Journal Website

Article history:
Received 10 April 2026
Revised 19 July 2026
Accepted 26 July 2026
Initial Publication 27 July 2026
Final Publication 01 May 2027

International Journal of Education and Cognitive Sciences

Volume 8, Issue 3, pp 1-15

E-ISSN: 3041-8828

Effectiveness of Bigdeli Neuro-Cognitive Learning in Improving Brain-Function Mapping Indicators Associated with Autism Spectrum Disorder

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Article Info

Article type:
Original Research

How to cite this article:
Bigdeli Shamloo, M., Kamarzarin, H., Mohammadi, M., & Pirzal Mobarak, F. (2027). Effectiveness of Bigdeli Neuro-Cognitive Learning in Improving Brain-Function Mapping Indicators Associated with Autism Spectrum Disorder. *International Journal of Education and Cognitive Sciences, 8*(3), 1-15.
https://doi.org/10.61838/kman.ijecs.420

ABSTRACT

Purpose: This study aimed to examine the effectiveness of Bigdeli Neuro-Cognitive Learning in improving EEG-based brain-function mapping indicators associated with ASD-related neurocognitive dysregulation.

Methods and Materials: A quasi-experimental pretest–posttest design with a control group was used. The sample included 66 participants with ASD-related symptoms, assigned to an intervention group (n = 33) and a control group (n = 33). The intervention group received six individual sessions of Bigdeli Neuro-Cognitive Learning over a three-month period, with sessions held approximately once every two weeks, while the control group did not receive the intervention. EEG-based brain-function mapping was conducted under open-eye conditions at pretest and posttest. The main outcomes included absolute and relative power indicators, high-beta activity, hyperconnectivity, and hypoconnectivity.

Findings: After controlling for baseline scores, the intervention group showed significantly lower posttest scores than the control group in absolute delta, absolute theta, relative delta, and hyperconnectivity. These findings indicate improvement in selected neurofunctional indicators related to slow-wave regulation and excessive connectivity patterns.

Conclusion: Bigdeli Neuro-Cognitive Learning may be a promising complementary intervention for improving selected brain-function mapping indicators associated with ASD-related neurocognitive dysregulation. However, future randomized studies with standardized behavioral ASD measures and follow-up assessments are needed.

Keywords: *autism spectrum disorder; Bigdeli Neuro-Cognitive Learning; brain-function mapping; qEEG; neurocognitive intervention; hyperconnectivity; neuroregulation*

1. Introduction

Autism spectrum disorder (ASD) is a heterogeneous neurodevelopmental condition characterized by persistent differences and difficulties in social communication and social interaction, together with restricted or repetitive patterns of behavior, interests, or activities. Its clinical presentation varies markedly across

individuals in terms of language development, intellectual functioning, sensory responsiveness, adaptive behavior, emotional regulation, and the level of support required in everyday life. In addition to the defining features of ASD, many affected individuals experience difficulties in attention regulation, executive functioning, cognitive flexibility, behavioral inhibition, sensory integration, and adaptive learning. These difficulties can influence academic performance, interpersonal relationships, family functioning, participation in social environments, and the capacity to respond flexibly to changing demands. Consequently, contemporary approaches to autism increasingly conceptualize ASD not as a uniform disorder with a single developmental pathway, but as a spectrum of neurodevelopmental profiles requiring individualized assessment and intervention. The Lancet Commission on the future of autism care and clinical research emphasized the importance of recognizing this heterogeneity and developing person-centered interventions that address meaningful functional outcomes rather than focusing exclusively on reducing observable symptoms (Lord et al., 2022).

The heterogeneity of ASD also creates substantial challenges for intervention research. Children and adolescents with apparently similar diagnoses may differ considerably in cognitive abilities, sensory profiles, behavioral regulation, communication skills, co-occurring conditions, and responsiveness to treatment. Therefore, an intervention that is beneficial for one subgroup may be less effective for another. This variability has encouraged researchers to move beyond broad treatment labels and investigate the specific mechanisms, conditions, and participant characteristics associated with intervention response. The updated Autism Intervention Meta-Analysis of Early Childhood Studies, known as Project AIM, demonstrated that reported intervention effects differ according to the type of intervention, outcome domain, research design, measurement strategy, and methodological quality of the included studies. The findings indicate that the evidence supporting many autism interventions remains uneven and that conclusions about effectiveness must be based on carefully defined outcomes and rigorous controlled designs (Sandbank et al., 2023). Accordingly, there is a continuing need to evaluate emerging interventions through objective indicators that can clarify not only whether change occurs, but also which neurocognitive or neurophysiological processes may be affected.

The amount and intensity of intervention have traditionally been regarded as major determinants of developmental outcomes in autistic children. However, recent evidence suggests that a greater number of intervention hours does not necessarily result in proportionally greater improvement. A comprehensive analysis of intervention amount and outcomes among young autistic children found limited evidence for a simple dose–response relationship between intervention intensity and developmental gains. These findings imply that intervention quality, individualization, developmental appropriateness, learning mechanisms, participant engagement, and correspondence between intervention targets and the child's needs may be more important than the number of treatment hours alone (Sandbank et al., 2024). This conclusion is particularly relevant to structured neurocognitive approaches that may be delivered in a limited number of sessions but incorporate individualized exercises, between-session consolidation, and targeted stimulation of cognitive and regulatory processes. Evaluating such interventions requires attention to the mechanisms through which repeated learning experiences may influence neural organization and functional regulation.

Executive functions represent one of the most important neurocognitive domains associated with functioning in ASD. Executive functions include interrelated processes such as sustained attention, working memory, inhibitory control, planning, cognitive flexibility, response monitoring, and the ability to shift adaptively between tasks or mental sets. Deficits in these processes can affect learning, behavioral regulation, social participation, problem-solving, and the performance of daily activities. A systematic review and meta-analysis of executive functioning in children with neurodevelopmental conditions demonstrated that clinically meaningful executive-function difficulties occur across several diagnostic groups, including ASD. Such difficulties are not merely secondary characteristics; they may contribute directly to challenges in communication, adaptive behavior, self-regulation, and academic performance (Sadozai & Uddin, 2024). Therefore, interventions that target attentional readiness, inhibition, cognitive flexibility, arousal regulation, and adaptive information processing may provide a theoretically relevant pathway for supporting broader functioning in individuals with ASD.

Neurocognitive training is based on the assumption that repeated, structured, and developmentally appropriate cognitive experiences can strengthen underlying processes involved in learning and self-regulation. Rather than teaching only isolated task-specific skills, neurocognitive interventions seek to modify fundamental mechanisms such

E-ISSN: 3041-8828

as attentional control, response selection, working memory, information-processing speed, and cognitive flexibility. Computerized cognitive training has become an increasingly prominent approach because it can provide repeated practice, immediate feedback, graded task difficulty, and individualized training parameters. A systematic review of computerized cognitive training in children and adolescents with ASD reported promising effects in selected cognitive domains, while also demonstrating considerable variation across programs, outcome measures, intervention duration, and participant characteristics. The review emphasized that cognitive-training effects should be interpreted cautiously and that stronger studies are needed to determine whether improvements generalize beyond trained tasks to everyday functioning (Zhang et al., 2025). These findings support further investigation of individualized neurocognitive learning methods that combine structured cognitive stimulation with objective neurofunctional assessment.

The theoretical basis for neurocognitive intervention is closely related to experience-dependent neuroplasticity. Neural systems remain responsive to repeated environmental input, cognitive practice, attentional engagement, and behavioral learning. When an individual repeatedly performs structured exercises requiring attention, inhibition, switching, integration of sensory information, or regulation of arousal, the associated neural pathways may become more efficient or functionally organized. In ASD, this possibility is especially important because difficulties in cognitive control and sensory regulation may be associated with atypical patterns of neural activation and network coordination. Neurocognitive intervention may therefore operate by providing repeated experiences that encourage more adaptive regulation of cortical activity. However, behavioral performance alone cannot fully clarify whether an intervention is accompanied by neurophysiological change. Objective methods such as electroencephalography and quantitative brain-function mapping may provide complementary information regarding alterations in oscillatory activity, arousal patterns, and functional connectivity.

Electroencephalography is a non-invasive method that records electrical activity generated by neuronal populations and allows researchers to examine oscillatory activity across frequency bands such as delta, theta, alpha, beta, and high beta. These frequency bands have been associated with different aspects of arousal, attention, information processing, inhibition, memory, and cognitive control. In ASD, atypical oscillatory patterns have been reported across multiple frequency ranges, although the direction and magnitude of these differences vary according to age, cognitive level, recording condition, analytic method, and clinical characteristics. A systematic review of oscillatory activity underlying cognitive performance in autistic children and adolescents found that variations in neural rhythms were associated with attention, executive functioning, social cognition, working memory, and cognitive flexibility. Nevertheless, the review also highlighted substantial methodological heterogeneity and cautioned against interpreting any single frequency-band pattern as a universal biomarker of autism (Larrain-Valenzuela et al., 2024). EEG-derived indicators should therefore be understood as complementary measures of neurofunctional regulation rather than independent diagnostic criteria.

Among EEG indicators, slow-wave activity in the delta and theta ranges is particularly relevant to attentional readiness, cortical arousal, maturational processes, and cognitive regulation. Excessive slow-wave activity during wakefulness may, in some clinical contexts, be associated with reduced cortical activation, inefficient attention, delayed information processing, or neurodevelopmental dysregulation. However, interpretation must always consider the participant's age, behavioral state, recording conditions, and the relative distribution of activity across frequency bands. Quantitative EEG-based brain-function mapping can provide both absolute power indices, reflecting the magnitude of activity within a frequency band, and relative power indices, reflecting the proportional contribution of that band to total recorded activity. Changes in absolute and relative power following an intervention may therefore provide preliminary evidence of neurofunctional reorganization. Such changes should not automatically be interpreted as direct reductions in autism symptoms, but they may indicate alterations in regulatory processes potentially relevant to attention, cognitive engagement, and adaptive functioning.

Functional connectivity is another important dimension of the neurobiology of ASD. Effective cognition and behavior depend not only on activation within individual brain regions but also on coordinated communication across distributed neural networks. Both excessive connectivity and insufficient connectivity have been discussed as possible features of atypical neural organization in ASD. Hyperconnectivity may reflect excessive, poorly differentiated, or inefficient coordination among neural systems, whereas hypoconnectivity may indicate

E-ISSN: 3041-8828

insufficient integration between networks required for complex cognitive, sensory, or social processing. A multimodal investigation combining EEG and functional near-infrared spectroscopy identified alterations in dynamic brain-network organization in individuals with ASD, providing evidence that autism may involve differences in both the strength and flexibility of functional connectivity (Li et al., 2024). These findings suggest that connectivity-related indicators may be useful outcomes in intervention studies seeking to determine whether structured cognitive learning is associated with changes in neural-network regulation.

Neurofeedback and EEG-informed interventions have emerged from similar theoretical foundations. Neurofeedback provides individuals with information about aspects of their brain activity and uses repeated training to promote neural self-regulation. Research has suggested that neurofeedback may improve selected cognitive functions in some children with ASD, particularly those related to attention, executive control, and behavioral regulation. For example, neurofeedback training has been associated with the recuperation of certain cognitive functions among autistic children, supporting the possibility that neural activity can be modified through structured learning and feedback (Kouijzer et al., 2023). Nevertheless, neurofeedback findings remain heterogeneous, and methodological concerns such as small samples, inconsistent protocols, limited blinding, variable outcome measures, and inadequate control conditions restrict definitive conclusions. These limitations underscore the importance of evaluating any EEG-informed or neurocognitive intervention through transparent procedures, comparison groups, baseline control, and appropriately cautious interpretation.

Technological advances have further expanded the possibilities for personalized EEG-based intervention. Wearable EEG systems and machine-learning algorithms may allow more accessible recording, adaptive feedback, automated pattern recognition, and individualization of neurofeedback protocols. Research on wearable EEG neurofeedback for children with ASD has demonstrated the feasibility of integrating machine-learning methods with neurophysiological training, potentially allowing interventions to respond more precisely to the participant's neural profile (Xu et al., 2024). Although such technologies are still developing, they illustrate a broader movement toward mechanism-oriented autism interventions in which behavioral and cognitive training is informed by objective data about neural activity. This movement also supports the use of EEG-based brain-function mapping to identify intervention targets, monitor neurofunctional changes, and investigate whether specific patterns of dysregulation respond differently to individualized training.

Bigdeli Neuro-Cognitive Learning can be situated within this broader movement toward individualized, mechanism-oriented learning interventions. The method is based on structured mental and cognitive exercises intended to influence learning, attention, regulation, and functional performance. Early work introducing the related mind simulation method proposed that structured simulation of cognitive and behavioral processes could facilitate rapid learning and improvement in domains that had previously been approached through more conventional training methods (Bigdeli Shamloo et al., 2021). The proposed framework assumes that carefully organized mental practice may activate cognitive representations, prepare relevant response systems, and support more efficient performance. Although the theoretical and empirical literature on the method remains limited, its emphasis on mental rehearsal, individualized cognitive engagement, and functional learning makes it relevant to contemporary discussions of neuroplasticity and neurocognitive rehabilitation.

Initial applications of the method have extended beyond a single clinical or educational domain. In adolescent sports training, mind simulation was examined as an approach to improving soccer skills, indicating that the method may influence the acquisition or refinement of complex, coordinated performance through structured cognitive rehearsal (Kamarzarin et al., 2021). Subsequent investigations further examined the general effectiveness of mind simulation and proposed that the approach could be adapted to different learning and therapeutic contexts (Kamarzarin & Bigdeli Shamloo, 2024). These studies provide a preliminary basis for conceptualizing the method as a form of active neurocognitive learning rather than passive imagination. Through repeated engagement with targeted cognitive processes, participants may strengthen attentional orientation, response preparation, inhibition of maladaptive patterns, and flexible adaptation to task demands.

Clinical evidence has also begun to emerge regarding the use of the method for communication-related difficulties. Bigdeli Neuro-Cognitive Learning, or the closely related mind simulation approach, has been applied to stuttering and communication attitudes, with findings suggesting potential improvements in speech-related functioning and individuals' attitudes toward communication (Kamarzarin &

E-ISSN: 3041-8828

Bigdeli Shamloo, 2025). Stuttering and ASD are distinct conditions and findings from one population cannot be directly generalized to another. Nevertheless, this work is relevant because it suggests that structured neurocognitive learning may affect complex functions involving attention, anticipation, motor planning, emotional regulation, and communication. The reported effects provide a rationale for investigating whether similar principles may be applied to neurocognitive dysregulation associated with ASD, provided that the intervention is adapted to the individual's developmental profile and evaluated using appropriate outcomes.

Bigdeli Neuro-Cognitive Learning may be particularly relevant to ASD because the intervention is designed to address processes such as attentional readiness, sustained attention, inhibitory control, sensory-cognitive integration, cognitive flexibility, arousal regulation, and adaptive information processing. These processes correspond to domains frequently affected in autistic individuals and are also related to EEG-based indicators of cortical activity and connectivity. An individualized protocol may permit exercises to be selected according to the participant's neurofunctional profile. For instance, elevated slow-wave activity may lead to greater emphasis on alertness and attentional engagement, whereas increased high-beta activity may call for exercises targeting arousal regulation and reduction of excessive activation. Similarly, atypical connectivity patterns may be addressed through tasks requiring integration, switching, coordination, and flexible processing. This individualized structure is consistent with contemporary autism care, which emphasizes matching intervention targets to the person's specific strengths, needs, and functional profile (Lord et al., 2022).

Despite these theoretical connections, controlled evidence regarding the effects of Bigdeli Neuro-Cognitive Learning on EEG-based brain-function mapping indicators in individuals with ASD-related symptoms remains insufficient. Existing autism research supports the potential value of cognitive training, neurofeedback, and individualized intervention, but it also warns against assuming effectiveness without rigorous empirical evaluation. Meta-analytic findings indicate that intervention outcomes may be influenced by methodological quality, measurement selection, participant heterogeneity, and the specificity of treatment targets (Sandbank et al., 2023). Moreover, the absence of a straightforward relationship between intervention intensity and outcome suggests that emerging interventions should be evaluated according to measurable functional and neurophysiological effects rather than session quantity alone (Sandbank et al., 2024). Therefore, examining pretest–posttest changes in absolute power, relative power, high-beta activity, hyperconnectivity, and hypoconnectivity may provide preliminary evidence regarding whether Bigdeli Neuro-Cognitive Learning is associated with improved neurofunctional regulation.

The present study is also important because it distinguishes neurophysiological indicators from direct clinical measures of core autism symptoms. EEG-based brain-function mapping can reveal changes in cortical activity and connectivity, but it cannot independently establish improvement in social communication, repetitive behavior, adaptive functioning, or quality of life. Accordingly, reductions in dysregulated slow-wave activity or excessive connectivity should be interpreted as changes in neurofunctional indicators associated with attentional, regulatory, and cognitive processes. Such evidence may help clarify possible mechanisms of intervention, generate hypotheses for future randomized trials, and support the integration of neurophysiological assessment with standardized behavioral and developmental measures. In this way, the study responds to calls for autism intervention research that is individualized, mechanism-focused, methodologically transparent, and attentive to the distinction between changes in proximal neurocognitive targets and broader clinical outcomes.

Therefore, the aim of the present study was to investigate the effectiveness of Bigdeli Neuro-Cognitive Learning in improving EEG-based brain-function mapping indicators, including absolute and relative frequency-band power, high-beta activity, hyperconnectivity, and hypoconnectivity, among individuals with symptoms associated with autism spectrum disorder.

2. Methods and Materials

2.1. Study Design and Participants

The present study used a quasi-experimental pretest–posttest design with a control group to examine the effectiveness of Bigdeli Neuro-Cognitive Learning in improving brain-function indicators associated with symptoms of autism spectrum disorder (ASD). The study was designed to compare changes in neurofunctional brain-map indices between participants who received Bigdeli Neuro-Cognitive Learning and participants who did not receive the intervention during the same study period.

E-ISSN: 3041-8828

The statistical population consisted of children, adolescents, and young adults with symptoms consistent with autism spectrum disorder who were referred to a neurocognitive and brain-function assessment center for evaluation and intervention. The final sample included 66 participants. Of these, 33 participants were assigned to the intervention group and received Bigdeli Neuro-Cognitive Learning, while 33 participants were assigned to the control group and did not receive the intervention during the study period.

Participants in the intervention group ranged in age from 3 to 22 years, and participants in the control group ranged in age from 4 to 20 years. Both male and female participants were included. Demographic information recorded for each participant included age, sex, handedness, and open-eye EEG recording status. All participants completed the brain-function mapping assessment under open-eye recording conditions.

Participants were included in the study if they met the following criteria: having a clinical diagnosis or clinically significant symptoms consistent with autism spectrum disorder, being within the child-to-young-adult developmental age range, being able to participate in EEG-based brain-function mapping under open-eye conditions, having complete pretest and posttest data, and obtaining informed consent from parents or legal guardians when the participant was under the age of legal consent.

Participants were excluded if they had uncontrolled epilepsy, severe neurological disease, acute psychiatric crisis, severe sensory or motor impairment preventing participation in assessment or training sessions, current use of interventions that could substantially interfere with the study protocol, or incomplete brain-map data. Participants with excessive movement artifacts during EEG recording were also excluded from final analysis if the recorded data were not clinically interpretable.

Participants were selected using purposive sampling from individuals referred to the neurocognitive assessment and intervention center. After initial screening and confirmation of eligibility, participants were placed into either the intervention or control group based on clinical scheduling, family consent, and availability for participation in the intervention program. Because the study was conducted in a clinical setting and full random assignment was not feasible, baseline scores were statistically controlled in the final analysis.

After obtaining permission from the neurocognitive assessment and intervention center, eligible participants were identified from clinical referrals. Parents or legal guardians were informed about the purpose of the study, the assessment procedure, the intervention structure, confidentiality principles, and the voluntary nature of participation. Written informed consent was obtained before the beginning of the study.

At the first stage, demographic information was collected, including age, sex, handedness, and clinical referral information. Then, all participants completed the baseline brain-function mapping assessment under open-eye conditions. The baseline assessment included absolute power, relative power, high-beta activity, hyperconnectivity, and hypoconnectivity indices.

Following the pretest assessment, participants in the intervention group received six individual sessions of Bigdeli Neuro-Cognitive Learning over a three-month period, with sessions held approximately once every two weeks. Participants in the control group did not receive this intervention during the same period. After three months, both groups completed the posttest brain-function mapping assessment under the same recording conditions as the pretest.

2.2. Data Collection Tools

The primary assessment tool in this study was EEG-based brain-function mapping. This assessment was used to examine neurofunctional patterns related to cortical activity and functional connectivity. Brain-function mapping was conducted under open-eye conditions for all participants. The assessment produced quantitative indicators related to spectral power and connectivity patterns.

The recorded indicators included absolute power and relative power in major EEG frequency bands, including delta, theta, alpha, beta, and high beta. The assessment also included indices of hyperconnectivity and hypoconnectivity. These indicators were interpreted as markers of brain-function organization, arousal regulation, attentional readiness, sensory-cognitive processing, and neural network coordination.

In the present study, the term “brain-function mapping” refers to the functional interpretation of EEG-derived indices rather than structural neuroimaging. Therefore, the assessment was not used to diagnose ASD independently, but to evaluate neurofunctional changes associated with the intervention.

E-ISSN: 3041-8828

The main dependent variables were brain-function mapping indices recorded at pretest and posttest. These variables included:

Absolute power indicators:

- AP_DELTA
- AP_THETA
- AP_ALPHA
- AP_BETA
- AP_HIGH_BETA

Relative power indicators:

- RP_DELTA
- RP_THETA
- RP_ALPHA
- RP_BETA
- RP_HIGH_BETA

Connectivity indicators:

- Hyperconnectivity
- Hypoconnectivity

Each variable was recorded in two stages: pretest and posttest. In the dataset, variables ending in "1" represented pretest values, and variables ending in "2" represented posttest values. For example, AP_DELTA1 represented pretest absolute delta power, while AP_DELTA2 represented posttest absolute delta power. The same coding logic was applied to other frequency-band and connectivity indicators.

The brain-function map scores were coded on a five-point scale. Higher values reflected greater deviation from optimal neurofunctional regulation, while lower values reflected a more regulated or normalized pattern. Therefore, a reduction in posttest scores compared with pretest scores was interpreted as improvement in the relevant neurofunctional indicator.

2.3. Intervention

The intervention group received Bigdeli Neuro-Cognitive Learning over a three-month period. The program consisted of six individual sessions delivered approximately once every two weeks. Each session lasted 45 to 60 minutes and was administered by trained neurocognitive specialists. This low-frequency format was selected to allow sufficient time for between-session practice, gradual neurocognitive adaptation, consolidation of regulatory strategies, and monitoring of changes in brain-function mapping indicators.

The six-session protocol was organized as follows. In the first session, the participant's baseline brain-function map was reviewed, and individualized neurocognitive targets were identified. The second session focused on attentional readiness, visual-auditory orientation, and basic response regulation. The third session emphasized inhibitory control, sustained attention, and reduction of impulsive response patterns. The fourth session targeted cognitive flexibility, sensory-cognitive integration, and adaptive switching between tasks. The fifth session focused on regulation of arousal, reduction of excessive activation patterns, and improvement of neurofunctional balance. The sixth session included consolidation of learned neurocognitive strategies, final regulation exercises, and preparation for posttest brain-function mapping.

The intervention protocol was individualized according to each participant's neurofunctional profile. In participants with elevated slow-wave activity, activities were designed to increase alertness, attention stability, and cognitive readiness. In participants with increased high-beta activity, exercises emphasized regulation, relaxation, attentional control, and reduction of over-arousal patterns. In participants with hyperconnectivity or hypoconnectivity patterns, the intervention focused on improving functional integration, cognitive flexibility, sensory regulation, and adaptive information processing. Progression across sessions was based on participant tolerance, task performance, behavioral engagement, and neurocognitive response.

The intervention did not aim to cure ASD. Rather, it aimed to improve functional neurocognitive indicators related to attention, regulation, cognitive flexibility, arousal balance, and connectivity patterns that may contribute to ASD-related symptoms.

Participants in the control group did not receive Bigdeli Neuro-Cognitive Learning during the study period. They underwent the same pretest and posttest brain-function mapping assessments as the intervention group. During the study period, they continued to receive any routine educational, family, or clinical support that they had already been receiving, but no structured Bigdeli Neuro-Cognitive Learning sessions were provided. The control group was included to determine whether observed changes in the intervention group could be attributed to the intervention rather than maturation, repeated testing, spontaneous change, or time effects.

2.4. Data Analysis

Before statistical analysis, the dataset was reviewed for coding accuracy, missing values, duplicate cases, and out-

E-ISSN: 3041-8828

of-range values. Participant names were removed from the analytic file and replaced with numerical codes to protect confidentiality. Demographic variables were coded as categorical or continuous variables as appropriate. Sex was coded as male or female, handedness was coded according to recorded hand preference, and age was analyzed as a continuous variable.

Brain-function mapping indicators were organized into pretest and posttest pairs. For each participant, change scores were calculated by subtracting posttest scores from pretest scores. Positive change scores indicated improvement when higher baseline values reflected greater dysregulation. The distribution of each variable was examined using skewness, kurtosis, histogram inspection, and the Shapiro–Wilk test. When parametric assumptions were not fully met, complementary non-parametric analyses were considered.

Data were analyzed using SPSS software. Descriptive statistics were first calculated for demographic variables and brain-function mapping indicators. Frequency and percentage were used for categorical variables, while mean, standard deviation, minimum, and maximum were used for continuous variables.

To evaluate baseline comparability between the intervention and control groups, independent-samples t-tests were used for continuous variables such as age, and chi-square tests were used for categorical variables such as sex and handedness. Descriptive statistics were then calculated for all pretest and posttest brain-function indicators in both groups.

To examine within-group changes from pretest to posttest, paired-samples t-tests were used for each brain-function indicator. If the assumption of normality was violated, the Wilcoxon signed-rank test was used as the non-parametric alternative. To examine between-group intervention effects while controlling for baseline scores, analysis of covariance was used. In each ANCOVA model, the posttest score was entered as the dependent variable, group membership was entered as the independent variable, and the corresponding pretest score was entered as the covariate.

The main ANCOVA model was defined as follows:

Posttest brain-function indicator = Group + Pretest brain-function indicator

The level of statistical significance was set at $p < .05$. Effect sizes were reported using partial eta squared for ANCOVA and Cohen's d for within-group changes. Effect sizes were interpreted as small, medium, or large according to conventional criteria. For variables with multiple related outcomes, multivariate analysis of covariance was also considered to examine the overall intervention effect across related neurofunctional domains.

3. Findings and Results

Before conducting the main analyses, the dataset was screened for missing values, coding errors, and out-of-range values. The original dataset included 66 participants, with 33 participants in the Bigdeli Neuro-Cognitive Learning group and 33 participants in the control group. During data screening, one participant in the control group had incomplete values in the extracted brain-function mapping indicators. Therefore, this case was excluded from the inferential analyses. The final analytic sample consisted of 65 participants, including 33 participants in the intervention group and 32 participants in the control group. All recorded brain-function mapping indicators were scored on a five-point scale. Pretest and posttest values were available for the main EEG-based functional brain-map indicators, including absolute power, relative power, high-beta activity, hyperconnectivity, and hypoconnectivity.

The intervention group included 33 participants, of whom 27 were male and 6 were female. The mean age of participants in the intervention group was 9.21 years with a standard deviation of 4.70. The age range in this group was from 3 to 22 years. The control group included 32 participants in the final analysis, of whom 25 were male and 7 were female. The mean age of participants in the control group was 8.66 years with a standard deviation of 3.72. The age range in this group was from 5 to 20 years. The two groups were broadly comparable in age distribution and gender composition.

Table 1

Demographic Characteristics of Participants

Variable	Intervention Group (n = 33)	Control Group (n = 32)
Age, M ± SD	9.21 ± 4.70	8.66 ± 3.72
Age range	3–22	5–20
Male, n	27	25

E-ISSN: 3041-8828

Female, n	6	7

Descriptive statistics were calculated for all brain-function mapping indicators at pretest and posttest. In the intervention group, the mean absolute delta score decreased from 3.48 at pretest to 2.67 at posttest. Absolute theta also decreased from 3.64 to 3.18. Relative delta decreased from 3.91 to 3.18. Hyperconnectivity decreased from 4.18 to 3.97. These descriptive changes suggest improvement in several neurofunctional indicators after Bigdeli Neuro-Cognitive Learning. In contrast, the control group did not show a consistent pattern of improvement. For example, absolute delta slightly increased from 3.12 to 3.22, absolute theta increased from 3.50 to 3.81, and hyperconnectivity slightly increased from 4.53 to 4.59. These findings suggest that the observed reductions in several neurofunctional dysregulation indicators were more evident in the intervention group than in the control group.

Table 2

Descriptive Statistics of Brain-Function Mapping Indicators by Group

Indicator	Group	Pretest M ± SD	Posttest M ± SD	Mean Change
AP Delta	Intervention	3.48 ± 1.73	2.67 ± 0.82	0.82
AP Delta	Control	3.12 ± 1.68	3.22 ± 1.75	-0.09
AP Theta	Intervention	3.64 ± 1.62	3.18 ± 0.81	0.45
AP Theta	Control	3.50 ± 1.27	3.81 ± 1.40	-0.31
AP Alpha	Intervention	2.67 ± 1.71	2.36 ± 0.74	0.30
AP Alpha	Control	2.22 ± 1.26	2.25 ± 1.50	-0.03
AP Beta	Intervention	2.91 ± 1.76	3.09 ± 0.88	-0.18
AP Beta	Control	2.56 ± 1.29	2.81 ± 1.62	-0.25
RP Delta	Intervention	3.91 ± 1.26	3.18 ± 0.98	0.73
RP Delta	Control	3.66 ± 1.52	3.69 ± 1.47	-0.03
RP Theta	Intervention	2.94 ± 1.43	3.64 ± 0.78	-0.70
RP Theta	Control	2.91 ± 1.55	3.03 ± 1.71	-0.12
RP Alpha	Intervention	1.48 ± 0.87	2.27 ± 0.63	-0.79
RP Alpha	Control	1.59 ± 1.04	1.72 ± 1.05	-0.12
RP Beta	Intervention	2.52 ± 1.64	3.33 ± 0.99	-0.82
RP Beta	Control	2.25 ± 1.41	2.38 ± 1.62	-0.12
AP High Beta	Intervention	3.24 ± 1.70	3.48 ± 0.87	-0.24
AP High Beta	Control	3.06 ± 1.34	3.28 ± 1.57	-0.22
RP High Beta	Intervention	2.82 ± 1.72	3.64 ± 0.90	-0.82
RP High Beta	Control	2.66 ± 1.45	2.75 ± 1.65	-0.09
Hyperconnectivity	Intervention	4.18 ± 1.24	3.97 ± 0.68	0.21
Hyperconnectivity	Control	4.53 ± 0.80	4.59 ± 0.95	-0.06
Hypoconnectivity	Intervention	2.67 ± 1.02	2.88 ± 0.55	-0.21
Hypoconnectivity	Control	2.84 ± 0.92	3.34 ± 1.29	-0.50

Note. Positive mean change indicates a reduction from pretest to posttest. AP = absolute power; RP = relative power.

Paired-samples t-tests were conducted to examine within-group changes from pretest to posttest. In the intervention group, the reduction in absolute delta was statistically significant, $t(32) = 2.99$, $p = .005$, Cohen's $d = 0.52$. Relative delta also decreased significantly, $t(32) = 2.86$, $p = .008$, Cohen's $d = 0.50$. These findings indicate that Bigdeli Neuro-Cognitive Learning was associated with significant improvement in slow-wave regulation indicators. The reduction in absolute theta in the intervention group approached statistical significance, $t(32) = 1.72$, $p = .096$, but did not reach the conventional significance level. Hyperconnectivity also decreased descriptively in the intervention group, but this change was not statistically significant in the within-group test, $t(32) = 0.84$, $p = .408$.

Some relative power indices increased after the intervention. Relative theta increased significantly, $t(32) = -3.00$, $p = .005$, relative alpha increased significantly, $t(32) = -4.18$, $p < .001$, relative beta increased significantly, $t(32) = -2.55$, $p = .016$, and relative high beta increased significantly, $t(32) = -2.65$, $p = .013$. Because these variables reflect relative distribution of brain activity rather than simple symptom severity, these changes should be interpreted as

E-ISSN: 3041-8828

neurofunctional reorganization rather than straightforward worsening or improvement. In the control group, absolute theta increased significantly from pretest to posttest, t(31) = -3.30, p = .002. Hypoconnectivity also increased significantly, t(31) = -3.36, p = .002. This pattern suggests that, without Bigdeli Neuro-Cognitive Learning, some neurofunctional dysregulation indicators tended to increase over time in the control group.

Table 3

Paired-Samples t-Test Results for Pretest–Posttest Changes

Indicator	Group	Mean Change	t	p	Cohen's d
AP Delta	Intervention	0.82	2.99	.005	0.52
AP Delta	Control	-0.09	-1.14	.263	-0.20
AP Theta	Intervention	0.45	1.72	.096	0.30
AP Theta	Control	-0.31	-3.30	.002	-0.58
AP Alpha	Intervention	0.30	1.01	.320	0.18
AP Alpha	Control	-0.03	-0.21	.839	-0.04
AP Beta	Intervention	-0.18	-0.59	.557	-0.10
AP Beta	Control	-0.25	-1.49	.147	-0.26
RP Delta	Intervention	0.73	2.86	.008	0.50
RP Delta	Control	-0.03	-0.30	.768	-0.05
RP Theta	Intervention	-0.70	-3.00	.005	-0.52
RP Theta	Control	-0.12	-0.94	.354	-0.17
RP Alpha	Intervention	-0.79	-4.18	< .001	-0.73
RP Alpha	Control	-0.12	-0.63	.536	-0.11
RP Beta	Intervention	-0.82	-2.55	.016	-0.44
RP Beta	Control	-0.12	-0.75	.458	-0.13
AP High Beta	Intervention	-0.24	-0.75	.458	-0.13
AP High Beta	Control	-0.22	-1.65	.109	-0.29
RP High Beta	Intervention	-0.82	-2.65	.013	-0.46
RP High Beta	Control	-0.09	-1.14	.263	-0.20
Hyperconnectivity	Intervention	0.21	0.84	.408	0.15
Hyperconnectivity	Control	-0.06	-0.36	.721	-0.06
Hypoconnectivity	Intervention	-0.21	-1.00	.325	-0.17
Hypoconnectivity	Control	-0.50	-3.36	.002	-0.59

Note. Positive mean change indicates reduction from pretest to posttest. Negative values indicate increase from pretest to posttest.

Analysis of covariance was conducted to compare posttest scores between the intervention and control groups while controlling for corresponding pretest scores. The results showed a statistically significant group effect for absolute delta, F(1, 62) = 10.73, p = .002, partial η^2 = .148. The adjusted posttest mean for absolute delta was lower in the intervention group than in the control group, indicating a significant intervention-related reduction in absolute delta activity.

A significant group effect was also found for absolute theta, F(1, 62) = 10.00, p = .002, partial η^2 = .139. The adjusted posttest mean was lower in the intervention group than in the control group. This finding suggests that Bigdeli Neuro-Cognitive Learning had a meaningful effect on theta-related brain-function regulation. The group effect for relative delta was also statistically significant, F(1, 62) = 7.37, p = .009, partial η^2 = .106. The adjusted posttest mean for relative delta was lower in the intervention group than in the control group. This finding supports the effectiveness of the intervention in reducing slow-wave dysregulation indicators.

A significant group effect was found for hyperconnectivity, F(1, 62) = 8.07, p = .006, partial η^2 = .115. The adjusted posttest mean for hyperconnectivity was lower in the intervention group than in the control group. This result suggests that Bigdeli Neuro-Cognitive Learning may have contributed to improved regulation of excessive functional connectivity patterns. The group effect for hypoconnectivity approached significance but did not reach the conventional threshold, F(1, 62) = 3.04, p = .086, partial η^2 = .047. Although the adjusted mean was lower in the intervention group than in the control group, this difference should be interpreted cautiously.

E-ISSN: 3041-8828

For relative theta, relative alpha, relative beta, and relative high beta, significant group effects were observed, but the adjusted posttest means were higher in the intervention group than in the control group. These changes may reflect redistribution or reorganization of relative frequency-band activity rather than simple symptom reduction. Therefore, clinical interpretation of these indicators requires caution and should be considered alongside behavioral and clinical outcomes.

Table 4

ANCOVA Results for Posttest Brain-Function Mapping Indicators Controlling for Pretest Scores

Indicator	Adjusted M Intervention	Adjusted M Control	F	p	Partial η^2
AP Delta	2.56	3.33	10.73	.002	.148
AP Theta	3.15	3.85	10.00	.002	.139
AP Alpha	2.28	2.34	0.06	.808	.001
AP Beta	3.02	2.89	0.22	.645	.003
RP Delta	3.11	3.76	7.37	.009	.106
RP Theta	3.63	3.04	6.24	.015	.091
RP Alpha	2.29	1.71	7.75	.007	.111
RP Beta	3.28	2.43	8.28	.006	.118
AP High Beta	3.45	3.32	0.22	.638	.004
RP High Beta	3.60	2.79	9.48	.003	.133
Hyperconnectivity	3.99	4.58	8.07	.006	.115
Hypoconnectivity	2.92	3.31	3.04	.086	.047

The findings showed that Bigdeli Neuro-Cognitive Learning produced statistically significant changes in several EEG-based brain-function mapping indicators. The strongest and most clinically interpretable effects were observed for absolute delta, absolute theta, relative delta, and hyperconnectivity. In all of these indicators, the intervention group showed lower adjusted posttest scores than the control group after controlling for baseline values. The effect sizes for these significant indicators were in the moderate range. The largest effect was observed for absolute delta, partial $\eta^2 = .148$, followed by absolute theta, partial $\eta^2 = .139$, relative delta, partial $\eta^2 = .106$, and hyperconnectivity, partial $\eta^2 = .115$. These results suggest that Bigdeli Neuro-Cognitive Learning had a meaningful effect on selected neurofunctional indicators related to slow-wave regulation and connectivity patterns.

4. Discussion and Conclusion

The present study examined the effectiveness of Bigdeli Neuro-Cognitive Learning in improving EEG-based brain-function mapping indicators associated with autism spectrum disorder. After controlling for baseline values, the intervention group demonstrated significantly lower posttest scores than the control group in absolute delta power, absolute theta power, relative delta power, and hyperconnectivity. The effect sizes for these outcomes were moderate, with the largest intervention effect observed for absolute delta, followed by absolute theta, hyperconnectivity, and relative delta. Within-group analyses similarly showed significant reductions in absolute delta and relative delta in the intervention group, whereas the control group showed a significant increase in absolute theta and hypoconnectivity over the study period. In addition, relative theta, relative alpha, relative beta, and relative high-beta activity increased significantly in the intervention group. These latter changes should not be interpreted as straightforward deterioration because relative power reflects the proportional distribution of activity across frequency bands and can change when activity in another band, particularly delta, decreases. Overall, the findings suggest that Bigdeli Neuro-Cognitive Learning was associated with measurable neurofunctional reorganization, particularly in slow-wave regulation and excessive connectivity patterns.

The significant reduction in absolute delta power was one of the clearest findings of the study. During wakefulness, excessive delta activity may be associated with reduced cortical readiness, attentional inefficiency, delayed information processing, or broader neurodevelopmental dysregulation, although its clinical meaning depends on age, recording conditions, and individual neurological characteristics. The lower adjusted posttest delta scores in the intervention group may therefore indicate improved cortical activation and attentional readiness following the intervention. This interpretation corresponds with evidence

E-ISSN: 3041-8828

showing that atypical oscillatory activity in autistic children and adolescents is related to cognitive processes such as attention, working memory, inhibitory control, cognitive flexibility, and social cognition (Larrain-Valenzuela et al., 2024). Because Bigdeli Neuro-Cognitive Learning included activities directed toward attentional readiness, response regulation, sensory-cognitive integration, and cognitive flexibility, repeated engagement in these tasks may have contributed to a more regulated pattern of slow-wave activity.

The significant reduction in relative delta provides further support for this interpretation. Relative power represents the proportion of total EEG activity accounted for by a particular frequency band. Therefore, the observed decline in relative delta suggests that the intervention may have reduced the dominance of slow-wave activity within the overall oscillatory profile. Such a redistribution may reflect greater neurocognitive readiness and more efficient engagement of faster frequency bands involved in active information processing. This finding aligns with the theoretical assumptions of cognitive training, according to which repeated and structured cognitive exercises can strengthen attention, inhibition, working memory, and flexible task engagement. A systematic review of computerized cognitive training among autistic children and adolescents reported potential improvements in selected cognitive functions, although intervention effects varied according to task design, duration, participant characteristics, and outcome measurement (Zhang et al., 2025). The present results extend this line of evidence by suggesting that structured neurocognitive learning may be accompanied not only by behavioral or task-related changes but also by alterations in EEG-derived indicators.

Absolute theta activity was also significantly lower in the intervention group than in the control group after baseline scores were controlled. The within-group reduction in theta in the intervention group approached significance, while the control group showed a significant increase over time. Theta activity is involved in several cognitive processes, including memory, monitoring, attention, and executive control, but excessive or poorly regulated theta during wakeful task states may also reflect attentional inefficiency or neurodevelopmental dysregulation. The divergence between the two groups suggests that Bigdeli Neuro-Cognitive Learning may have prevented the worsening of theta-related dysregulation while supporting a more adaptive neurofunctional pattern. This finding is particularly relevant because executive-function difficulties are widely documented in ASD and can affect behavioral regulation, social communication, adaptive functioning, and learning. A systematic review and meta-analysis concluded that executive-function delays are clinically meaningful across neurodevelopmental conditions, including autism, and should be considered central targets in assessment and intervention planning (Sadozai & Uddin, 2024). The intervention's focus on sustained attention, inhibitory control, response monitoring, and cognitive flexibility may therefore explain its effect on theta-related regulation.

The reduction in hyperconnectivity was another important result. Functional hyperconnectivity may represent excessive, rigid, or inefficient coordination among neural systems. In ASD, atypical connectivity has been associated with difficulties in sensory integration, information filtering, cognitive flexibility, and the coordination of complex social and adaptive responses. The significantly lower adjusted hyperconnectivity scores in the intervention group suggest that the intervention may have contributed to more balanced network-level functioning. This interpretation is consistent with multimodal evidence demonstrating altered dynamic brain-network organization in ASD. Research combining EEG and functional near-infrared spectroscopy has shown that autistic individuals may display atypical patterns of connectivity and reduced flexibility in the organization of functional networks (Li et al., 2024). By requiring participants to integrate sensory information, shift between tasks, regulate responses, and adapt to changing cognitive demands, Bigdeli Neuro-Cognitive Learning may have promoted more differentiated and efficient coordination among neural systems.

The finding regarding hyperconnectivity is also consistent with the broader conceptualization of ASD as a condition involving distributed network-level differences rather than dysfunction in a single brain region. Contemporary models emphasize that social communication, executive functioning, and sensory regulation depend on coordinated interactions among multiple neural systems. Excessive synchronization may reduce the flexibility required to process changing environmental demands, whereas excessively weak connectivity may impair the integration of information across regions. In the present study, hyperconnectivity improved significantly, while the group difference in hypoconnectivity approached but did not reach statistical significance. This pattern may indicate that the intervention was more effective in reducing excessive network coordination than in strengthening insufficient connections.

E-ISSN: 3041-8828

It is also possible that hyperconnectivity is more responsive to short-term regulatory training, whereas hypoconnectivity requires a longer intervention period or more intensive practice. Because the study used summary brain-map scores rather than source-localized network analyses, this explanation remains provisional.

The increases in relative theta, alpha, beta, and high-beta activity require careful interpretation. Because relative power values are interdependent, decreases in delta activity can produce proportional increases in other frequency bands even when their absolute power does not increase substantially. In the present study, absolute beta and absolute high beta did not differ significantly between groups, while several relative indices increased. This pattern suggests redistribution of the overall EEG spectrum rather than simple overactivation. An increase in relative alpha may reflect greater organization of resting-state or attentional processes, while relative beta changes may indicate increased involvement of faster frequencies associated with active cognitive processing. Nevertheless, the rise in relative high beta should not automatically be interpreted as beneficial because excessive high-beta activity can also be associated with overarousal. The absence of corresponding increases in absolute high beta makes a redistribution explanation more plausible, but behavioral and clinical measures would be necessary to determine the functional meaning of these changes.

The findings are generally consistent with previous studies of neurofeedback and EEG-informed intervention in ASD. Neurofeedback is based on the premise that individuals can learn to modify patterns of neural activity through repeated training and feedback. Evidence has suggested that neurofeedback may improve selected cognitive functions in autistic children, particularly attention and executive regulation (Kouijzer et al., 2023). Although Bigdeli Neuro-Cognitive Learning is not identical to conventional neurofeedback, both approaches emphasize repeated neurocognitive engagement and the regulation of neural functioning. The present study adds preliminary support to the possibility that structured cognitive exercises tailored to an individual's neurofunctional profile may alter EEG-based indicators. Similar developments are evident in wearable EEG neurofeedback systems supported by machine-learning algorithms, which aim to personalize intervention according to patterns of brain activity (Xu et al., 2024). The current findings therefore fit within a broader movement toward individualized and data-informed neurocognitive intervention.

The results may also be explained through the principle of experience-dependent neuroplasticity. Repeated practice of attentional, inhibitory, switching, and sensory-integration tasks may strengthen regulatory pathways and reduce inefficient activation patterns. Bigdeli Neuro-Cognitive Learning was delivered through individualized sessions that targeted the participant's specific brain-function profile. Participants with elevated slow-wave activity received exercises intended to improve alertness and cognitive readiness, while those with excessive activation patterns received tasks emphasizing regulation and attentional control. Such individualized matching may have increased the relevance of the intervention and contributed to the observed changes. Earlier work on the mind simulation method proposed that structured mental and cognitive rehearsal could facilitate rapid learning in different domains (Bigdeli Shamloo et al., 2021). Its application to adolescent soccer-skill training also suggested that cognitive simulation may support the acquisition and refinement of coordinated performance (Kamarzarin et al., 2021). These earlier findings provide a conceptual basis for understanding how repeated cognitive rehearsal may influence functional regulation.

Subsequent research on the effectiveness of mind simulation has further proposed that the method may be applicable across educational and therapeutic settings (Kamarzarin & Bigdeli Shamloo, 2024). More specifically, research on stuttering and communication attitudes found that the method was associated with improvement in speech-related and communication outcomes (Kamarzarin & Bigdeli Shamloo, 2025). Although these findings cannot be generalized directly to ASD, they suggest that the intervention may affect complex processes involving attention, anticipation, response preparation, emotional regulation, and communication. The present results extend this emerging evidence by indicating that the intervention may also influence neurophysiological indicators. However, because the study measured brain-function mapping outcomes rather than direct behavioral symptoms, the findings should be interpreted as preliminary evidence of neurofunctional change rather than confirmation that the core characteristics of ASD were reduced.

The findings also need to be considered in relation to the broader evidence base for autism interventions. Project AIM demonstrated that intervention effects vary substantially across studies and that many reported benefits become smaller when methodological quality and risk of bias are taken into account (Sandbank et al., 2023). This underscores

E-ISSN: 3041-8828

the need for caution when interpreting findings from a quasi-experimental study. Similarly, evidence regarding intervention amount indicates that greater intensity does not necessarily produce better outcomes, and that intervention quality, individual fit, and the relevance of targeted mechanisms may be more influential than the number of hours delivered (Sandbank et al., 2024). The six-session structure of Bigdeli Neuro-Cognitive Learning may therefore have been effective because it emphasized individualized targets and between-session consolidation rather than high treatment intensity. Nevertheless, stronger research is needed before conclusions can be drawn about its optimal duration or comparative effectiveness.

The findings are also compatible with contemporary recommendations for person-centered autism care. Autism is highly heterogeneous, and intervention planning should account for differences in language, cognition, sensory functioning, adaptive skills, co-occurring conditions, and support needs (Lord et al., 2022). Bigdeli Neuro-Cognitive Learning was individualized according to brain-function profiles, which corresponds with this emphasis on personalized care. However, neurophysiological indicators should not be used in isolation to determine treatment needs. Brain-function mapping may provide useful supplementary information, but it cannot replace comprehensive clinical assessment, standardized behavioral measures, developmental history, caregiver reports, or direct observation. Accordingly, Bigdeli Neuro-Cognitive Learning should be regarded as a potentially complementary intervention rather than a comprehensive or curative treatment for ASD.

The present study had several limitations. The quasi-experimental design and nonrandom assignment reduce the certainty with which observed differences can be attributed exclusively to the intervention. The sample was relatively small and included participants across a wide age range, from early childhood to young adulthood, despite substantial developmental differences in EEG activity and autism presentation. The available outcomes were limited to summarized EEG-based brain-function mapping indicators and did not include standardized measures of social communication, repetitive behavior, executive functioning, adaptive functioning, sensory regulation, or quality of life. The study also lacked a follow-up assessment, preventing evaluation of whether the observed changes were maintained. Furthermore, the control group continued routine services, and the extent of these services was not standardized. The wide interval between intervention sessions may also have allowed maturation, environmental experiences, and other treatments to influence the results.

Future research should use randomized controlled designs with larger and developmentally more homogeneous samples. Studies should stratify participants according to age, cognitive level, language ability, symptom severity, and baseline EEG profile to determine which individuals are most likely to benefit. Standardized behavioral and clinical measures should be administered alongside EEG assessments to establish whether neurofunctional changes correspond with improvements in attention, executive functioning, social communication, sensory regulation, adaptive behavior, and caregiver-reported functioning. Follow-up assessments are needed to examine durability, and comparative studies should evaluate Bigdeli Neuro-Cognitive Learning against established cognitive-training, neurofeedback, and behavioral interventions. Future research should also compare different session frequencies and intervention durations and use source-localized EEG or network-specific connectivity analyses to clarify the neural mechanisms underlying change.

In practice, Bigdeli Neuro-Cognitive Learning may be considered as a complementary component of individualized support for people with ASD-related neurocognitive dysregulation. Clinicians should use it only after comprehensive assessment and should formulate intervention goals in functional terms, such as improving attention, cognitive flexibility, arousal regulation, or sensory integration. EEG-based brain-function mapping may assist with identifying potential targets and monitoring change, but treatment decisions should also incorporate behavioral observation, developmental assessment, family priorities, and standardized clinical measures. The intervention should be integrated with educational, developmental, behavioral, occupational, speech-language, and family-centered services rather than replacing them. Practitioners should also explain clearly that changes in EEG indicators do not necessarily demonstrate improvement in the core clinical features of ASD.

Authors' Contributions

All authors significantly contributed to this study.

Declaration

In order to correct and improve the academic writing of our paper, we have used the language model ChatGPT.

E-ISSN: 3041-8828

Transparency Statement

Data are available for research purposes upon reasonable request to the corresponding author.

Acknowledgments

We hereby thank all individuals for participating and cooperating us in this study.

Declaration of Interest

The authors report no conflict of interest.

Funding

According to the authors, this article has no financial support.

Ethical Considerations

In this study, to observe ethical considerations, participants were informed about the goals and importance of the research before the start of the interview and participated in the research with informed consent.

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E-ISSN: 3041-8828