**Autism: an Enigma**
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**Abstract**
Prior to the 1980s, autism was largely perceived as a rare disorder, affecting less than 0.05% of the population. However, this statistic has radically changed. Today, the United States is ostensibly afflicted with an autism epidemic. The latest report of incidence indicates an appalling 18 fold increase. However, does this rising trend in prevalence truly reflect an increase in incidence? Many analysts argue in opposition to the reported rise, claiming that the rise reflects changes and improvements in case ascertainment. This paper assesses the validity of arguments in favor of and opposed to the notion of an autism epidemic. After analyzing changes in diagnostic criteria, nomenclature, age at diagnosis, methodology, socio-cultural influences, as well as diagnostic substitution, the paper concludes that autism has not truly reached epidemic proportions.

**Introduction**
In the last two decades, the number of children diagnosed with autism has surged in the United States. According to the Center for Disease Control and Prevention (CDC), approximately 1 in 110 children in the United States have an Autism Spectrum Disorder (ASD). This accounts for an astounding 18 fold increase compared to the diagnosis of 1 in 2000 in the early 1980s. However, is this truly indicative of an autism epidemic? ASDs comprise a group of psychological and social disorders including Autistic Disorder, Asperger Syndrome, and Pervasive Developmental Disorder-Not Otherwise Specified (PDDNOS). These subtypes are differentiated by age of onset, severity of symptoms, and the extent of language delay and intellectual disability. Characteristic symptoms of autism include impaired social interaction, delays in language, and repetitive behaviors. ASDs can usually be diagnosed before age three and last throughout an individual’s life. Some children display symptoms of autism within the first few months of birth. However, the majority of children exhibit symptoms up until 24 months or later. A few even develop normally for up to two or three years and then suddenly stop developing mentally (ASDs 1). Thus, diagnosing ASDs is extremely difficult and haphazard since there is no single foolproof medical test that can detect autism. While both government-sponsored and private industries are diligently working towards identifying the etiology of autism, the origins of autism continue to be shrouded in mystery. Over the years, analysts have developed rather broad diagnostic methods which fallaciously lead to the assumption that autism has reached epidemic proportions. In the research article, “Unpacking the Complex Nature of the Autism Epidemic,” Dr. Helen Leonard and colleagues suggest that autism cannot be qualified as an epidemic. Throughout the article, she discusses the impact of changes in diagnostic criteria, reduction in age at diagnosis,
improved case ascertainment, socio-cultural influences, and diagnostic substitution on ASD prevalence. Leonard emphasizes that the determination of the extent of prevalence of autism is crucial to understanding the etiology and prevention of neurodevelopmental disorders such as ASDs (Leonard 548). Thus, the notion of an autism epidemic is rather inflated.

**Diagnostic Changes**

During the past three decades, diagnostic criteria of autism have been continuously tailored, resulting in an increasing frequency of false positive cases of autism. Until the 1960s, the term “autism” was used informally to refer to symptoms of schizophrenia and was not yet regarded as a sovereign disorder. After the American Psychiatric Association (APA) published Diagnostic and Statistical Manual II (DSM-II) in 1968, autism was formally recognized within the diagnosis of childhood schizophrenia. However, schizophrenia at the time was a rare condition, which subsequently led to the assumption that autism too was a rare condition, as autism was regarded as a corollary of childhood schizophrenia. In the minds of many researchers, the entities remained one and the same, until the APA published DSM-III (1980). This new manual formally recognized autism as separate from schizophrenia and categorized it as one of Pervasive Developmental Disorders (PDD). The relaxed criteria defined autism as a sovereign disorder, independent in its symptoms, etiology, and prognosis. As a result, diagnoses of autism suddenly skyrocketed (Eyal 26).

Diagnostic standards were further broadened in terms of age and criteria for determining autistic individuals. For instance, with the publication of DSM-III-R (1987), the age of onset of 30 months was dropped. Instead, the age of onset could be applicable anytime during infancy and childhood. Moreover, the diagnostic criterion of complete lack of social awareness was modified to merely abnormal social responsiveness. The altered standard for diagnosis constituted three main symptoms: lack of social play; adequate speech but incapacity to engage in sustained conversations; restricted range of interests. This basis of diagnosis remained in DSM-IV (1994), although standards were further broadened. For example, whereas DSM-III and DSM-III-R “required individuals to meet six of six criteria for an autism diagnosis…the 1994 version (DSM-IV), which is currently in use, requires individuals to meet any eight of 16 criteria” (Lilienfeld 59). According to “The Autism Epidemic: Fact or Artifact?,” five studies comparing DSM-III and DSM-IV criteria from 1970 to 2000 revealed increases of 1.4 to 1.6-fold frequencies in diagnoses. Specifically, the study examined 454 children
previously diagnosed by the standards of DSM-III and subsequently measured by DSM-IV criteria (Wazana 732). Thus, changing diagnostic criteria to reflect better understanding of autism predictably results in a high rate of “false positive cases” (Leonard 549).

Nonetheless, firm believers of naturalist explanations for increasing diagnoses may claim that these changes in criteria reflect a better understanding of autism. However, they would be hard-pressed to refute such claims, “since the only way to diagnose autism is using the very same behavioral criteria that have changed, and there is no objective marker to use in order to check for their validity” (Eyal 31). In other words, the ambiguity in the etiology of autism, whether the disorder results from genetic, neurological, or environmental factors, makes it impossible to judge an actual increase in autism. Modification of the diagnostic criteria to reflect better understanding of autism results in changing objective analyses. Diagnoses based on these criteria tend to be inflated statistics.

**Nomenclature**

The concept of autism has evolved over the years, as reflected by its increasingly inclusive quality. Understanding autism remains a challenge because of its very broad definition. The meaning as well as the nomenclature of autism has undergone change as it became an entity comprising a broad spectrum of disorders. These changes, in turn, have led to the inclusion of other similar illnesses such as Asperger’s syndrome and PDDNOS. Throughout this metamorphosis, as the diagnostic boundaries of autism expanded an increasing percentage of individuals, formerly diagnosed under other categories of illnesses, were suddenly “autistic.”

In 1943 Dr. Leo Kanner of Johns Hopkins University described autism for the first time. He coined the term based on his observation of 11 children who had withdrawn from human interaction. During the 1940s through the ‘60s, the medical community felt that children who had autism were schizophrenic. This quality of autism as a corollary of childhood schizophrenia was recorded in DSM-II. However, the DSM-III revised this definition, describing autism as a distinct entity and classified it as one of the Pervasive Developmental Disorders. Moreover, the term, Autism Disorder (AD) was introduced. According to the CDC, AD refers to a stronger strain of autism, defined by “significant delays in language, social and communication impairments, and severely limited interests” (ASDs 1). The revision of DSM-III to DSM-III-R saw the introduction of PDDNOS, which refers individuals who display minor symptoms of autistic disorder.
These individuals might only have social and communication challenges. Furthermore, Asperger’s syndrome was later introduced within the Autism Spectrum Disorders. People with Asperger’s syndrome usually have some milder symptoms of autistic disorder. They might have social challenges and unusual behaviors and interests. However, they typically do not have problems with language or intellectual disability. Together, Asperger’s syndrome, PDDNOS, and AD comprise ASDs (Lord 11).

Although there are no case studies that provide statistical analysis of increasing diagnoses from the time autism was termed to its formal recognition as AD under DSM-II, Dr. J. G. William's article “Systematic Review of Prevalence Studies of Autism Spectrum Disorders” describes such increases while comparing typical autism (AD) to ASDs. Specifically, William’s analysis extracts published prevalence studies from MEDLINE and EMBASE and uses the charted statistics to compare prevalence rates of AD to ASDs. The studies indicate that the estimated prevalence of typical autism was 7.1/10,000 compared to 20/10,000 under the broader ASDs. Thus, the diagnosis of merely AD accounts for about 7 cases and the inclusion of PDDNOS and Asperger’s syndrome increase the prevalence rates to 13 additional cases. These findings reinforce the notion of increasing diagnoses arising from a more inclusive autism, which consequently leads to the misconception of rising ASD prevalence (William 6).

Plausibly, many critics question the necessity of having the general concept of ASDs. They believe that among the three disorders comprising ASDs, there is little to no biological homogeneity. Moreover, each disorder has essentially its own specific package of symptoms and behaviors. Throughout the article, “Autism Spectrum Disorders,” Dr. Catherine Lord and S.L. Bishop debunk such notions. They claim that “despite ongoing attempts to ‘unpack’ autism into separable components, significant and early-arising difficulties in basic aspects of social communication and restricted, repetitive behaviors or interests are the commonalities that strongly define this group” (Lord 16). In other words, individuals who are diagnosed with ASD are characterized by mainly three main domains of symptoms that differentiate them from other diagnostic groups. These symptoms include impaired social activity, repetitive behaviors, and limited interests.

**Age at Diagnosis**

Shifting the age of diagnosis to a younger age leads to increasing frequencies of ASD prevalence, although these rates are misrepresentative of autism incidence. Throughout the article, “The Changing Prevalence of Autism in California,” Dr. Lisa A. Croen analyzes such a shift. In her study, Croen examines the “shift of age at diagnosis
from a distribution based on the 1987 California birth cohort to distributions based on the 1991, 1992, and 1996 birth cohorts" (Croen 211). The average ages at diagnosis corresponding to these years were 6.8, 4.9, 4.4, and 3.3 years, respectively. The trend represents a negative function of age in terms of diagnoses, for the mean age at diagnosis for children changed from 6.8 years in 1987 to 3.3 years in 1994. As age at diagnosis decreases, more children with ASD will be included earlier in the cohort. For example, “whereas in the 1987 cohort, by age 5, only 20% of cases of AD are identified, in the 1992 cohort 98.7% are identified” (Wazana 722). Thus, the diagnosis of children at younger ages increases prevalence rates. Such trends are misleading and do not represent a true rise in incidence. Rather, they are indicative of changes in diagnostic perception.

Methodology

In addition to changing age at diagnosis and diagnostic criteria, methodological considerations have also improved the efficiency of case ascertainment, resulting in an artificial ASD increase. According to “Prevalence of Parent-Reported Diagnosis of Autism Spectrum Disorder among Children in the US,” Dr. Michael D. Kogan and colleagues suggest that multiple factors in case ascertainment reflect a rise in ASD prevalence. Kogan asserts that “the last 10 years have seen dramatic increases in available diagnostic services; much greater awareness of the condition among parents, doctors, and educators; and a growing acceptance that autism can co-occur with other conditions” (Kogan 1397). The accumulative effects of such considerations inevitably result in increasing rates of autism prevalence.

Furthermore, methods of screening populations also reflect improved case ascertainment. In “The Incidence of Clinically Diagnosed Versus Research-Identified Autism in Olmsted County, Minnesota, 1976–1997,” Dr. William J. Barbaresi and colleagues assay “the potential for misleading interpretation of results from epidemiological studies that rely on clinical diagnosis of autism to identify cases” (Barbaresi 464). While examining the rate of clinical diagnosis of ASD in Olmsted County, Minnesota, Barbaresi’s findings indicate a rate of 1.5/100,000 from 1980-1983 and 33.1/100,000 from 1995-1997. Alternatively, the rates of research-identified autism were 5.5/100,000 from 1980-1983 and 44.9/100,000 from 1995-1997. Whereas the former case indicates a 22-fold increase, the latter case represents an eight-fold increase. Thus, clinical diagnoses (previously identified cases) reflect a 22-fold increase in apparent change of incidence. However, such a rise is rather unreliable as there is no comprehensive epidemiological approach to identifying ASD incidence. Barbaresi
affirms that the rise can be attributed to enhanced case ascertainment rather than a “true increase in the number of children affected by autism” (Barbaresi 468).

Legal changes may result in improved methods for diagnosis as well. In the article “Three Reasons not to Believe in the Autism Epidemic,” Dr. Morton Ann Gernsbacher investigates the Individuals Disabilities Education Act (IDEA) ratified by Congress in 1991. The federal law governs how states and public agencies provide care to children with disabilities and requires school districts to provide precise counts of children with disabilities. However, “IDEA has resulted in sharp surges in the reported numbers of children with autism” (Gernsbacher 57). Nonetheless these “administrative-based estimates” are not representative of careful diagnoses of autism and therefore lead to distorted prevalence rates of autism. In short, educational based diagnoses are also misrepresentative of ASD prevalence rates.

Socio-cultural Influences

Certain social influences have also misleadingly spurred ASD diagnoses. Throughout the article, “Social Influence and the Autism Epidemic,” Dr. KY Liu endorses the belief that societal influences significantly contribute to the rise in prevalence of autism. However, Liu’s standpoint reflects an “epidemic of discovery” rather than an “epidemic” of autism incidence (Liu 1389). Her thesis pivots about the notion of “information diffusion” arising from close proximity among families. Liu asserts, “that children living in very close proximity to a child previously diagnosed with autism are significantly more likely to be diagnosed with autism than are comparable children who lack such exposure” (Liu 1387).

From 2000–2005, Liu and colleagues conduct a case study to examine the validity of this notion of increasing autism prevalence arising from social diffusion. While assaying a sample population of 953,464 during this five-year period, Liu arrives to the conclusion that, “compared with children who are 501 meters–1 kilometer away from their nearest neighbor with autism, those in close proximity (1–250 meters) to a child with autism have a 42% higher chance of being diagnosed with autism in the subsequent year” (Liu 1408).

Leonard mentions that “socioeconomic disparities may also contribute to…ASD prevalence” (Leonard 551). When presented with such a statement, many would initially reject such an outlandish claim. After all, the assertion at first glance seems self-contradictory. If children from socially disadvantaged backgrounds tend to receive fewer diagnoses than children from more advantaged backgrounds, would not ASD prevalence
be representative of mainly the advantaged groups? Should not this reflect a decrease in ASD prevalence?

The counter-argument remains valid to the extent that socioeconomic influences do not truly and directly reflect a rise in ASD prevalence. However, these influences affect the age at diagnosis, which consequently contribute to increases of autism diagnoses. Despite not being diagnosed as early as socially advantaged groups, children of families of disadvantaged groups will eventually receive diagnoses. When these low-income families do receive diagnoses of autism for children, the ASD prevalence would appear to have increased suddenly. Thus, the incidence of autism remains constant. Rather, certain families receive diagnoses later as a result of socioeconomic differences.

**Diagnostic Substitution**

The rising number of false positive cases of ASD may also be attributed to diagnostic substitution. In defining this hypothesis in her medical journal, “Is There Really an Autism Epidemic,” Dr. Scott O. Lilienfeld asserts “it is possible that the overall ‘pool’ of children with autism-like features has remained constant but that the specific diagnoses within this pool have switched,” (Lilienfeld 59). In other words, Lilienfeld suggests that the incidence of autism may have remained stagnant or increased marginally. Rather, the names or diagnoses ascribed to such disorders may have changed.

Liu endorses this notion of diagnostic substitution when she examines the sample population of California for socio-cultural influences affecting ASD prevalence. While examining these social interactions, she notes that “proximity also increases the chance of autism rather MR [Mental Retardation] diagnosis” (Liu 1388). Thus, diagnostic substitution in favor of autism tends to increase ASD prevalence as well.

In his novel, “The Autism Matrix: The Social Origins of the Autism Epidemic,” Dr. Eyal Gil examines the circumstances resulting in diagnostic substitution. One possibility is that some children may be diagnosed with autism rather than other neuropsychiatric illnesses to “facilitate receipt of needed services, particularly from publicly funded programs such as Early Intervention and special education programs” (Kogan 1400). However, Gil suggests a more subtle motive behind diagnostic substitution. He asserts that an ASD diagnosis for a child is much more appealing for a parent than other mental disorders. Unlike mental retardation or other illnesses, an autism diagnosis usually implies the idea of a “critical window of opportunity,” a period during childhood when intensive intervention can significantly impact “neural pathways” (Gil 23). Ultimately,
parents would prefer an ASD diagnosis for their child because usually, the symptoms of autism can be significantly mitigated.

**Conclusion**

In short, the notion of an autism epidemic is unfounded. Autism incidence has likely increased only marginally throughout the past few decades in the USA. In other word, the pool of autistic individuals has remained more or less constant. Rather, the names and criteria designated to the disorder have changed. Unfortunately, the true incidence and prevalence of ASD cannot be confirmed, as researchers have not yet discovered any biological marker, such as a specific gene, to diagnose the disorder. Current diagnosis is, at best, haphazard. The three main criteria include delays in communication, limited interests, and impaired social interactions. However, there are no specific guidelines for diagnosis, as the criteria changes with improving case ascertainment. Shifting age at diagnosis, changes in diagnostic criteria and nomenclature, as well as socio-cultural influences comprise the fundamental factors that contribute to a misleading rise in autism. Roy Richard Grinker uses a fitting analogy of “a perfect storm” when describing the factors that contribute to the notion of an autism epidemic. He states that the epidemic is merely an illusion, whereby, “all of these factors com[e] together and act together to give us a situation that feels in your gut like an epidemic” (Grinker 143).

**Bibliography**


