

Editorial

## To Diagnosis or Not to Diagnosis?

Trachtman JN\*

Elite Performance and Learning Center, PS, Seattle,  
Washington, USA

\*Corresponding author: Joseph N Trachtman, Elite  
Performance and Learning Center, PS, 1010 NE 63rd  
Street, #101, Seattle, Washington, USA

Received: May 30, 2017; Accepted: July 13, 2017;

Published: July 20, 2017

### Editorial

This editorial is being written from the perspective of a clinician, educator, and researcher who began the treatment and diagnosis of autism in 1970. As is well known there is a reported increase in the incidence of the diagnosis of Autism Spectrum Disorder (ASD). The Centers for Disease Control and Prevention reports the increase from 1 in 150 in 2000 to 1 in 68 in 2012 [1]. Is this an accurate statistic or is it due to a change in the diagnostic criteria, a bias because of funding considerations to either researchers or parents, or some other extrinsic changes? Perhaps an answer can be derived from the application of signal detection theory by weighing the benefits of making a diagnosis against the adverse effects of making a diagnosis [2].

Certainly we must make every effort to provide remediation and assistance to anyone with a disability. The desire to help must be viewed through the prism of labeling any child in a way that can lead to life-long consequences of poor self-image, loss of confidence, self-fulfilling prophecy of failure, and thus even overall well-being [3]. While some children have mental, physical, emotional, social, and/or perceptual challenges, all of these can be remediated either completely or partially. In fact, a child with challenges typically will blossom when they begin to overcome their disability, as they build their self-confidence when experiencing "I tried, and succeeded"! Most, if not all, of these disabilities, will have health insurance reimbursement to ease the financial burden on parents.

It has been reported that children diagnosed ASD have a higher incidence of the genetic mutation Methylene tetrahydrofolate Reductase (MTHFR) than children without the diagnosis [4]. Other reports further state that a helpful treatment for these children is to take a methylfolate supplement [5]. A report notes that children with MTHFR have low muscle tone [6]. As is known, motor development is the precursor to sensory-motor processing, and remediation of these sensory-motor deficiencies can eliminate or reduce the sensory-motor symptoms and signs that result in the ASD diagnosis [7].

Furthermore, the actual cause of autism remains unknown [8]. Unlike an infection, where the bacteria, virus or fungus can be identified, there are no hard signs for autism. The diagnosis is often

based on soft signs and symptoms; this can be problematic, and may lead to an increase in the reported incidence of ASD [9].

We should consider whether "teaches each child according to his way" is a much better approach than labeling the child as ASD. In other words, address the particular disability. If there is more than one disability, a hierarchy can be made so the most important will be addressed first. The drugs prescribed for ASD have side effects, some claims have no scientific basis, and validation studies may be flawed in their scientific technique [10-12].

In summary, the primary concern of a parent, health care practitioner, or educator is the welfare of his child, patient, or student, respectively. Accordingly, the short and long term benefits versus potential adverse effects should be evaluated before making a diagnosis and treatment. It is hoped that in the near future, with breakthroughs in genetics and electronic diagnostic equipment, there will be an even more effective understanding, treatment and diagnosis of ASD.

### References

1. Centers for Disease Control and Prevention.
2. Green DM, Swets JA. Signal detection theory and psychophysics (A reprint, with corrections of the original 1966 ed.). Krieger Publishing Co. 1967; 42: 578.
3. Ullmann LP, Krasner L. A psychological approach to abnormal behavior. Englewood Cliffs, NJ: Prentice Hall. 1975.
4. Paşca SP, Dronca E, Kaucsár T, Crăciun EC, Endreffy E, Ferenz BK, et al. One carbon metabolism disturbances and the C677T MTHFR gene polymorphism in children with autism spectrum disorders. *J Cell Mol Med.* 2009; 13: 4229-4238.
5. James SJ, Melnyk S, Jernigan S, Lehman S, Seidel L, Gaylor DW, et al. A functional polymorphism in the reduced folate carrier gene and DNA hypomethylation in mothers of children with autism. *Am J Med Genet B Neurophysiol Genet.* 2010; 153: 1209-1220.
6. Trachtman JN.
7. Gesell A, Ilg FL, Bullis GE. Vision: Its development in infant and child. New York: Paul B. Hoeber, Inc. 1950; 43: 792.
8. Trachtman JN. Background and history of autism in relation to vision care. *Optometry.* 2010; 79: 391-398.
9. Rutter M. Incidence of autism spectrum disorders: Changes over time and their meaning. *Acta Paediatr.* 2005; 94: 2-15.
10. Trachtman JN. The efficacy of the use of Ritalin for hyperactive children: A critical evaluation. *Journal of Behavioral Optometry.* 1991; 2: 179-185.
11. Ioannidis JPA. Why most published research findings are false. *PLOS Med.* 2005; 2: 124.
12. FDA Consumer Updates. 2107.