How Good Science is used to Promote Bad Treatments: The Story of Brain Inflammation, Autism, and Hyperbaric Oxygen Therapy

SCIENCE, FADS, AND APPLIED BEHAVIOR ANALYSIS by Dr. Thomas Zane

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Pseudoscientific treatments are those that have little to no empirical evidence of effectiveness, yet are portrayed as having roots in science. One characteristic of most fad treatments is the use of scientific jargon to make the treatment appear more scientific than it actually is and thus appear to be supported by more evidence than it actually does. For example, advertisements for countless products refer to ‘scientific testing” or being “thoroughly researched.”

A glaring example of what appears to be a pseudoscientific approach to advance a treatment for autism for which there is currently little to no empirical support is the use of hyperbaric oxygen therapy (HBOT) for children with autism. Hyperbaric oxygen chambers are pressurized containers that increase the atmospheric pressure (e.g., up to 1.3 times normal pressure) and the concentration of oxygen. These chambers originally were designed to treat decompression sickness of deep-sea divers, but they are purported to treat other physical conditions, such as carbon monoxide poisoning and wound healing (e.g., Leach, Rees, & Wilmshurst, 1999; Feldmeier, 2003).

Daniel A. Rossignol and colleagues have conducted several studies on the effect of hyperbaric oxygen chambers on different symptoms of autism (e.g., Rossignol & Rossignol, 2006; Rossignol, Rossignol, James, Melnyk, & Mumper, 2007; Rossignol, et al., 2009). In reviews of the literature provided in these studies, the author(s) cited neuroinflammation of the brain of children with autism as one of the main reasons for using HBOT with children on the spectrum. For example, during an interview discussing how HBOT might improve autism symptomology, Rossignol noted that “a recent study” (no citation provided) found neuroinflammation of studied children with autism (Rossignol & Small, 2006). The danger of neuroinflammation, as described by Rossignol, is “hypoperfusion” (i.e., decreased blood flow to the brain), which could possibly have the negative effects of limited cognition, poor attention, and other behavioral manifestations associated with autism spectrum disorders. Rossignol pointed out that HBOT has been shown to increase the amount of oxygen that is carried via the plasma and thus infused into the tissues. He referred to at least one animal study that showed that HBOT reduced inflammation; thus, possibly the same results could be obtained using HBOT with children with autism. He assumed that if the brains of children with autism were inflamed with reduced blood flow, this could be causing some of the autism symptoms, so by using HBOT, one could increase oxygen flow to the brains and thus alleviate these negative conditions. Rossignol ends his interview by saying,

"...the thing that excites me so much about hyperbaric oxygen therapy, is the anti-inflammatory effects, which I think is going to help a lot of conditions, not just autism... Certainly, it seems like a lot of people talk about increase in oxygenation of the brain as being the mechanism of improvement with autism (p. 951).

All three studies by Rossignol and his colleagues (Rossignol and Rossignol, 2006; Rossignol, et al., 2007; 2009) iterated the same point about neuroinflammation. The general hypotheses by proponents of HBOT are that the brains of children with autism show evidence of neuroinflammation; HBOT might reduce this inflammation; and the result could be improved functioning. As hypothesized by Rossignol and Rossignol (2006), “...autism is...characterized by... neuroinflammation.......HBOT... has potent anti-inflammatory effects and reduces oxidative stress.” (p. 217).

To support the belief of neuroinflammation of the brain of autistic subjects, and thus the use of HBOT that they promote, Rossignol and colleagues cited Vargas, Nascimbene, Krishnan, Zimmerman, and Pardo (2005), who were one of the first to find evidence of neuroinflammation. Vargas and colleagues conducted brain autopsies on 15 persons diagnosed with autism, ranging in age from 5-44 years. A control group of nine individuals with no diagnosis was studied as well. Brain tissues from the subjects were collected and the researchers conducted numerous detailed medical analyses (i.e., immunocytochemistry, cytokine protein arrays, enzyme-like immunosorbent assays). One of the major findings was “... an active neuroinflammatory process...” mostly in the cerebellum of the patients with autism (2005; p. 67).

However, Vargas and colleagues were very circumspect in their conclusions and implications. In fact, they published a “Frequently
Lastly, the authors asked this question in their FAQ: “If there is neuroinflammation in the brain of some autistic patients, is treatment with anti-inflammatory or immunomodulatory medications indicated?” (p. 3). Their answer was clear – “At present, there is no indication for using anti-inflammatory medications in patients with autism” (p.3). In addition, Pardo, Vargas, and Zimmerman (2005) argued that treatment of neuroinflammation in children with autism was not yet clinically indicated. They asserted that there was not yet a clear understanding of the role of neuroinflammation in autism, and it could possibly be the case that neuroinflammation was in fact part of the healing process (Neuhaus, Archelos, & Hartung, 2003).

Thus, researchers who were instrumental in discovering the presence of neuroinflammation in the brains of persons with autism provided very clear and public limitations of how their research might be applied to possible clinical interventions. Further, they cautioned against assuming that neuroinflammation should automatically be considered a debilitating condition. If there is a possibility that neuroinflammation may not be a condition of autism, then using the findings of Vargas and colleagues in support of HBOT to reduce inflammation is questionable. However, Rossignol and others have been conducting research on the effect of HBOT on various symptoms of ASD assuming that neuroinflammation is harmful and its elimination (through the mechanisms of increased atmospheric pressure in the chamber) will lead to reduced inflammation of tissue and improvement. Although Rossignol cited the Vargas and Pardo work on their finding of neuroinflammation, Rossignol never included, in his research papers, the strong precautions disseminated by these authors. At the very least, the proponents of HBOT should more formally repeat the cautions described by Vargas and colleagues.

At this time, HBOT does not have strong empirical evidence of effectiveness in alleviating symptoms of autism (e.g. Granpeesheh, Tarbox, Wilke, Allen, & Bradstreet, 2010; Lerman, Sansbury, Hovanetz, Wolever, Garcia, O’Brien, & Adedipe, 2008). Thus, it must be considered at this time to be a fad treatment. However, its proponents invoke the trappings of science to make the treatment seem more effective than it actually has been proven to be. A little bit of good science (i.e., presence of neuroinflammation) seems to be used inappropriately to promote an unproven therapy (HBOT) as effective. Unfortunately, this is a strategy used by advocates of pseudoscience, but a strategy that will ultimately fail with more knowledge of what comprises real scientific method and the development of a skeptical attitude.

References


