

# Neurofeedback Training for Social Cognitive Deficits

## A Systematic Review

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**Abstract**—Orndorff and his colleagues [1] suggested that if a neural activity is considered a treatment variable instead of outcome, it widens the scope of research and has a specific implication for social neuroscience. Given this, the empirical evidence is collected and analyzed where neural activity as self-manipulation design through neurofeedback training specifically for social cognition deficit is done. The objective of the present article is to provide a systematic review of 1) how NFT is utilized to treat social cognitive deficits, 2) how NFT is utilized to target Social Cognition Deficit in ASD, 3) examining the directions, strengths, and quality of evidence to support the use of NFT for ASD. The databases for studies were searched in PubMed, MEDLINE, EMBASE, Springer, Science Direct, Psychinfo, and Google Scholar, using combinations of the following keywords: ‘Neurofeedback,’ ‘Autism Spectrum Disorder,’ ‘Mu Rhythm’ and ‘Social Cognition.’ Studies were eligible for inclusion if they were specific to 1) autistic and typically developed population, 2) intervention study, 3) Delivered by NFT, 4) participants showed social cognitive deficit and/or improvement. Total one eighty-seven studies were found of key interest; out of which 17 studies were eligible for inclusion in this review. All studies reported the improvement in different domains of social cognition and were moderately methodologically sound. Eleven out of seventeen studies satisfied the trainability and interpretability criteria suggested by Zoefel and his colleagues [2]. The conclusion from the present review is in line with comments of Marzbani and colleagues [3] that, ‘current research does not provide sufficient conclusive results about its efficacy.’ The patterns and directions concluded from studies related to protocol, methodology and results are discussed in detail in the present review.

**Keywords**—Social Cognition Deficit; Neurofeedback Training; Autism Spectrum Disorder; Systematic Review; Intervention Studies.

## 1 Introduction

The great philosopher, Aristotle said, ‘Man is by nature a social animal,’ [4] in their MIT summer course (Brain, Mind, and Machine) lecture and emphasized that ‘Social intelligence is the crux of human intelligence.’ It is clear that social intelligence/cognition is a key part. The day to day life requires cooperation, competition or simply work with other people and thus it is essential to be able to

understand self and others. Social cognition, the term much similar to the usage of term cognition, refers to all mental process involved in social interaction. Few terms which are dominantly used interchangeably with social cognition are empathy, the theory of mind, mentalizing, mind-reading, intentionality, intention and inter-subjectivity (refer Table 1).

**Table 1.** Terms associated with Social Cognition and their description

S. No.	Term	Description
1	Empathy	Feeling the feelings of others
2	Theory of Mind	Metacognitive understanding of own and others mind
3	Mentalizing	Understanding oneself and others as subjective being with mental states
4	Mind-reading	Ability to attune our behavior to the minds and anticipated actions of others
5	Intentionality	Nature of mind and mental states to be 'about something else.'
6	Intention	Ability to form an image of goal state and pursuit of the goal state
7	Inter-subjectivity	Ability to coordinate mutual interactions in light of our perception of the subjectivity and intentionality of others.

Social cognition is an extensive term, and traditional understanding is varied in different areas like social psychology, social neuroscience, developmental studies, and clinical or psychopathological studies.

### 1.1 Social cognition deficit

The seminal work by Kanner [5] led to the interest in the disease which is characterized by the social cognitive deficit. In clinical or psychopathological context social deficits are common and contribute a great deal to the burden of mental illness or disability (refer Table 2). This has been linked to poor quality of life, mental health problems, unemployment, and loneliness [6], [7] & [8].

**Table 2.** Indications of Social Cognitive Impairment

Social withdrawal or avoidance of social contact
•Loss of social grace
•Limited eye contact
•Rude or offensive comments without regard for the feelings of others
•Loss of etiquette in relation to eating or other bodily functions
•Extended speech that generally lacks focus and coherence
•Neglect of personal appearance (in the absence of depression)
•Disregard of the distress or loss of others
•Inability to share in the joy or celebration of others when expected or invited
•Failure to reciprocate socially, even when obvious social cues are given
•Poor conversational turn-taking
•Overtly prejudiced or racist behavior
•Increased or inappropriate interpersonal boundary infringements
•Failing to understand jokes or puns that are clear to most people
•Failure to detect clear social cues, such as boredom or anger, in conversational partners
•Lack of adherence to social standards of dress or conversational topics
•Excessive focus on particular activities to the exclusion of important social or occupational demands

Source: Henry, Von Hippel, Molenberghs, Lee & Sachdev (2016) [9]

In April 2008, the American Psychiatric Association's (APA) DSM-5 [10] Task Force began work of proposing revisions to the criteria for the disorders referred to in DSM-IV. The social cognitive deficit has been implicated in different categories of disorders, e.g., Psychiatric disorders, a neurodegenerative disorder, and developmental disorder (refer Table 3).

**Table 3.** Disorders with Social Cognitive Impairment

<b>Psychiatric Disorders</b>
•Schizophrenia
•Bipolar disorder
•Antisocial personality disorder
•Major depressive disorder
•Posttraumatic stress disorder
•Social phobia
•Anorexia nervosa
•Personality disorders (for example, borderline, antisocial, narcissistic, schizoid, avoidant personalities)
<b>Neurodegenerative Disorders</b>
•Frontotemporal dementia
•Alzheimer disease
•Amyotrophic lateral sclerosis
•Parkinson disease
•Huntington disease
•Progressive supranuclear palsy
•Corticobasal degeneration
•Multiple sclerosis
<b>Acute Brain Damage</b>
•Traumatic brain injury
•Stroke
<b>Developmental Disorders</b>
•Autism spectrum disorder
•Fragile X syndrome
•Williams syndrome
•Angelman syndrome
•Prader-Willi syndrome
•Turner syndrome
•Rett syndrome
•Attention deficit hyperactivity disorder
•Severe conduct disorder
•Fetal alcohol syndrome

Source: Henry, Von Hippel, Molenberghs, Lee, & Sachdev (2016) [9]

## 1.2 Social cognition deficit in broader autism phenotype

The autism spectrum disorder (ASD), a relatively high prevalence disorder, is marked by the social cognitive deficit. The Asperger’s disorder, pervasive developmental disorder, and autistic disorders are clubbed and given a diagnosis of ASD. The standard criteria’s for diagnosing ASD according to DSM- 5 [10] are shown in Table 4.

**Table 4.** Diagnostic criteria for ASD

A.	<p><b>Persistent deficits in social communication and social interaction across multiple contexts, as manifested by the following, currently or by:</b></p> <p style="text-align: center;"><b>Deficits in social-emotional reciprocity</b></p> <p style="text-align: center;"><b>Deficits in nonverbal communicative behaviors used for social interaction</b></p> <p style="text-align: center;"><b>Deficits in developing, maintaining, and understand relationships</b></p>
B.	<p>Restricted, repetitive patterns of behavior, interests, or activities, as manifested by at least two of the following, currently or by history:</p> <p>Stereotyped or repetitive motor movements, use of objects, or speech</p> <p>Insistence on sameness, inflexible adherence to routines, or ritualized patterns of verbal or nonverbal behavior</p> <p>Highly restricted, fixated interests that are abnormal in intensity or focus</p> <p>Hyper- or hypo-reactivity to sensory input or unusual interest in sensory aspects of the environment</p>
C.	<p>Symptoms must be present in the early developmental period (but may not become fully manifest until social demands exceed limited capacities, or may be masked by learned strategies in later life).</p>
D.	<p>Symptoms cause clinically significant impairment in social, occupational, or other important areas of current functioning.</p>
D.	<p>These disturbances are not better explained by intellectual disability (intellectual developmental disorder) or global developmental delay.</p>
<p>Adapted from <a href="https://www.cdc.gov/ncbddd/autism/hcp-dsm.html">https://www.cdc.gov/ncbddd/autism/hcp-dsm.html</a> [11]</p>	

Autism is labeled as a spectrum because a wide variation is reported in related to the types and severity of symptoms. ASD occurs in all races, ethnicity, social and economic background and is more prevalent in males than females. Kanner [5] observation that the parents, siblings, and relatives of autistic patients are found to be “serious-minded, perfectionistic individuals, with an intense interest in abstract ideas” and showed lack of interest in developing relationships with others. This observation led to the twin study by Folstein & Rutter [12]. They theorize that milder but qualitatively similar traits to defining features of ASD are found among non-affected relatives of ASD individuals. This theory has come to be known as the broader autism phenotype (BAP). The theory explains the social cognitive deficit as a continuum where the typically developed children exist at the extreme pole of functional social cognition and ASD patient at the extreme dysfunctional pole. Within this continuum, the family members, sibling, and relative exist at the point close to the threshold of functional to dysfunctional.

### 1.3 Neurological bases of social cognition deficit

The search for the biological basis of social cognition is a relatively recent; no conclusive findings are there so far to explain the pathologies and individual differences in the outcome of the interaction of situational/environmental and biological factors. It can be said that the deficit in social cognition processes results from damage to the brain areas or their connections that participate in social processing. Responsible area of the brain for cognitive functioning should be well understood because; a minute dysfunction in one structure of brain or damage to white-grey matter [13] can disrupt at the functional level of cognitive processes. Some of the recent studies looked at the specific brain areas or networks involved in eye gazing, facial expressions, on-line mentalizing and reported the critical role of the medial frontal cortex [14]. Based on another set of evidence it can be said that any kind of disturbance at the tempo-parietal junction may result in one's inability to view a situation from another person's perspective, which can also lead to abnormal moral reasoning [15],[16]. The main cause of these disturbances is tempo-parietal junction that plays a central role in integrating social, attentional, memory and language processing to build a social context for behavior [17] (see Figure 1).

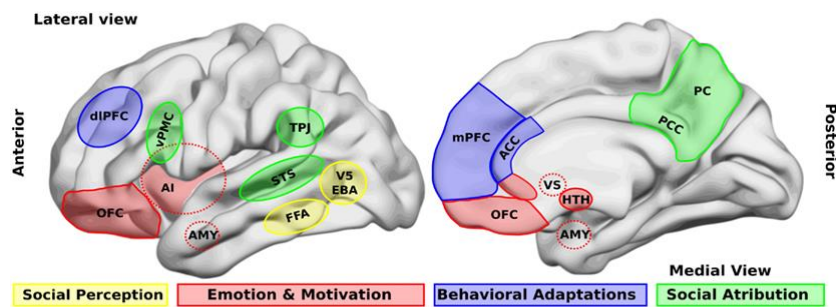


Fig. 1. Brain areas that participate in social processing.

A simple classification of brain areas involved in social processing differentiates regions that participate in four related processes. The first is the perception of basic social stimuli, such as biological motions (V5), part of the body (extra-striate body area, EBA), and faces (fusiform face area, FFA). Another process includes an emotional and motivational appraisal, where the amygdala (AMY), the anterior insula (AI), the subgenual and perigenual anterior cingulate cortex (ACC), as well as the orbitofrontal cortex (OFC) participate. These cortical structures are in interaction with subcortical structures as the ventral striatum (VS), and the hypothalamus (HTH). These structures, in turn, interact with other regions which participate in the goal-directed, adaptive behaviors, and the categorization processes, such as the dorsolateral and the medial prefrontal cortex (dIPFC, mPFC) and the ACC. Finally for social attribution, areas like the ventral premotor cortex (vPMC), the superior temporal sulcus (STS), the AI, the posterior cingulate cortex (PCC), and the precuneus (PC) participate in more automatic, bottom-up inferences of other people's mental states;

whereas structures like the mPFC and the temporoparietal junction (TPJ) are involved in more cognitive theory of mind skills (Adapted from [18]).

Oberman and his colleagues [19] suggested that the mirror neuron system (MNS hereafter) theory is one theory which can link the neuroanatomical and functional mechanism explaining the behavioral symptom typical of ASD. The preliminary evidence is also reported by Oberman [20]. It is hypothesized that MNS is a neural substrate of all social cognitive behavior related to observation and imitation that allows understanding other's goals and actions [21], [22]. The activity of mirror neurons disrupted with imitation and resulted in autism's core features of social impairment and communication difficulties. The activity of MNS is observed as Mu rhythm in the EEG waveform. Marshall and his colleagues [23] reported that Mu rhythm is responsible for the development of imitation at a very early stage of life.

Further, it serves a very important role in the development of, i.e. motor skills, interactive social behavior. A group of researchers believes that excess of mu waves is found in diseased population, i.e. ASD and this excess of Mu leads to affect various motor functions, attention, and various cognitive processes. This hypothesis comes up with the evidences which proved the coexistence of mu rhythms with psychopathological symptoms such as anxiety, aggressiveness, hyperactivity and several other psychosomatic features [24].

#### **1.4 Neurofeedback intervention for social cognition deficit**

The early studies established that behavioral intervention is far more effective than pharmacological treatment for neurodevelopmental disorders [25], [26], [27]. Later, the behavioral interventions were also criticized for their high cost and time consumption. Pineda with his colleagues [28] specifically point out that these tools were only used for high functioning autistic population. Whereas, low functioning ASD patient who has greatest barrier in social communications needs more tools and have more possibility to improve in such disorders at an early stage [29], [30], [31] suggested a biomedical intervention other than these therapies to target the core symptoms and problems associated with autistic spectrum disorder (ASD), i.e., intervention by Neurofeedback training (as supported by [3], [32], [61], [62], [63]).

The NFT studies on epilepsy and ADHD has received much attention; however NFT studies on ASD patient started majorly in new millennium only [33]; though few case studies were reported in the 90s [34]; [35] and [36]. At the turn of the new millennium, studies were reported with the rigorous investigation [37]; [38] and [39] with the majority of studies following the alpha-theta protocol or coherence training. To best of our knowledge, the first study which trained mu rhythm was conducted by Jarusiewicz [40]. The pilot study conducted by Jarusiewicz included twenty-four autistic children and individualized NFT was given to them. However, the majority of them received mu training on the sensory-motor strip. Still, Jarusiewicz has not used the term mu rhythm training or target social cognitive deficit specifically. Therefore, the study which explicitly trained mu rhythm for improvement in social cognition can be labeled to the study by Oberman [20].

## **2 Objectives**

Considering the above background in mind the present systematic review aims to evaluate the evidence for the effectiveness of neurofeedback training for improvement of social cognition deficit. To achieve this aim following research questions are framed to guide the present review:

1. How Neurofeedback Training (NFT) is utilized to treat social cognitive deficits,
2. How NFT is utilized to target Autism Spectrum Disorder (ASD),
3. Examining the directions, strengths, and quality of evidence to support the use of NFT for social cognition deficit in ASD

The expected primary outcome of this review is to ameliorate the evidence for NFT for social cognition deficit in ASD and secondary outcome is to assess the quality, methodology, and strength of studies conducted on people having social cognition deficit.

## **3 Literature Search**

Three approaches were used to search for relevant articles CRD [41]: searching electronic databases, 2) visually scanning reference list from relevant studies, and 3) searching relevant internet resources. Combining all three approaches helps to overcome selection bias, expand the search and inclusion of grey literature. The first step was the electronic database search, conducted twice by two reviewers in PubMed, MEDLINE, EMBASE, Springer, Science Direct, Psychinfo, and Google Scholar, using combinations of the following keywords: 'Neurofeedback,' 'Autism,' and 'Social Cognition.' Then the reference of articles was searched; followed by the search in relevant internet sources. The search was conducted during November/December 2017, and the same was repeated in March/April/May 2018 by both the reviewers separately. After completing the search, all the articles were combined and both the reviewers screened the articles first based on title and abstract of the article then in the second step full article was reviewed. After screening the articles separately, reviewers discussed the studies and assessed them on pre-decided inclusion and exclusion criteria's, until they reached the consensus.

### **3.1 Study selection**

The inclusion and exclusion criteria for this review were based on the objective of the study.

Inclusion criteria: Search made for these major points were specific to: Search made for these major points were specific to 1) autistic and typically developed population, 2) intervention study, 3) delivered by NFT, 4) participants showed social cognition deficit and improvement.

Exclusion criteria: Publications were excluded if: 1) conducted as co-morbid situation with ADD, ADHD or any other developmental disorder, 2) behavioral

intervention only (no NFT), 3) Used NFT but not as treatment, 4) not the primary data work (editorial or reviews), and 5) Not available in English or no full text available

### 3.2 Data extraction

The screening and selection of studies followed the PRISMA statement [42]. The data extraction followed the CDC guidelines [11]; Guideline by [43] was also consulted. The extraction was performed by the reviewers (the authors), and elements included followed PICOS principles (Population, Intervention, Comparators, Outcome, and Study design). Hence the result table (Table 6 and 7) includes participant details, study design, intervention, and comparator: NFT protocol, mu rhythm, and other waves targeted, and finally outcome. Articles were read multiple times to ensure that the relevant details are captured.

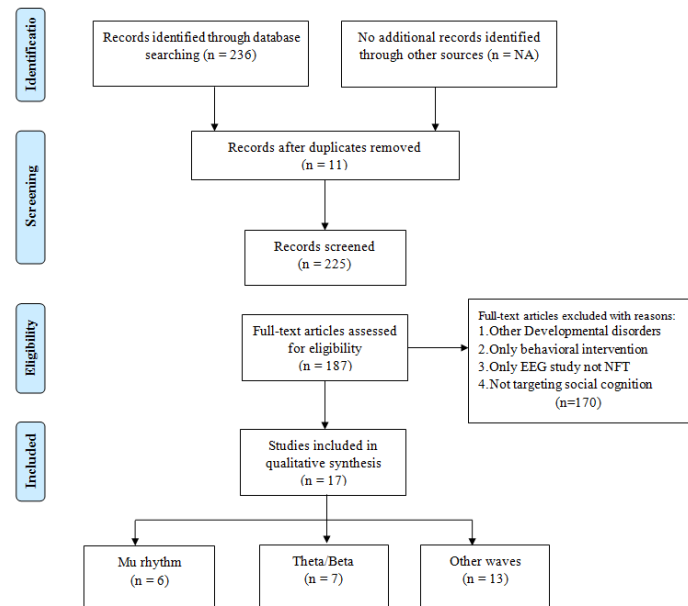


Fig. 2. PRISMA Flow Diagram

### 3.3 Quality assessment and data synthesis

After the final selection of articles, each article was independently assessed for level of evidence, methodological strength, and quality. The level of evidence was assessed by the Oxford Center for Evidence-Based Medicine (OCEBM) [44]. OCEBM give the assessment criteria in the form of a hierarchy of the likely best evidence. The latest version released in 2011 has updated the levels and ease of use. The quality assessment was conducted by using Downs and Black critical appraisal checklist [45]. The list is valid, reliable and widely recommended tool for assessment



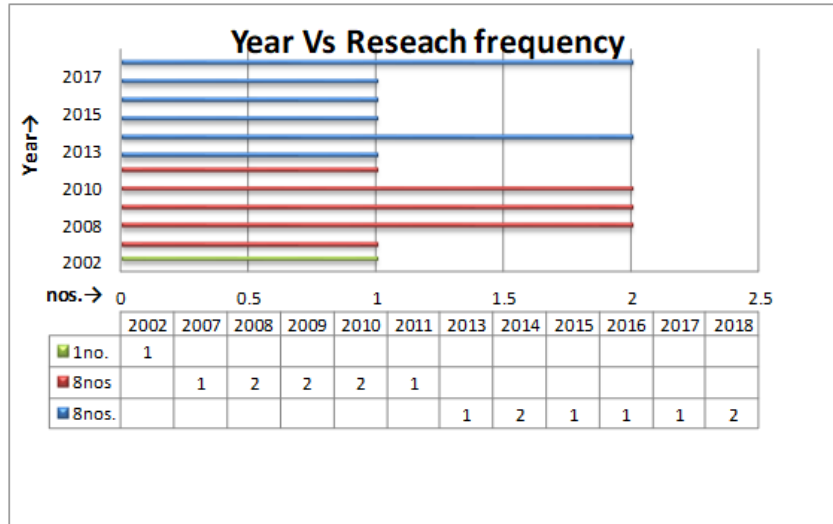
of randomized and non-randomized studies. The checklist includes 26 items and provides scores for study quality for reporting, internal validity, and external validity. The results of the quality assessment by the Downs and Black checklist and OCEBM are reported in table number 5.

**Table 5.** Downs and Black Critical Appraisal Score [45] and Oxford Level of Evidence [44]

Sl. No.	Study	Downs and Black Critical Appraisal Score						Oxford level of evidence
		Quality	External Validity	Study Bias	Selection Bias	Power	Total	
1	Jarusiewicz (2002)	7	1	5	3	5	21	3
2	Coben & Padolsky (2007)	9	0	5	3	5	22	3
3	Pineda et al (2008a)	7	3	6	6	2	24	2
4	Pineda et al (2008b)	6	1	7	6	5	25	2
5	Kouijzer et al. (2009a)	-	-	-	-	-	-	-
6	Kouijzer et al (2009b)	5	0	5	1	5	16	3
7	Kouijzer et al (2010)	7	2	5	4	5	23	2
8	Thompson, Thompson & Reid (2010)	6	1	3	0	5	15	4
9	Karimi, Hagshenas & Rostami (2011)	2	1	1	0	0	4	4
10	Pineda et al (2013)	7	0	4	1	5	17	4
11	Coben et al (2014)	9	1	4	3	5	22	3
12	Steiner et al (2014)	5	2	5	1	5	15	4
13	Friedrich et al (2015)	9	1	4	4	5	23	3
14	Hemmati et al (2016)	2	3	2	0	5	12	4
15	Datko, Pineda & Muller (2017)	8	1	4	0	5	18	18
16	Carrick et al. (2018)	7	3	6	5	5	26	2
17	Mutang, Madlan and Bahari (2018)	3	0	3	0	0	6	4

## 4 Result

From the 236 articles found in the initial searches, a total of 17 studies were eligible for inclusion in the current analysis (see PRISMA diagram; Figure 2). Even with the particular objective to find the neurofeedback intervention studies; the articles found were greatly heterogynous, ranging from randomized matched control design to single case studies. The year-wise frequency for published empirical studies can be seen in figure 3 given below:



**Fig. 2.** Showing year-wise frequency of empirical NFT studies on social cognition deficit in ASD

As the CDC [11] recommends that the studies with the non-randomized design are not sufficient and suitable for meta-analysis or quantitative data synthesis, the present study develops on and provides the narrative synthesis of data. The data is narratively synthesized is presented below.

#### 4.1 Study design

As mentioned above, the studies are very heterogeneous in design. Out of 17 studies, only four studies were randomized and matched control studies, whereas, four studies were non-randomized matched, control group. With less methodological rigor but convenient to available resources there were three between subject group study, 3 were within-subject pre-posttest studies, 1 was a case series, and 2 were single case studies (refer Table 6).

#### 4.2 Study participants

As per the inclusion criteria, the studies on ASD population only were selected. Among 17 studies only one study by Thompson and colleagues [46] specifically provided the sample details of several participants with Asperger’s syndrome and several other ASD. All other studies have provided the total ASD detail only. The study by Pineda and team [47] and Datko and his colleagues [48] recruited typically developed individuals as a comparison group; all other studies have divided the ASD participants in control or waitlist or placebo group. The placebo effect was explored by Pineda and colleagues in both of their studies [49]. Majority of studies took

precaution in recruiting participants for NFT when no other treatment was going concurrently. Otherwise, it was reported in the article (refer Table 6).

**Table 6.** Study design and participants' details

S.No	Author	Study Type	Participant Detail	Study Design
1.	Jarusiewicz 2002	intervention study	12 ASD Children, matched and divided in Control and Experimental Group	Matched Control
2.	Coben and Padolsky 2007 comp. group	Intervention Study	37 ASD children in experimental and 12 matched ASD children in controls froup	Matched Control
3.	Pineda et al. 2008a	Intervention Study	8 HF ASD males randomly assigned in experimental (n=5) and placebo (n=3) condition	Randomized Matched Control
4.	Pineda et al. 2008b	Intervention Study	19 HF ASD participants randomly assigned in experimental (n=10, males) and placebo (n=9, 3 females) condition	Randomized Matched Control
5.	Kouijzer et al. 2009a	Intervention Study	14 HF ASD participants in experimental (n=7) and waitlist control (n=7)	Non-randomized matched waitlist control group
6.	Kouijzer et al. 2009b	Follow-up study		
7.	Kouijzer et al. 2010	Intervention and Follow-up study	20 HF ASD randomly assigned in the treatment and control group	Randomized Matched Control
8.	Thompson et al. 2010	Longitudinal intervention study	159 participants (Asperger's Syndrome =150, ASD = 9)	Case series
9.	Kairimi et al. 2011	Intervention study	A six-year-old boy with ASD	Single case study
10.	Pineda et al. 2013	Intervention study	13 ASD (10 males), 11 (7 males) TD participants	Between Subject Control group
11..	Coben et al. 2014	Intervention study and imaging study	37 ASD participants (31 males), 12 waitlist control group (10 males)	Waitlist control group
12.	Steiner et al. 2014	Intervention study	10 ASD participants (9 males)	pre-post test
13.	Friedrich et al. 2015	Intervention study	N=13 (12 males) ASD participants	Between-group pre-post test
14.	Hemmati et al. 2016	Intervention study	26 ASD participants	pre-post test
15.	Datko et al. 2017	Intervention and imaging study	17 HF ASD(13 males), 11 TD (7 males)	pre-post test
16.	Carrick et al. 2018	Intervention study	34 ASD (28 males) randomly assigned in the control and experimental group	randomized matched control pre-post test
17.	Mutang, Madlan and Bahari 2018	Intervention study	12-year-old boy with high functioning ASD	Single case study

### 4.3 Study protocols

Majority of studies on social cognition reported here are done by targeting the theta/beta protocol (n=8) and mu rhythm (n=6). The theta/beta protocol was used in different variations from inhibition of excessive theta alone, or in combination to enhancing beta or to increase the theta/beta ratio. Some of the scientists' targeted mu rhythm in their studies [49], [47], [50] and [48]. Thompson and colleagues [46] targeted SMR and beta spindles which are very similar to the mu protocol. Other than these protocols, Coben & Padolsky [37] targeted reducing hyper-connectivity and Coben, and colleagues again conducted a study to reduce hyper coherence [31]. Some studies [31], [48] provided additional imaging information with eLORETA and fMRI techniques. The protocol wise distribution of study can be seen in figure 4 given below:

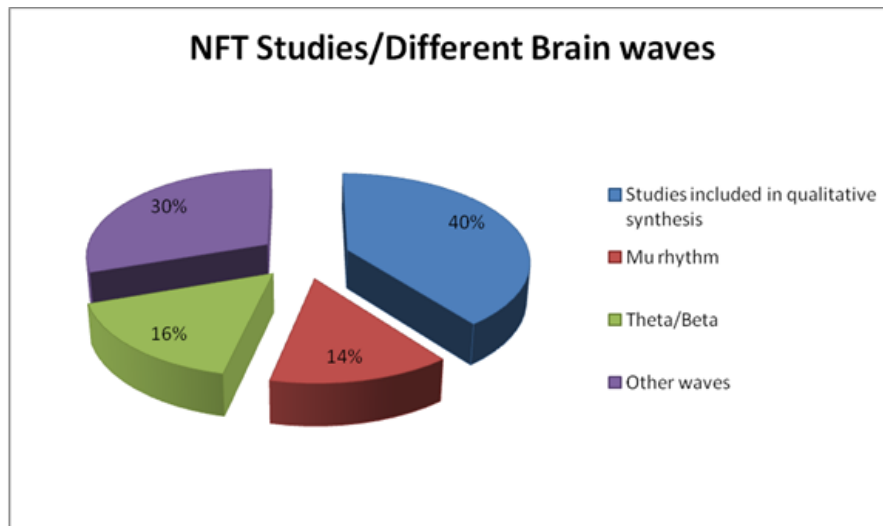


Fig. 3. Showing protocol-wise distribution of studies included in the analysis

### 4.4 Study measures

The autism treatment evaluation checklist (ATEC), behavior rating inventory of executive function (BRIEF), social responsiveness scale (SRS) and parental inputs were the most common tools used for the assessment of symptoms and more specifically social cognition process. The ATEC is a 77 item diagnostic tool developed by Rimland and colleagues [51]. The scale covers speech/language/communication, sociability, sensory/cognitive awareness, and behavior areas. The BRIEF was initially developed by Gioia and colleagues [52]. Currently, it provides the assessment on three significant indexes, inhibitory self-control, flexibility, and emergent metacognition. A composite score as a global composite score for the executive functioning is also generated. The SRS is a very commonly used tool for

social cognition measurement, which was developed by Bölte and colleagues [53]. The scale covers areas of social awareness, social cognition, social communication, social motivation, and restricted interest and repetitive behavior.

**Table 7.** Study Protocols, Measures and Key Findings

S.No	Author	Protocol	Measures used	Key Findings
1.	Jarusiewicz 2002	Customized protocol guided by EEG Spectrum	ATEC, FEAS, The Othmer Assessment	Improvement of ATEC, Cognitive awareness, socialization.
2.	Coben and Padolsky 2007 comp. group	Reducing hyper-connectivity	ATEC, GADS, GARS, BRIEF, PIC-2	Reduced cerebral hyper-connectivity was significantly associated with clinical improvements
3.	Pineda et al. 2008a	Mu rhythm	TOVA, The Apraxia Imitation Scale, ATEC	Increased control on mu rhythm and increased sustained attention, imitation ability and cognitive awareness in the experimental group
4.	Pineda et al. 2008b	Mu rhythm	ADI-R, ADOG-G, WASI, MSI, TOVA, The Apraxia Imitation Scale, ATEC	The decrease in amplitude but increase in phase coherence in mu rhythms, improvement in sustained attention and ATEC in the experimental group
5.	Kouijzer et al. 2009a	Inhibition of theta and rewarding beta over the right hemisphere	CCR-2, Auti-R, parental interview	A linear decrease in theta power, increase in beta power, improvement in attention control, cognitive flexibility, social behavior, and communication
6.	Kouijzer et al. 2009b			Maintenance of improvement after 12-month follow-up
7.	Kouijzer et al. 2010	Inhibition of excessive theta, QEEG guided for amplitude and electrode location	Social Behavior: SCQ, SRS, and CCC-2 Executive Function: TOSSA, Stroop TesT, TMT, MCST, TOL	Reduction of excessive power in 60% of participants, improvement in reciprocal social interaction, communication skill, and set-shifting. Maintenance of improvement in the 6-month follow-up.
8.	Thompson et al. 2010	Decreasing slow wave and beta spindling and increasing SMR	TOVA, IVA, Australian Scale for AS, Conner's Global Index, ADD-Q, Wide range Achievement Scale, Wechsler Intelligence Scale	The decrease in symptoms of Asperger's, improvement in social, intellectual and academic performance.
9.	Kairimi et al. 2011	Increasing slow wave 4-7 Hz	Atieh Center Primary Assessment Questionnaire	Normalized activity for 4-7 Hz, reduction in high beta and aggressive behavior, improvement in memory and social communication.
10.	Pineda et al. 2013	Rewarding mu rhythm (8-12 Hz) at C4 and inhibiting theta (4-8 Hz) and beta (13-30 Hz)	SRS, ATEC, Vineland-II	Normalization of dysfunctional MNS and improvement on social awareness, communication, and motivation in the ASD group but not in the TD group
11.	Coben et al. 2014	QEEG guided, eLORETA	GARDS, GADS, and diagnostic interview	Hypothesized neurophysiologic change and reduction in autistic symptoms (social

		supplemented hypercoherence reduction		awareness, communication, social interaction and restricted pattern of behavior) followed by NFT
12.	Steiner et al. 2014	Increasing theta/beta ratio	PERMO, CPT, CRS3-P, ASRS and CARS	improvement in attentional control and social and communication subscale of ASRS
13.	Friedrich et al. 2015	Mu suppression and enhancement	MSI, RMET, Emotion Imitation Task, and parental assessment on VABS	Learned to control mu rhythm by suppression and enhancement both, improvement in emotional responsiveness and imitation in a social situation
14.	Hemmati et al. 2016	Enhancing theta activity	CARS	Theta enhancement and improved social communication
15.	Datko et al. 2017	Mu-NFT, imitation related brain activation	Autistic symptom assessment by ADOS, ADI, WASI, outcome assessment by ATEC, SRS and Imitation task in fMRI	The positive social cognitive behavioral effect, neurophysiological changes in imitation related brain areas.
16.	Carrick et al. 2018	Reduction of abnormal EEG pattern in the delta, theta, and beta	QEEG, Posturography, ATEC, SRS-2, BRIEF, ABC, QABF	Improvement toward normalization in QEEG, sociability, ABC ad QABF measure in the active group
17.	Mutang, Madlan and Bahari 2018	Enhancing beta and inhibiting theta	ATEC	Improvement in speech and sociability and enhancement of beta

#### 4.5 Key findings

The basic premise of neurofeedback is that the intervention leads to changes in the EEG signals which in turn produces changes in the behavior. However many of the studies are not able to show the changes in both the parameters (i.e., EEG and behavioral changes). After reviewing the past literature [2] suggested the parameters for the validation of neurofeedback studies, which is: There should be spectral effects within the trained frequency band caused by the training (trainability). These spectral changes should not affect other frequency bands (independence). Finally, it is reasonable to choose a frequency band that is associated with certain functions to increase the probability of reliable behavioral effects as well as applicability (interpretability).

In the present analysis, 11 out of 17 studies reported changes in targeted frequency and associated behavioral and social cognitive changes. Thus 11 studies [37], [49],[38],[58],[54], [55],[50], [56], [48] and [57] satisfy the trainability and interpretability criteria by Zoefel and colleagues [2]. In the absence of sufficient data and quantitative analysis, it is not possible to comment on the independence criteria of Zoefel, Huster, and Herrmann (2011). All 17 studies noted an improvement in several outcome measures. The improvement in social cognitive domains were noted in cognitive/social awareness [40],[49], [47]; socialization [40], [38], [58], [46] and [59]; communication [38], [58], [54], [55], [47], [31], [60], [50], [56], and imitation [49], [50] and [48]. In other cognitive domains, the improvement was reported in attention

[49], [38], [58] and [60] and cognitive flexibility [38], [58] and [54]. It should be noted that though the majority of the studies were methodologically sound (see table 5, Downs and Black critical appraisal score [45] and Oxford level of evidence [44], and reported improvement; this assertion cannot be supported here with conviction. As the heterogeneity of the studies does not allow for quantitative analysis of the pre-post comparison.

## **5 Discussion**

The present systematic review specifically targeted the social cognition deficit and hence from the first study conducted in 2002 (by Jarusiewicz) [40] until the studies conducted in 2018 (17 year time period) is covered here. The review of available literature yielded a total of 17 empirical intervention studies specifically for ASD population. The results indicate moderate support for evidence-based treatment of social cognitive deficit.

Since 2002, the studies in this area has gradually progressed. To best of our knowledge Jarusiewicz (2002) [40] study was the first study which explored the NFT intervention for ASD patients and targeted social cognitive symptoms. Coben & Padolsky [37] also commented that till 2007 Jarusiewicz's study was the only controlled study documenting the effectiveness of neurofeedback for Autism. Jarusiewicz [40] studies did an empirical study on 12 ASD in matched control design. The study does not name that it targets mu rhythm, but fifty-seven percent of participant received protocol with rewarding 10-13 Hz on C4. The study also targeted symptom related to cognitive awareness and socialization which are part of social cognition. Coben & Padolsky [37] extended Jarusiewicz's [40] study on 37 ASD children.

Pineda et al. [49] did the first intervention study which specifically explored mu rhythm protocol for behavioral symptoms in ASD population. The second study by Pineda et al. [49] was the methodological modification of their first study. Further, a study by Pineda and group [47] was the first one which specifically explored the effectiveness of mu rhythm NFT for improvement of social cognitive impairments (Mu- MNS-Social Cognitive Deficit Hypothesis). Datko and colleagues [48] examined the effect of sensory-motor mu based NFT on imitation based brain activation. This study was the first study which directly localizes the effect of mu-NFT on brain regions involved in action observation and imitation by using fMRI.

This methodological progress adds value to the improvement in symptoms and deficits reported in the studies. However, the heterogeneity of the studies in terms of design, follow-up, and reporting of details are still the limitations.

The popularity of NFT intervention is partly due to its highly individualistic approach; a few of the studies analyzed here also have reported QEEG based intervention designing. Still looking at the studies, it seems that theta/beta and mu rhythm protocol are the most popular one for targeting social cognition deficit.

The typical approach and practices applied in recruiting the participants, assignment to the group and assessment measures help in comparison but also are

indicating the limitations of studies in this area. It is noteworthy that very few studies have done the follow-up assessment and thus it is not possible to provide evidence for the longevity of improvement. Similarly only Pineda and colleagues have explored the placebo effect which is pertinent for NFT like interventions to answer situational motivations.

NFT interventions are novel, high engaging (mostly computer based) and susceptible to biases, given these, the non-randomized design without any follow-up with the majority of studies limits the applicability and generalizability of the findings. Few limitations are part of the review process itself. As both the reviewers are new to this field, there is a possibility of errors in comprehension, interpretation, assessment, and analysis of studies.

As per the present analysis, it is possible to conclude that NFT intervention with theta/beta and mu rhythm protocol shows promising evidence for improvement in social cognition deficit, specifically in ASD. However, this conclusion should be interpreted with caution because of the limitations of the study design and result reporting (very few studies gave full details of participants, follow-up data and actual p-values). Additionally, none of the studies reported any adverse effect.

## 6 Conclusion

Overall, majority of studies satisfied the trainability and interpretability criteria of Zoefel and colleagues work [2], thus providing moderate evidence for improvement in social cognition deficit following NFT with theta/beta and mu rhythm protocol in ASD population. However, future studies need to follow a more rigorous study design (randomized control group design) with an assessment of placebo effect and longevity of improvement (follow-up). A thorough description of participants' details, no. of sessions, feedback details, and analysis (p-value) should also be reported to determine the dose-related responses.

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