Functional Aspects of Autism Spectrum Disorder Review
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INTRODUCTION

Autism spectrum disorder (ASD) is a developmental disorder categorized by deficits in social communication and repetitive and stereotyped interests and behaviors [1]. Autism is among the most enigmatic disorders of child development, with a dramatic increase in prevalence from 1 in 500(0.20%) and incidence rate is approximately 1 in 90,666 in 2018(according to Rehabilitation council of India) [2]. While the global burden of ASD is currently unknown, in the United States, the annual societal cost of the condition was recently predicted to be $126 billion and $34 billion in the UK [3].

Currently, one of the most burdensome complaints among parents of children with autism is disrupted sleep, with more than 40–80% of children experiencing sleep problems, compared with 25–40% in typically developing children (TYP) [4,5].

The neuropathological basis of autism has not been determined, and much of the work has focused on autism due to dysfunction of mesolimbic (dopaminergic) brain areas (ventromedial prefrontal cortex, medial temporal lobe, striatum and limbic thalamus) because damage to these brain regions can cause features of autism (impaired social and emotional functioning, stereotyped behaviors, mannerisms and obsession) [8]. This hypothesis is supported by studies which have reported that (i) in animals, social deficits and stereotypical behavior are associated with damage to the medial temporal lobe in infancy [7] (ii) in humans, autistic-type patterns of behavior are associated with abnormalities in the temporal lobe caused by other neurodevelopmental disorders (e.g. tuberous sclerosis) [8] and (iii) individuals with autism are impaired on 'frontal' executive tasks [9]. Non-limbic areas such as the parietal lobe have also been suggested as important in etiology because the inattention of children with autism to salient social cues resembles inattention and neglect following parietal lobe damage [10]. Some
children with autism are also impaired on neurological tests sensitive to parietal dysfunction [11]. Other investigators have proposed that developmental abnormalities of the cerebellum [12] or dysfunction of cerebellar–cortical serotonergic pathways are patho-aetiological factors for autism [13]. Consistent with this, acquired cerebellar lesions have been associated with deficits in social and emotional behavior, executive dysfunction and obsessionality [14].

Reviews of the literature on social deficits in autism have appeared recently [15], so the relevant studies are only summarized here. A study of Social Behavior is the earliest descriptions of the social impairment in autism [16, 17]. These take the form of clinical impressions. These include lack of “apparent affection”, withdrawal from people, lack of attention to people, noncommunicative use of language, lack of communicative gestures, treating parts of people as detached Objects, lack of eye contact, treating people as inanimate objects, lack of behavior appropriate to cultural norms, attention to the nonsocial aspects of people, lack of awareness of the feelings of to others, and lack of savories-faire [18]. This escalation and economic problem identify individuals with ASD as one of the highest priority populations for clinical research and treatment development.

The following are the list of some conducts found in autism children:

- They prefer to be alone.
- They never respond to their name and seems like deaf.
- Never show eye contact.
- Difficult in mingling with same age children.
- They do not point to ask for something.
- They never try to attract others by their activity.
- They never imitate adults’ action
- Rarely or never use gestures.
- Extreme fear
- They enjoy flapping, spinning, rotating objects
- Show extreme distress in others

Behavioral studies have shown that typically developing (TD) children inherently value and pursue social stimuli such as a hug or smile from a parent [19]. In contrast, individuals diagnosed with autism spectrum disorders (ASD) appear indifferent to faces and social interactions [20]. Clinical observations and previous studies suggest the hypothesis that early developmental dysfunction of brain pathways linking social stimuli and reward [21] lead to autistic individuals’ deficits in social and emotional reciprocity [22].

Dopaminergic projections from the ventral tegmental area (VTA) to cortico limbic regions are important in mediating the effects of reward on behavior [23], and neuroimaging studies have shown that neural activity in regions of the brain where dopaminergic neurons project, including the ventral medial prefrontal cortex, ventral striatum, posterior cingulate and precuneus, are modulated by eye contact, a social reward signal [24]. Dysfunctions in this pathway that may contribute to the lack of social motivation in ASD have also been previously explored using behavioral [25], event-related potential (ERP), event-related potential (ERP) [26] and structural imaging studies [27].

Our brain is endowed with the ability to detect and respond to simple social signals such as eye contact, as well as to infer from more complex behaviors intrinsically social qualities of other people such as fairness or cooperation. Individuals suffering from high-functioning autism spectrum disorders (HFASD), a neurodevelopmental disorder, are impaired in understanding social cues and in responding to them. These patients generally have normal language or general intellectual abilities, yet in everyday life they avoid eye contact and do not spontaneously interact with people [28]. On formal tests of social cognitive skill, they show specific impairments in understanding the intentions of others and lack of fast intuitive judgments about social contexts [29]. The pathogenesis of autism is unclear, although mutations in genes implicated in synaptogenesis have been identified [30] and different neurochemical, neurophysiological, and neuropathological abnormalities have been demonstrated in these patients [31]. An interesting current hypothesis has implicated oxytocin in the etiology of autism, and in the social disorders that are the hallmark of HF-ASD [32].

There are many kinds of treatment available for autism such as Behavior and communication approaches, dietary approaches and Medication, Complementary and alternative medicines. [33] Applied Behavior analysis, Physical therapy improves gross motor skills and helps to handle sensory integration issues [34]. Occupational therapy helps to treat sensory issues, sensory integration therapy, speech therapy improves communication skills.

Epidemiological survey of handicapped children in the London revealed that social impairment is not restricted to autism but is also found among other mentally handicapped people [35]. They found that 21.2 of every 10,000 children aged under 15 years in the area exposed impairments of reciprocal interaction and, of these, 4.9 had a history of typical autism. Furthermore, they found that the social impairment could be distinguished into three types: social aloofness, passive interaction, and active-but-odd interaction. This latter description referred to social behavior that was undertaken mainly to indulge some repetitive, idiosyncratic preoccupation, showing no interest in the other person’s needs. Pharmacological treatment can help ameliorate some of the behavioral symptoms of ASD, including irritability, aggression and self-injuries.
behavior. Additionally, by reduce interfering disruptive behavior.

Social adaptation requires specific cognitive and emotional competences. Individuals with high-functioning autism or with asperger syndrome cannot understand or engage in social situations despite preserved intellectual abilities. Recently, it has been suggested that oxytocin, a hormone known to promote mother-infant bonds, may be implicated in the social deficit of autism. The behavioral effects of oxytocin with autism, in a simulated ball game where participants interacted with fictitious partners, found that after oxytocin inhalation, patients exhibited stronger interactions with the most socially cooperative partner and reported enhanced feelings of trust and preference. Also, during free viewing of pictures of faces, oxytocin selectively increased patients’ gazing time on the socially informative region of the face, namely the eyes. Thus, under oxytocin, patients respond more strongly to others and exhibit more appropriate social behavior and affect, suggesting a therapeutic potential of oxytocin through its action on a core dimension of autism [36]. Risperidone is the first FDA approved medication for the treatment of symptomatic condition associate with ASD children and adolescence, including aggressive behavior deliberate self-injury and temper tantrums.

Loneliness and friendship were examined in 22 high-functioning children with autism and 19 typically developing children equated with the autistic children for IQ, CA, gender, mother’s education, and ethnicity. Children between the ages of 8 and 14 were asked to report on both their understanding and feelings of loneliness and the quality of their friendship. Compared to typically developing children, children with autism were both lonelier and had less complete understandings of loneliness. Although all children with autism reported having at least one friend, the quality of their friendships was poorer in terms of companionship, security, and help. Fewer associations were found between loneliness and friendship for the autistic than for the non-autistic children, suggesting less understanding of the relation between loneliness and friendship. Implications of these results are discussed for conceptualizing the social deficits in autism [37].

Thus, the finding that lesions to discrete brain areas may result in clinical symptoms that are also present in people with autism recommends a neurobiological basis and implicates dysfunction of the mesolimbic areas, parietal cortex and cerebellum. However, the studies only provide partial insight into the biological basis of autistic disorder.

OUTCOME

The main goal of Autism Research is to learn more about, what causes Autism and to develop drugs that can improve the quality of life of people living with this ailment in different levels. The prevalence of Autism is increased significantly, this increase may be largely attributed to broader diagnostic criteria and one of the key goals is to enable a new level of research that was not possible previously on animal studies. Receiving an accurate Autism spectrum diagnosis at younger age is associated with more positive functional outcomes in later life as a result of ASD diagnosis and receipt of the above targeted treatment. Since pragmatically there is no amble evidence in support of Autism care and permanent cure.

The above situation inspired me to contribute something constructively to address the society afflicted by this disorder which is not adequately analyzed hitherto considering the density of this disorder and mental agony encountered by the parents’ siblings of the Autism affected kids is inexplicable. Here again according to my understanding Autism is more prone among boys as compared with girls (4:1) ratio remarkably seemingly alarming hence requiring at most care to be attached to identify remedial medicines are explored in a quickest pace to reduce further Autism penetration across the globe.

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REFERENCES


