.³¹ If particular modes of impulse inhibition and affect-control cohere with specific social structures, what can be said of the political formation crystallized in ADD? If, at the turn of the century, the good of all was the virtue toward which the volitional inhibition worked, by the 1990s, social norms seemed to have more to do with rational self-management. In the last decades of the twentieth century, the government of children's behavior operated not from the dictates of society in general, but at the level of the nuclear family, according to the family investment in the future of the child.

Sociologist Nikolas Rose has characterized self-making under advanced liberalism as a "new prudentialism," in which individuals were held responsible for management of their own fate through risk analysis of the consequences of present actions—they were required to bring the future into the present, making it calculable and thus manageable (Rose, 1996). Individuals and communities, through the notion of responsibility, became interested in governing themselves. The rationalities of the marketplace began to take over for bureaucracies in regulating the individual, working at a distance, through a logic of consumer choice. Describing the emergence of a set of new disorders and their accompanying therapies, Rose

30. As Ludwik Fleck (1935, p. 35) put it, "in science, just as . . . in life, only that which is true to culture is true to nature." There is a substantial literature in the overlapping fields of medical anthropology and science studies on the relation of social context to disease entity in psychiatry. See, for example, Hacking, 1999; Young, 1995.

31. "Every particular characteristic that we attribute to [civilization]—machinery, . . . forms of state, or whatever else—bears witness to a particular structure of human relations, to a particular social structure, and to the corresponding forms of behavior" (Elias 1994, 47).

might have been giving a list of symptoms of executive dysfunction: “Failures of management of the self, lack of skills of coping with family, with work, with money, with housing, are now all, potentially, criteria for qualification as a psychiatric subject” (Rose, p. 14).

Was there something distinctive about this psychiatric subject as the behavioral sciences moved toward organic explanations of psychopathology? How did it relate to the popularity of organic explanations in the contemporary behavioral sciences? ADD provides a case of the process whereby new forms of government, based on self-management, emerge. In order to make the “psychiatric subject” responsible for its own well-being, its will had to be divided. Illness was located in the brain, in the circuitry making self-organization possible, while the motivation to improve remained a part of the patient’s personhood. This form of organic causality was distinguishable from the degeneracy theory of Still’s day, for in the 1990s the child as moral being, with a future at stake, was dissociated from a malfunctioning (but correctable) organism. “Part of what it takes to succeed in a complex world, in other words, is the ability to inhibit our impulses,” wrote a science journalist, summarizing expertise on ADD and executive function. The child with ADHD, he continued, “cannot apply himself because he cannot regulate his behavior in a consistent manner. He is at the mercy of the temptations and distractions in his immediate environment” (Gladwell, 1999, p. 82–83). It made sense, in this context, to understand Ritalin as a “cognitive aid,” a kind of executive function supplement.

Although Virginia Douglas cited William James in her definitional paper, it would not be correct to say that she was reviving a nineteenth century conception of will. Rather, she was helping to inaugurate a set of diagnostic categories that would facilitate the convergence of neuroscience and psychological measurement. In the new milieu of psychiatric knowledge, ADD thrived as a disorder that proved malleable through medication and which blamed none of the victims: neither parents, children, nor society. There were no pharmacological innovations to instigate this process, nor did neurologists discover an organic pathology; instead, the most relevant new technology was the standardized questionnaire. Behavioral checklists and cognitive tests helped to stabilize the disorder and make it reproducible.³² In doing so, they bound together the various actors—patient and family support groups, physicians, school administrators, managed-care administrators, and pharmaceutical company representatives—whose alliance successfully made temperament and behavior a matter of health.

REFERENCES

- Achenbach, T. M., & Edelbrock, C. S. (1983). *Manual for the child behavior profile and child behavior checklist*. Burlington, VT: Department of Psychiatry, University of Vermont.
- American Psychiatric Association. (1968). *Diagnostic and statistical manual of mental disorders* (2nd ed.). Washington, DC: Author.
- American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.) DSM-III. Washington, DC: Author.
- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.) DSM-IV. Washington, DC: Author.
- Aries, P. (1962). *Centuries of childhood: A social history of family life*. New York: Random House.
- Barkley, R. (Ed.). (1990). *Attention-deficit/hyperactivity disorder: A handbook for diagnosis and treatment*. New York: Guilford Press.
- Barkley, R. (1997a). Attention-deficit/hyperactivity disorder, self-regulation, and time: Toward a more comprehensive theory. *Developmental and Behavioral Pediatrics*, 18, 271–279.
- Barkley, R. (1997b). Behavioral inhibition, sustained attention, and executive functions: Constructing a unifying theory of ADHD. *Psychological Bulletin* 121(1), 65–94.

32. For the analysis of knowledge production as the stabilization of entities, see Latour, 1993.

- Barkley, R. (1998). Attention-deficit hyperactivity disorder. *Scientific American*, 278, 67.
- Beitchman, J. H., & Young, A. R. (1997). Learning disorders with a special emphasis on reading disorders: A review of the past 10 years. *Journal of the American Academy of Child and Adolescent Psychiatry*, 36, 1020–1032.
- Berrios, G. E., & Gili, M. (1995). The will and its disorders. *History of Psychiatry*, 6, 87–104.
- Bradley, C. (1937). The behavior of children receiving benzedrine. *American Journal of Psychiatry*, 94, 577–585.
- Burnham, J. (1988). *Paths into American culture: Psychology, medicine, and morals*. Philadelphia: Temple University Press.
- Canguilhem, G. (1989). *The normal and the pathological*. New York, NY: Zone Books.
- Cantwell, D. (Ed.). (1975). *The hyperactive child: Diagnosis, management, current research*. New York: Halsted.
- Cantwell, D. (1976). Genetic factors in the hyperkinetic syndrome. *Journal of the American Academy of Child Psychiatry*, 15, 214–223.
- Carey, W. B., & McDevitt, S. C. (1995). *Coping with children's temperament: A guide for professionals*. New York: Basic Books.
- Castellanos, F. X. (1997). Toward a pathophysiology of attention-deficit/hyperactivity disorder. *Clinical Pediatrics*, 36, 381–393.
- Castellanos, F. X., Giedd, J. N., Marsh, W. L., Hamburger, S. D., Vaituzis, A. C., Dickstein, D. P., Sarfatti, S. E., Vauss, V. C., Snell, J. W., Lange, N., Kaysen, D., Krain, A. L., Ritchie, G. F., Rajapakse, J. C., & Rapoport, J. L. (1996). Quantitative brain magnetic resonance imaging in attention-deficit hyperactivity disorder. *Archives of General Psychiatry*, 53, 607–616.
- CHADD. (1999). Press release: New regulations “a triumph” for AD/HD.
- Chess, S. (1960). Diagnosis and treatment of the hyperactive child. *New York State Journal of Medicine*, 60, 2379–2385.
- Chess, S. (1988). Child and adolescent psychiatry come of age: A fifty year perspective. *Journal of the American Academy of Child Psychiatry*, 27, 1–7.
- Connors, K. (1969). A teacher rating scale for use in drug studies with children. *American Journal of Psychiatry*, 126, 884–888.
- Damasio, A. (1994). *Descartes' error: Emotion, reason, and the human brain*. New York: Putnam.
- Dehaene, S., & Changeux, J.-P. (1991). The Wisconsin card sorting test: Theoretical analysis and modeling in a neuronal network. *Cerebral Cortex*, 1, 62–79.
- Denckla, M. B. (1994). Measurement of executive function. In G. R. Lyon (Ed.), *Frames of reference for the assessment of learning disabilities*. Baltimore: Paul H. Brookes.
- Denckla, M. B. (1996). Research on executive function in a neurodevelopmental context: Application of clinical measures. *Developmental Neuropsychology*, 12, 5–15.
- Dobzhansky, T. (1966). A geneticist's view of human equality. *The Pharos*, 29, 12–16.
- Douglas, V. I. (1972). Stop, look and listen: The problem of sustained attention and impulse control in hyperactive and normal children. *Canadian Journal of the Behavioral Sciences*, 4, 259–282.
- Ebaugh, F. G. (1923). Neuropsychiatric sequelae of acute epidemic encephalitis in children. *American Journal of Diseases of Children*, 25, 89–97.
- Elias, N. (1994). *The civilizing process*. Cambridge, MA: Basil Blackwell.
- Elliott, G. R. (1997). Personal communication.
- Enzer, N. B., Phillips, I., & Cohen, R. L. (1984). An Overview of the ‘Project Future’ report “Child psychiatry: A plan for the coming decades.” *Journal of the American Academy of Child Psychiatry*, 23, 569–571.
- Feingold, B. (1975). *Why your child is hyperactive*. New York: Random House.
- Fleck, L. (1979). *Genesis and development of a scientific fact*. Chicago: University of Chicago Press. (Original work published in 1935).
- Foucault, M. (1978). *The history of sexuality. Volume I: An introduction*. New York: Random House.
- Fox, P. (1989). From senility to Alzheimer's disease: The rise of the Alzheimer's disease movement. *The Milbank Quarterly*, 67, 58–102.
- Fried, M. R. (1968). Review of I. N. Berlin & S. A. Szurek, *Learning and its disorders*. *Journal of the American Academy of Child Psychiatry*, 7, 560–563.
- Fuster, J. (1995). Memory and planning: Two temporal perspectives of frontal lobe function. In H. H. Jasper, S. Riggio, & P. S. Goldman-Rakic (Eds.), *Epilepsy and the functional anatomy of the frontal lobe*. New York: Raven Press.
- Garber, S. W., Garber, M. D., & Pizman, R. F. (1996). *Beyond ritalin: Facts about medication and other strategies for helping children, adolescents and adults with attention deficit disorders*. New York: Villard Books.
- Gladwell, M. (1999, February 15). Running from ritalin. *The New Yorker*, 74(46), 80–84.
- Goldman-Rakic, P. S. (1992). Working memory and the mind. *Scientific American*, 267(3), 110–117.
- Hacking, I. (1995). *Rewriting the soul: Multiple personality and the sciences of memory*. Princeton, NJ: Princeton University Press.
- Hacking, I. (1999). *The social construction of what?* Cambridge, MA: Harvard University Press.
- Hale, N., Jr. (1995). *The rise and crisis of psychoanalysis in the United States: Freud and the Americans, 1917–1985*. New York: Oxford University Press.
- Hallowell, E. M., & Ratey, J. J. (1994). *Driven to distraction*. New York: Pantheon Books.
- Hancock, L. N. (1996, March 18). Mother's little helper. *Newsweek*, 127(12), 51–56.

- Haraway, D. (1989). *Primate visions: Gender, race, and nature in the world of modern science*. New York: Routledge.
- Healy, D. (1997). *The anti-depressant era*. Cambridge, MA: Harvard University Press.
- Hinshaw, S. P. (1987). On the distinction between attentional deficits/hyperactivity and conduct problems/aggression in child psychopathology. *Psychological Bulletin*, 101, 443–463.
- Hinshaw, S. P. (1997). Personal communication.
- Hinshaw, S. P., & Anderson, C. A. (1996). Conduct and oppositional defiant disorders. In E. J. Mash & R. A. Barkley (Eds.), *Child psychopathology*. New York: Guilford Press.
- Horn, M. (1989). *Before its too late: The child guidance movement in the United States, 1922–1945*. Philadelphia: Temple University Press.
- Institute of Medicine. (1990). *Research on children and adolescents with mental, behavioral, and developmental disorders: Report of a study*. Rockville, MD: U.S. Dept. of Health and Human Services, Alcohol, Drug Abuse, and Mental Health Administration, National Institute of Mental Health.
- James, W. (1950). *The principles of psychology*. London: Dover Books. (Original work published in 1890.)
- Kagan, J. (1994). *Galen's prophecy: Temperament in human nature*. New York: Basic Books.
- Kanner, L. (1957). *Child psychiatry* (3rd ed.). Springfield, IL: C. C. Thomas.
- Kanner, L. (1972). *Child psychiatry* (4th ed.). Springfield, IL: C. C. Thomas.
- Kirk, S. A., & Kutchins, H. (1992). *The selling of DSM: The rhetoric of science in psychiatry*. New York: Aldine de Gruyter.
- Kolata, G. (1996, December 17). Ritalin use is lower than thought. *New York Times*, B10.
- Latour, B. (1993). *We have never been modern*. Cambridge, MA: Harvard University Press.
- Leland, J. (1995, October 30). A risky rx for fun. *Newsweek*, 126(18), 74.
- Leventhal, D. S. (1968). The significance of ego psychology for the concept of minimal brain dysfunction in children. *Journal of the American Academy of Child Psychiatry*, 7, 242–251.
- Levy, S. (1959). Post-encephalitic behavior disorder—a forgotten entity: A report of 100 cases. *American Journal of Psychiatry*, 115, 1062–1067.
- Leys, R. (1991). Types of one: Adolf Meyer's life chart and the representation of individuality. *Representations*, 34, 1–28.
- Luhrmann, T. M. (in press). *Of two minds: The growing disorder in American psychiatry*. New York: Knopf.
- National Advisory Mental Health Council. (1990). *National plan for research on child and adolescent mental disorders: A report requested by the U.S. Congress*. Rockville, MD: U.S. Dept. of Health and Human Services, Alcohol, Drug Abuse, and Mental Health Administration, National Institute of Mental Health.
- Pick, D. (1989). *Faces of degeneration: A European disorder, c. 1848–c. 1918*. Cambridge, UK: Cambridge University Press.
- Rabinow, P. (1996). *Essays on the anthropology of reason*. Princeton, NJ: Princeton University Press.
- Rafferty, F. T. (1975). Community mental health centers and the criteria of quantity and universality of services for children. *Journal of the American Academy of Child Psychiatry*, 14, 5–17.
- Ribot, T. (1890). *The psychology of attention*. Chicago: Open Court.
- Richters, J. E., Arnold, L. E., Jensen, P. S., Abikoff, H., Connors, C. K., Greenhill, L. L., Hechtman, L., Hinshaw, S. P., Pelham, W. E., & Swanson, J. M. (1995). NIMH collaborative multisite multimodal treatment study of children with ADHD: I. Background and rationale. *Journal of the American Academy of Child and Adolescent Psychiatry*, 34, 987–1000.
- Rose, N. (1996). *Inventing our selves: Psychology, power, and personhood*. Cambridge, UK: Cambridge University Press.
- Rutter, M. (1977). Brain damage syndromes in childhood: concepts and findings. *Journal of Child Psychology and Psychiatry*, 18, 1–21.
- Schrag, P., & Divoky, D. (1975). *The myth of the hyperactive child: And other means of social control*. New York: Pantheon Books.
- Shorter, E. (1997). *A history of psychiatry: From the era of the asylum to the age of Prozac*. New York: Wiley.
- Showalter, E. (1997). *Hystories: hysterical epidemics and modern culture*. New York: Columbia University Press.
- Silver, L. B. (1980). The crisis in child psychiatry recruitment in the U.S.—circa 1980. *Journal of the American Academy of Child Psychiatry*, 19, 711–719.
- Smith, R. (1992). *Inhibition: History and meaning in the sciences of the mind and brain*. Berkeley, CA: University of California Press.
- Starr, P. (1982). *The social transformation of American medicine*. New York: Basic Books.
- Still, G. F. (1902). Some abnormal psychical conditions in children. *The Lancet*, 12 April, 1008–1012; 19 April 1077–1082; 26 April, 1163–1168.
- Strauss, A. A., & Lehtinen, L. E. (1947). *Psychopathology and the education of the brain-injured child*. New York: Grune and Stratton.
- Stephens, S. (Ed.). (1995). *Children and the politics of culture*. Princeton, NJ: Princeton University Press.
- Sulloway, F. (1996). *Born to rebel: Birth order, family dynamics, and creative lives*. New York: Pantheon Books.
- Swanson, J., Castellanos, F. X., Murias, M., Lohoste, G., & Kennedy, J. (1998). Cognitive neuroscience of attention deficit hyperactivity disorder and hyperkinetic disorder. *Current Opinion in Neurobiology*, 8, 263–271.
- Swanson, J., Posner, M. I., Cantwell, D., Wigal, S., Crinella, F., Filipek, P., Emerson, I., Tucker, D., & Nulcioglu,

- O. (1998). Attention-deficit/hyperactivity disorder: Symptom domains, cognitive processes, and neural networks. In R. Parasuraman (Ed.), *The Attentive Brain*. Cambridge, MA: MIT Press.
- Thomas, A., Chess, S., & Birch, H. (1968). *Temperament and behavior disorders in children*. New York: New York University Press.
- Thomas, A., & Chess, S. (1977). *Temperament and development*. New York: Brunner/Mazel.
- Turkle, S. (1978). *Psychoanalytic politics: Freud's French revolution*. New York: Basic Books.
- Vatz, R. E. (1994, July 27). Attention deficit delirium. *Wall Street Journal*, A16.
- Wender, P. (1995). *Attention-deficit hyperactivity disorder in adults*. New York: Oxford University Press.
- Werry, J. S. (1997). "Attention-deficit hyperactivity disorder with bipolar disorder, a familial subtype?" *Comment. American Journal of Child and Adolescent Psychiatry*, 36, 1303–1304.
- Wilson, M. (1993). DSM-III and the transformation of American psychiatry: A history. *American Journal of Psychiatry*, 150, 399–410.
- Wright, R. (1994). *The moral animal: The view science of evolutionary psychology*. New York: Pantheon Books.
- Young, A. (1995). *The harmony of illusions: Inventing post-traumatic stress disorder*. Princeton, NJ: Princeton University Press.