

American Academy of Pediatrics Recognizes Neurofeedback for ADHD

In October of 2012 the American Academy of Pediatrics gave neurofeedback their top rating in application to the behavioral symptoms of ADHD. This means that neurofeedback has met the highest standards currently being applied to the appraisal of psychosocial interventions. Although in our minds some kind of recognition could have happened—and should have happened—decades earlier, it was nevertheless surprising when it occurred.



Just what led to the decision to elevate neurofeedback from somewhere out in the ozone to being ranked along with medication as having met the highest research criteria? The staff at BrainTrain in the UK have compiled a nice document that summarizes the research basis on which the AAP relied. [\[Read Document on braintrainuk.com \(pdf\)\]](http://braintrainuk.com)

In order to be ranked as a “Level 1 Best Support” treatment, neurofeedback had to be evaluated in at least two controlled studies of sufficient size, conducted by two independent groups. The method had to show itself to be superior to placebo, and to be equivalent in outcome to another level 1 or level 2 treatment. The clinical approach had to be manualizable.

Two fairly recent studies carried the burden. The first study compared frequency-based training with slow-cortical-potential or SCP-based training. The comparison group got computerized attention skills training. Neurofeedback yielded the better outcomes in this relatively large study that involved some 102 children (Gevensleben et al., 2009).

The second study was much smaller in size, involving some 20 children in two groups (15 actives, five controls). The distinguishing feature here was that fMRI data were acquired to document the changes induced with the neurofeedback training. These measurements yielded the expected confirming findings, manifesting localized changes in activation that were not seen in the control group. fMRI data were also taken during a continuous performance test, leading to the observation of additional features in the fMRI that discriminated between the experimental and control groups (Beauregard & Levesque, 2006; Levesque, Beauregard & Mensour, 2006).

It should be noted that by 2012 these studies were already three to six years old. It seems it took a while for these studies to percolate into broader awareness. Moreover, the Gevensleben study may not actually represent the best evidence. It found only a modest effect size of 0.6, which is not that impressive. Overall, only 52% of the children in the neurofeedback group benefited significantly. Additionally, over 40% of parents involved in the study were unable to distinguish systematically the children who had done neurofeedback

versus those who had done the attention training, or they picked the wrong group. That's not far from the ~50% that would have been expected if all parents had simply guessed.

When I originally read the Gevensleben paper, it struck me that if our clinical results all along had been no better than what he reported, we would not have been able to stay in business during those early years, when our emphasis was largely on ADHD and public skepticism about neurofeedback was greatest. Imagine nearly half of one's clientele terminating the training at some point with a sense of disappointment, not to mention being somewhat poorer for it. More than that, it is unlikely that the application of neurofeedback to ADHD would have gotten off the ground in the first place. In that event, of course, Gevensleben would never have had a reason to undertake his study.

Let us briefly review the trajectory that has brought us to the present state. The initial impetus was provided when Joel Lubar first worked with Barry Sterman with a focus on controlling seizures. In this context it was observed that hyperactivity also subsided with the SMR training when that was an issue with a particular child. Now this must have been a fairly robust effect in order to draw attention in the first place, and it must have been observed with enough consistency to vector Lubar's subsequent career trajectory in that direction. Then came Joel's initial studies with a joint focus on hyperkinesia and learning disabilities. Again the effects of the SMR training cannot have been subtle or the project would have been abandoned.

In those early days every study undertaken by both Barry and Joel presented a hazard of single-point failure. A negative outcome might well have terminated further pursuits along the same lines. Moreover, a positive outcome could not just mean meeting some criterion of statistical significance. There had to be cases in there that simply removed all doubt that something useful had been accomplished in the training. It is these successes that inspired further pursuits. Of failures there were no doubt many along the way.

In retrospect the growth of the field depended on a concatenation of events that in nearly all cases needed to favor the researcher with a strikingly positive outcome, statistics be damned. None of the early initiatives would meet anyone's ideal of how definitive efficacy research should be conducted. The agenda was exploratory research, and that is conducted according to different rules than apply to full-bore efficacy studies. No apologies needed. There can be no doubt that throughout this process both Lubar and Sterman were personally persuaded beyond reasonable doubt that they were teasing out a robust phenomenon. The purpose of their early research was to place solid science before their peers, to refine the knowledge base and to determine mechanisms. It was not done to extinguish their own doubts as to whether there was anything worth studying.

When neurofeedback was cut off from the funding pipeline in the mid-eighties despite all of the progress that had been made, the work was carried forward largely by a handful of clinicians scattered around the country and abroad. The rules by which clinicians operate differ even more radically from the researcher's ideal, so inevitably a conflict was set up between the research-guided perspective and that of the practitioner-scientist. Lubar and Sterman still held to the hope that a definitive study would be persuasive to the research community. Clinicians were just not prepared to wait for the day. Unfortunately the resulting proliferation of clinical agendas was contrary to the more focused, monolithic research objective.

It did not really matter. The critics of neurofeedback were not looking for better science. Instead, they were looking to stop the progress of neurofeedback in its tracks. Hardly anyone else was paying attention. Matters were much as they are today on the subject of climate change. The denialists are not looking for more conclusive evidence. Their minds are already made up. As a matter of public policy it is not even important to be certain about global warming. Because of the time constants involved, it is important to act upon even a reasonable suspicion that there might be a problem. By the time the skeptics are persuaded, catastrophe

may already be at hand. It seems clear that the skeptics are agenda-driven, and that has no scientific remedy.

The same held true for neurofeedback. The issue was not the quality of the science. The critics had an agenda, and it was the consolidation of the pharmacological treatment model of ADHD. Neurofeedback represented an 'existential threat' to pharmaceutical hegemony in application to ADHD. The studies would never be good enough to satisfy these critics. Their own utterances give them away. After all, the first question for research is not whether the claims for neurofeedback are established to the highest standards. The first question is whether the matter is worthy of further inquiry. Yet the critics were insisting on the best evidence as a pre-condition to even beginning the conversation. To demand that the proponents of neurofeedback—mainly independent practitioners by now—furnish top-echelon data derived from impeccably designed large-scale controlled studies before their attention was merited is the equivalent of telling your fireplace: "Give me fire and I will give you wood." That works really well only if you don't actually want a fire. And they didn't.

The Clinical Evidence

But consider the evidence that was available just from some of these clinicians. In 2000 we published the results of TOVA data gathered from some 32 practices utilizing a common method—our own approach to SMR-beta training (Kaiser and Othmer, 2000). The results are shown in **Figure 1** for the impulsivity measure. Substantial normalization is shown for the subset of participants who started out at least one standard deviation in deficit. There is only one conclusion that can reasonably be drawn from this data, which is that an active treatment is involved. At a minimum, the results call for further inquiry. What is absolutely and categorically ruled out is the judgment that there is nothing here worth pursuing. And yet that is what happened among the critics.

Whereas the critics kept harping on the placebo model, in the real world that is not the issue. We already have Ritalin, so the practical issue is how neurofeedback stacks up against stimulant medication. In 1995 Rossiter and LaVaque published a comparison study between stimulant medication and SMR-beta neurofeedback (Rossiter and LaVaque, 1995). The results are illustrated in **Figure 2** for the four subtests of the TOVA. The results were broadly comparable between the two groups. Neurofeedback matched stimulant medication at its greatest strengths: resolving impulsivity and inattention. In 2004 Rossiter repeated the comparison with even larger groups: 62 versus 46 in the earlier study. The results were even better, as both neurofeedback protocols and medication management had improved over the decade. Rossiter found neurofeedback to be equivalent to stimulant medication in all four test categories, as shown in **Figure 3**. Significantly, the post-training scores were all above norms. The clinical population had been rendered largely indistinguishable from normal.

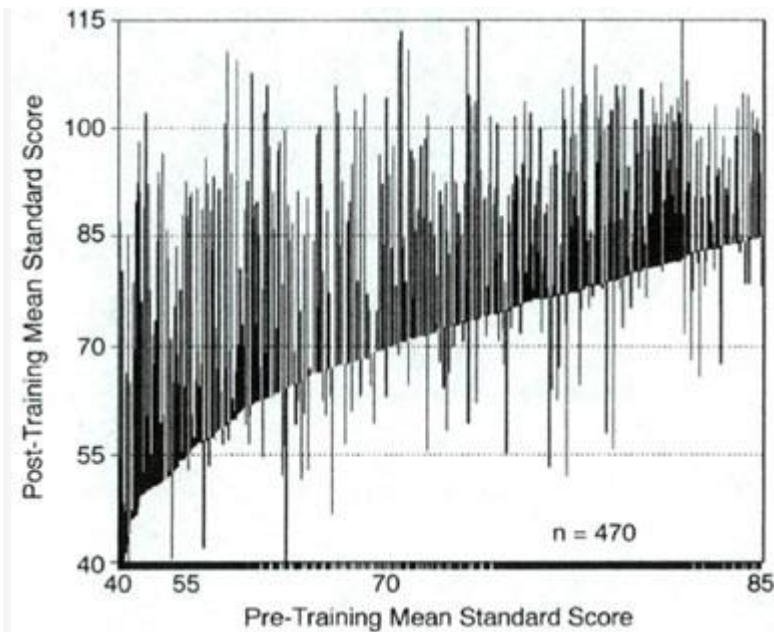


Figure 1 Shown are changes in impulsivity score obtained with the TOVA (Test of Variables of Attention) for a cohort covering some 32 neurofeedback practices. All utilized the protocol referred to as “C3-beta/C4-SMR” that was standard for our practitioner network at the time (1999) in application to ADHD. Standard scores are shown rank-ordered by starting value, and include only those who started off one standard deviation or more in deficit. Data are shown for 470 children and adults. Positive changes in score are both systematically observed and non-trivial in magnitude in most cases.

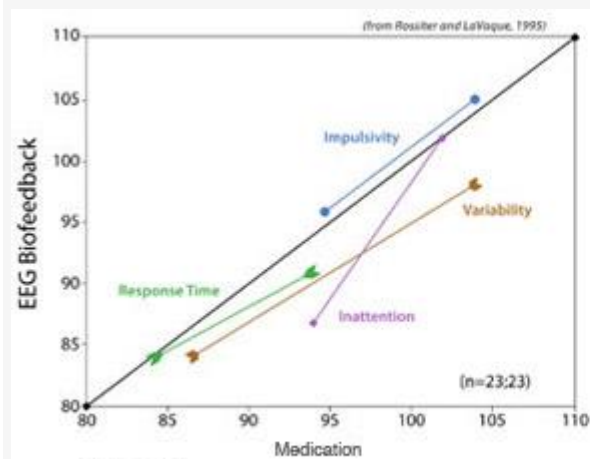


Figure 2

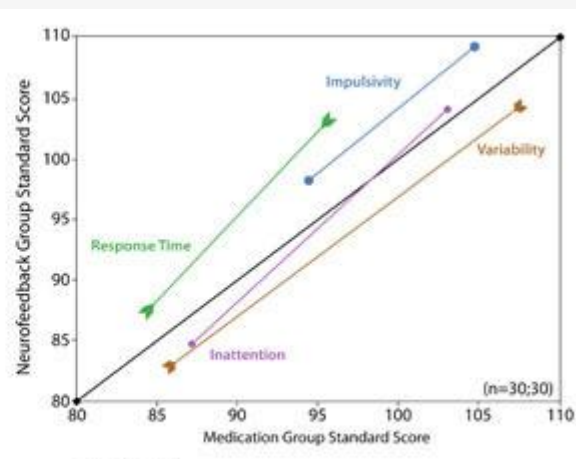


Figure 3

Figure 2 Comparison of EEG neurofeedback (SMR/beta) with stimulant medication utilizing the TOVA as a measure, derived from Rossiter and LaVaque (1995). Pre-post data are shown. Data falling parallel to the diagonal indicate comparable change in both cohorts. Two subtests meet this criterion: impulsivity and variability. The inattention score reveals a slight bias in favor of neurofeedback, whereas reaction time indicates a bias in favor of medication.

Figure 3 Comparison of EEG neurofeedback with stimulant medication using the TOVA as a measure, based on Rossiter (2004). All four subtests of the TOVA exhibit comparable change with neurofeedback and stimulant medication. All post-training scores are above norms in the neurofeedback group.

The effect sizes were large, ranging from 1.0 to 1.7 for the EEG group and from 0.8 – 1.8 for the medication group. There was other supportive data as well. Effect sizes for the Behavior Assessment Scale (BASC) ranged from 1.2 to 1.8, and yielded 1.6 on the Brown Attention Deficit Disorder Scale. This is what successful neurofeedback training looks like.

These findings should have ended all debate. As we know, they did not. Since that time we have also had the results of the follow-up on the Multimodal Treatment study (Jensen et al., 2007), which failed to identify any lingering benefit of stimulants three years after the study was initiated. And we have the additional finding back in 2006 that occasionally Ritalin leads to sudden death due to cardiac events. Those inconvenient truths appear to have been interred. Apparently nothing can slow the Big Pharma juggernaut.

Recognition of neurofeedback had to await the arrival of new researchers on the scene. Observe that the studies on which the AAP acted were all done outside of the United States. It remains true that the academic research enterprise of the United States has not contributed meaningfully to the development of EEG neurofeedback for decades. With psychopharmacology in the ascendancy, behavioral approaches were down-rated in the mid-eighties, and the NIH withdrew its sponsorship of research into this promising new frontier of training brain behavior.

In his latest publication on the subject, Gevensleben writes: “Despite a number of open questions concerning core mechanisms, moderators and mediators, NF (theta/beta and SCP) training seems to be on its way to become a valuable and ethically acceptable module in the treatment of children with ADHD” (Gevensleben et al., 2013). As a statement to the professional community and fellow academics alerting them to neurofeedback, this is certainly welcome. But if neurofeedback is even now only on its way to becoming ethically acceptable, the implication is that it hasn’t been up to now. We strongly disagree.

Ethical questions do arise on all sides in clinical work, but the basic issue of whether it is ethical to provide neurofeedback clinically is not among them. Neurofeedback has been a valuable and efficacious treatment for ADHD already for more than thirty years. We do not need Gevensleben’s research to confer legitimacy on what has been going on for decades. There was never a real question about whether we were merely peddling an expensive computer-aided placebo. We’re happy to see the revival of mainstream research interest in this field, and understand the beneficial effect that formal research can have on mainstream acceptance of neurofeedback. But we will not countenance history being rewritten belatedly. After the original ground-breaking research was accomplished, the thrust of innovation shifted firmly to the clinical domain. It is the clinical community that now embodies the state of the art of the field. Academic research is in catch-up mode—and it is far behind. That will be the topic of the next installment of this newsletter thread.

[Siegfried Othmer, PhD](mailto:drothmer.com)
drothmer.com

References

Beauregard, M., & Levesque, J. (2006). Functional magnetic resonance imaging investigation of the effects of neurofeedback training on neural bases of selective attention and response inhibition in children with attention-deficit/hyperactivity disorder. *Applied Psychophysiology and Biofeedback*, 31, 3–20.

Gevensleben, H., Holl, B., Albrecht, B., Vogel, C., Schlamp, D., et al. (2009). Is neurofeedback an efficacious treatment for ADHD?: A randomized controlled clinical trial. *Journal of Child Psychology and Psychiatry*, 50, 780–789.

Gevensleben H., Kleemeyer M., Rothenberger L.G., Studer P., Flaig-Röhr A., Moll G.H., Rothenberger A., Heinrich H. (2013) Neurofeedback in ADHD: Further Pieces of the Puzzle, *Brain Topography*, 2013 Apr 6, [Epub ahead of print].

Jensen PS, Arnold LE, Swanson JM, et al (2007). 3-Year Follow-up of the NIMH MTA Study, *J Am Acad Child Adolesc Psychiatry*, 46(8), 989-1002

Kaiser D.A., Othmer S. (2000). Effect of Neurofeedback on Variables of Attention in a Large Multi-Center Trial, *Journal of Neurotherapy*, 4(1), 5-15.

Levesque, J., Beauregard, M., & Mensour, B. (2006). Effect of neurofeedback training on the neural substrates of selective attention in children with attention deficit/hyperactivity disorder: A functional magnetic resonance imaging study. *Neuroscience Letters*, 394, 216–221.

Rossiter T.R., La Vaque T.J. (1995). A comparison of EEG biofeedback and psychostimulants in treating attention deficit/hyperactivity disorders. *J Neurotherapy*, 1, 48-59.

Rossiter T. (2004). The effectiveness of neurofeedback and stimulant drugs in treating AD/HD: part II. Replication. *Appl Psychophysiol Biofeedback*, 29(4), 233-43.



This entry was posted on Wednesday, November 27th, 2013 at 8:08 pm and is filed under [ADD / ADHD, Neurofeedback, Research](#). You can follow any responses to this entry through the [RSS 2.0](#) feed. You can [leave a response](#), or [trackback](#) from your own site.