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Attention Deficit Hyperactivity Disorder and Scientific Epistemology

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Attention Deficit Hyperactivity Disorder (ADHD) continues to be controversial with arguments for and against its veracity being waged by individuals representing a variety of disciplines from behavioral scientists to philosophers. Our perspective focuses on the epistemological underpinnings of what is now commonly known as ADHD. Its ignominious history and current disputes may stem from a "pessimistic" epistemology, meaning that truth is only the province of persons in authority and power. The authoritative organizations that govern the diagnostic labels and criteria are the American Psychiatric Association and their Diagnostic and Statistical Manual and the World Health Organization that sponsors the International Classification of Disease. We contrast the pessimistic epistemology with criteria for truth from the scientific method. Although scientific scrutiny has been and is being applied subsequent to "authoritarian edicts" of the disorder, we opine that ADHD currently does not have status beyond that of the "hypothetical construct." Moreover, current brain-based causal models have failed to provide rigorous supporting data that comes from testing falsifiable hypotheses.

Keywords: ADHD, epistemology, scientific method.

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INTRODUCTION

The world's first scientific organization, the Royal Society of London, represented, in part, a revolt against truth via authority in 17th century Europe. The motto of the Royal Society "Nullius in Verba" (on the words of no one) attests to the notion that scientific epistemology is based on such principles as quantification, hypothesis testing, refutation, and replication, and in applied areas, arriving at nosologies based on empirical demonstrations rather than on the edicts of persons in power positions. Not only are authoritarian edicts contrary to scientific inquiry, one obvious problem with truth via authority is that persons in authority may not agree with each other. Such is the case in the broad formulations of two authoritative bodies responsible for the nosology of what is currently termed (at least in the United States) "Attention Deficit Hyperactivity Disorder" (ADHD).

EPISTEMOLOGICAL CONCERNS

The current conceptualizations and diagnostic criteria for Attention Deficit Hyperactivity Disorder are governed by two groups: the World Health Organization (WHO) and their International Classification of Disease (the tenth and latest edition was published in 1993) and the American Psychiatric Association, publisher of the Diagnostic and Statistical Manual of Mental and Emotional Disorders, now in its fourth edition (American Psychiatric Association, 1994). Both employ what Achenbach (2003) termed a "top-down" approach. That is, a committee composed of experts (top) negotiated and eventually arrived at an appellation and established, through a consensus of opinion, diagnostic criteria (bottom). The experts certainly use clinical experience and intuition in their judgments and are influenced by germane data; but the ultimate criterion for truth is consensus by the expert panel. The diagnostic category was not operation-

ally defined; rather, there were decision-rules for inclusion and exclusion. Technical aspects of the diagnosis (forms of reliability and validity) are investigated after the committee opines regarding the nature of the disorder. Both groups provide provisional, periodically mutating diagnostic criteria for mental and emotional disorders. Subsequent research is guided, not by the heuristics of scientific theory, but by the nuances of authority-determined dicta. A resultant diagnostic classification would thus be "phenomenological", based on intuition, appearance, and subjective interpretation.

In contrast, the "bottom-up" approach is data-based. It begins with designation of a large number of symptoms measured in a multimodal manner via for example, direct clinical observations, self-reports, and reports from significant people such as parents and teachers. If consistencies between and among measurement methods are extant, the data would be analyzed to ascertain which behaviors tend to covary with each other but are distinctive in relation to other symptoms. If symptoms such as inattentiveness, over-activity, and impulsivity emerged as convergent and distinctive, a verbal (diagnostic) label that reflects a common element among them would be derived; this comes after data collection and analysis, not before. Hypotheses as to why such observables converge could then be entertained and investigated with study designs that allow for the possibility of refutation. The data themselves "speak" not persons in power positions. Theories regarding etiology of a reliable, scientifically established entity could then be formulated and tested, leading to the possibility of diagnosis based on established causal variables.

It is not astonishing to find that there are some fundamental differences between the two current systems adhering to authoritative "top-down" diagnostic formulations. For instance, what is currently "Attention Deficit Hyperactivity Disorder" in the Diagnostic and Statistical Manual of Mental Disorders, (American Psychiatric Association, 1994) is termed "Hyperkinetic Disorder" (HD) in ICD-10. A major disparity between ICD-10 and DSM-IV is that ADHD (or Hyperkinetic Disorder) is unitary in the former, but has three separate categories in the latter. Another difference is that ICD-10 requires expression of the trinity of symptoms (inattention, hyperactivity, impulsivity) across home and school; DSM-IV requires only that "some" of the symptoms be cross-situational. Further, co-morbidity with other disorders is rather freely allowed in DSM-IV but is quite restricted according to ICD-10 criteria. Not surprisingly, when direct comparisons are made between DSM-IV and ICD-10 diagnoses, markedly different prevalence rates obtain (in Lee et al., 2008).

In the meantime, for the last fifteen years or so, practitioners have acceded to the authoritarian approach to truth, and have placed children and adolescents into one of the three DSM subtypes of ADHD although the subtypes have not been unequivocally substantiated nor are there clear indicators for levels of symptom severity necessary to infer abnormality (Nig et al., 2002). In addition, practitioners have endured the successive mutations and changing DSM verbal labels for the diagnostic construct: "Hyperkinetic Reaction involving Motoric Disinhibition" (DSM-II: American Psychiatric Association, 1968), "Attention Deficit Disorder" (DSM-III: American Psychiatric Association, 1980), the addition of "hyperactivity" to "inattentiveness" and the resultant relegation of "hyperactivity" to secondary status (i.e., "Attention Deficit Hyperactivity Disorder" (DSM-IIIR: American Psychiatric Association, 1987)), and most recently the various aforementioned subtypes.

Whereas clinicians, researchers, and philosophers of science continue to debate many of the peripheral nuances of ADHD (or HD), the fundamental problem in the classification may be more central. That is, ADHD simply may not exist as a distinctive diagnostic entity. There are contradictions, ambiguities and illogic in every aspect (see Tait, 2009; Visser et al., 2009). Moreover, from a medical perspective it has neither an established cause nor an objective diagnostic test and does not meet criteria for disease identification (Furman, 2008). Certainly, children can be inattentive, impulsive and overly-active. The basic question is whether or not these symptoms co-occur consistently with intensity and duration both intra-individually and across situations to a sufficient degree to comprise a reliable mal-

adaptive entity. The answer according to Furman (2008) is that the triad of symptoms can be a non-specific component of a variety of medical and psychosocial conditions. An important consideration is that the symptoms might be better construed as features of other technically superior externalizing (i.e., poorly controlled) disorders. Regardless, the current status of ADHD is what one might expect from a diagnostic category concocted by authoritative opinion rather than a bottoms-up scientific approach: Discordant "official" names for the disorder, questions about the unitary nature of the syndrome, adequate reliability only for empirically derived rating scales in which DSM categorical data are transformed into continuous variables (Collett et al., 2003), moderate levels of predictive and concurrent validity, again with rating scales, and essentially no construct validation (see Carey, 2002). This is in the context of extreme variability among individuals placed in this classification, and pronounced co-morbidities with disorders that likewise may subsume impulsivity, hyperactivity, and inattentiveness (e.g., conduct disorder, oppositional defiance).

TWO CONTEMPORARY EXECUTIVE FUNCTION CAUSAL MODELS

Essential acquiescence to an authoritative epistemology has precluded a typical sequence in application of rigorous methodology- e.g., observation, measurement, construct explication, and evaluation of predictive accuracy. Even though ADHD may be a social and cultural invention by persons in authority, it, whatever "it" be (perhaps nothing more than a reification of the holy trinity of symptoms) is nonetheless assumed to be ontologically valid with roots in genetic and neurological abnormalities as evinced by the most frequent type of treatment using psychostimulant medications. In addition, the situation has been further muddied with the acceptance of spectrum-disorders and adult forms of the postulated disease entity. In keeping with this perspective, some of the most respected researchers and theoreticians have forged ahead, often with brilliance and fecundity, to construct biological, brain-related etiological models. Foremost among these are models involving executive functions mediated by the prefrontal cortex. Perhaps the most cited model of this type is the Behavior Disinhibition Model of Barkley, initially espoused in 1997, according to which the symptoms (or at least some of the symptoms) of ADHD result from an inability to suppress inappropriate, prepotent responses to stimuli. In simplified form, this leads to a failure to develop adequately or execute other components of executive functioning such as impulse control, attention and working memory. The inability to suppress is referred to as "disinhibition" and is postulated to exist as the core cognitive process upon which other executive functions depend. The model thus places emphasis on the one symptom of impulsivity as the core deficit (Barkley, 1997).

While tacitly accepting disinhibition as an executive function deficit in the etiology of ADHD, Rapport (2001) has argued cogently that inhibition cannot occur until *after* an environmental event (stimulus) has been registered or encoded. Logically, an individual cannot control an inappropriate impulse until such a response has been activated, i.e., until the environmental stimulus has been perceived and processed in some manner. Inhibition (or disinhibition) cannot precede but must follow the registration of a stimulus. Recognition of this has led to the development of a model in which executive function primacy is assigned to *working memory*.

As an executive function, working memory is a set of cognitive processes involved in various phases of adaptation. In the original Rapport (2001) model, these processes include recognition of sensory inputs by matching stimulus elements to commensurate material that has been stored, followed by activation of the stored representations, and a comparative judgment involving the degree to which the input stimuli correspond to what has been stored. According to this model, working memory is involved in accessing stored memories and retrieving information about responses that are appropriate for the input stimuli. The failure of working memory functions not only results in disorganized behavior (impulsivity) but also motivates children actively to seek more rapid inputs to working memory (hyperactive stimulus-seeking) because

of the rapid rate in which these processes fade.

In its earliest formulation the Rapport model neglected inattention but accounted for impulsivity and hyperactivity symptoms. Borrowing from Baddeley's model of working memory (2001), the concept of a "central executive" or an "attentional control system" was introduced. This has no memory storage functions in itself but coordinates two subordinate systems of working memory specialized for verbal and nonverbal inputs. The central executive construct allowed Rapport to re-emphasize attentional processes as part of his model. Recent studies have tended to confirm deficiencies in both types of working memory when boys diagnosed with ADHD are compared with typically developing youngsters (Rapport et al., 2008; Rapport et al., 2009).

PREFRONTAL CORTEX DYSFUNCTIONS AND SCIENCE

The argument as to which of these executive function (prefrontal) models is most tenable has occurred in the main at a verbal, conceptual, and "debate" level, not via scientific scrutiny. They are thus arguments about various abstractions that are presumed—but not shown—to be based in various indeterminate neural circuits etc. Their ontological status is thus somewhat suspect, and one can be forgiven the suspicion that—as classically occurred with Greek philosophers from Plato onward—they may be only reified abstractions, examples of what Alfred North Whitehead once famously termed the "fallacy of misplaced concreteness" (Whitehead, 1997).

Presumably, these theoretical models could potentially yield a critical investigation related to differential predictions: Which of the executive functions is primarily dysfunctional in ADHD? This should be something that could be done by any competent researcher in this area, and yet the arguments remain verbal and not data based; a study could easily be envisaged that would allow probabilistic conclusions about which set of inter-related constructs, Rapport, et al., versus Barkley, is most adequate (see Kuhn, 1970). As of this writing, such an investigation has not been attempted.

These theoretical attempts to explicate a neurological basis for the ADHD diagnostic category have been highly influential. But like the authoritarian violations of scientific orthodoxy, several canons of science have not been followed by investigators. Neither theory to date has provided a statement of an event that would potentially refute the model or components thereof. The research is replete with confirmatory hypotheses (and indeed corroborating data) but not with hypotheses that could potentially be falsified; instead, discordant data are simply explained post hoc and ad hoc (see Johnson et al., 2009). Neither theory has been subjected to a potentially refutable investigation nor has a study been carried out to compare differential predictions or the primacy of disinhibition versus working memory in accounting for the triad of ADHD symptoms. We are in agreement with Visser and Jehan (2009) that attempts to localize the disorder in terms of specific brain pathology have resoundingly failed.

Executive function theories have extensive corroborating evidence. Indeed, there is considerable documentation of activation and perfusion deficits in the hypothesized areas of the prefrontal cortex corresponding to impulse control and working memory (for meta-analytic reviews, see Willcutt et al., 2005; Alvarez et al., 2006). But, as mentioned, the studies thus far have been geared toward hypothesis substantiation, not possible refutation.

There are other violations of rigorous scientific methodology. For instance, Furman (2008) has reviewed data suggesting that studies involving prefrontal involvement have failed adequately to control brain volume differences or more general types of brain dysfunctions. This means that in neuroimaging research obtained prefrontal findings may be accounted for by low brain volume or more pervasive neuropathology, thus failing to support hypothesized prefrontal deficiencies. Moreover, hypotheses about simple brain region analyses fail to recognize the interconnectedness among areas of the brain or complex brain circuits that might be involved in attention, hyperactivity and impulse control.

It is axiomatic to suggest that an adequate neurobiological theory must account for all extant, germane facts concerning how the brain mediates behavior. Such a theory must be congruent

with what is known about the prefrontal cortex, interconnections of the cortex with subcortical structures, together with existing information about brain development and maturation as well as age-related changes in symptom expression.

It is important to emphasize—but seldom explicitly noted—that working memory and impulse control are not localized in the prefrontal cortex alone but include a complex circuitry involving the basal ganglia, cerebellum, and the reticular formation. It is the subcortical structures (e.g., striatum) that are most vulnerable to early insults and likely account for the early onset of the symptom triad, as they are also the first to appear during development. In contrast, Halperin and Schulz (2006) report that early prefrontal damage rarely causes significant social or behavioral difficulties. In fact, prefrontal dysfunctions should not lead to a manifestation of symptoms until relatively late in development when this region starts to mediate cognitions and related behaviors. It should also be noted that contrary to the two explanatory models we've reviewed, the developmental trend is for an attenuation of ADHD symptoms during adolescence with myelination and other maturation processes that occur in the prefrontal cortex. Extant ADHD symptoms stemming from earlier damage to or deficiencies in subcortical aspects of inhibition and working memory may be compensated for with the increasing involvement of the higher brain centers. These are aspects which are virtually ignored by the working memory and impulse control theorists. Their existence suggests that there are glaring serious deficiencies in the prefrontal cortex dominated theories. It is also important to emphasize that, as abstractions, working memory and impulse control are typically discussed without reference to developmental processes or to the emergence of brain structures and functional connections needed to support them. To that extent, they rather resemble the "soul," another abstract entity with a long and problematic--but even more influential--position in the psychological literature.

CONCLUSIONS

The prodigious work left to be done in relation to the development of a neuroscience basis for ADHD is eloquently presented by Coghill et al., (2005). The current essay addresses a more basic question: it concerns whether or not ADHD is "real," in the sense of "cutting nature at the joints" and reflecting processes corresponding to knowable neurobiological structures or functions, or whether, to the contrary, it conforms to what MacCorquodale and Meehl (1948) termed a "hypothetical construct," its existence self-contained and incapable of being established with reference to anything outside itself. "Affirmative reification" is the answer according to epistemological assumptions of authoritative organizations having nosological power—supported in the intuitions, clinical experiences, and feelings of many mental health practitioners who, in an iterative process resembling auto-suggestion, see what they believe and believe what they see. It seems to us that the criteria for truth and knowledge proffered by a scientific epistemology would be more supportive of a current "hypothetical construct" status for ADHD. In this review, we have pointed out problems with diagnostic classifications determined in the main by authoritative opinion. We have cited research suggesting that the designated symptoms of ADHD may not be distinctive and have cited studies suggesting it does not meet standards for a reliable, homogeneous disease entity. Causal models, based on the presumed neuropathology of symptoms or the dysfunction of sketchily identified circuits (prefrontal cortex) have not generated or tested falsifiable hypotheses and have failed to adhere to other scientific standards. Not only is the field a long distance away from the development of a neuroscience of ADHD (Coghill et al., 2005), but the diagnostic construct itself may not be worth further attempts of revivification. It may be better to scrap it and think things through from first principles again—especially by considering the way that the putative functions (working memory, impulse control) emerge neurodevelopmentally and what circuits are necessary for their effective implementation. Thus far, and in a more positive vein, contributions of Barkley (1997) and Rapport, et al., (2008, 2009) do suggest the

possibility of eventual empirical verification of brain malfunctions as part of ADHD etiology. Established measures of working memory and disinhibition may in the future provide operational definitions for core ADHD symptoms, linking empirical and theoretical domains and moving the disorder from hypothetical construct status to an etiological diagnostic category. But above all, we emphasize Nullius in verba. Not even ours.

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