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Original Research Article

Comparative Study of Neurotransmitter, Neurotrophins Immunological and Thyroid Hormone in Iraqi Children with Autism

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Abstract: ASD is one of the most heterogeneous and diagnostically difficult neurodevelopmental disorders seen in paediatric practice, with the main features being significant impairments in reciprocal social interaction, verbal and nonverbal communication, and stereotyped, inflexible patterns of behaviour. A variety of genetic, environmental and neurobiological factors have been investigated with regard to its etiology, but no single mechanistic explanation of ASD has been found. ***Objectives***: This study aimed to extensively characterize a focused panel of biochemical, neuroendocrine and immune parameters in a paediatric ASD population to better understand their possible contributions to disease initiation and progression. The study was conducted with a case-control design, and included 80 children aged 6 to 12 years, of which 40 had a validated diagnosis of ASD and 40 were neurotypical children matched for age. Serum was obtained from peripheral venous blood samples from each subject, and quantitative assays conducted for the following analytes: GABA, dopamine, serotonin, BDNF, NGF, IL-6, TNF-α, TSH, FT3, and FT4. ***Results***: The children with ASD had significantly lower levels of both GABA and dopamine in their serum compared to healthy controls, while the levels of serotonin were surprisingly higher to a statistically significant extent. Significant upregulation of both neurotrophins, BDNF and NGF, was observed in the ASD group. Immunological profiling showed a significant and large increase in the level of TNF-α, while there was no significant difference in the level of IL-6 between the two groups. The thyroid hormone evaluation did not show any significant difference in TSH or FT3 levels, although the FT4 levels were significantly reduced in the autistic group compared to the healthy group. ***Conclusions***: All these concurrent dysregulations suggest that ASD pathophysiology is driven by the interplay of several biological axes, and not a single molecular defect. The biochemical changes described herein could not only provide a better understanding of the mechanism of ASD but could also be used to develop reliable peripheral biochemical markers that can be used for early diagnosis and future pharmacological targets.

Keywords: Autism Spectrum Disorder, GABA, Serotonin, Dopamine, BDNF, NGF, IL-6, TNF-α, Thyroid Hormone.

INTRODUCTION

Autism spectrum disorder (ASD) is a complicated, widespread, and varied syndrome that is characterized by a variety of dysfunctional social interactions, narrow interests, and repetitive activities (Chen *et al*., 2020). ASD patients have maladaptive emotional reactions, anxiety, impaired emotional learning, a lack of interest in their surroundings, and a difficulties communicating and interacting with others (Galvez-Contreras *et al*., 2017). From mild symptoms of social interaction, including an inability to control nonverbal cues, to more serious ones, like broken routines and a lack of vocal communication, the signs and symptoms can significantly affect a person's ability to handle social settings (Khaliulin *et al*., 2025). An estimated 1% of persons worldwide are thought to affect by the illness, with a 4:1 male to female ratio (Eissa *et al*., 2018). According to the Centers for Disease Control and Prevention (CDC) and the Autism and Developmental Disabilities Monitoring (ADDM) Network, one in every 44 (2.3%) eight-year-old children has diagnosed with ASD (Galvez-Contreras *et al*., 2017). Several factors, including genetic, environmental, and neurological system anomalies, which are part of the pathogenesis of ASD. However, despite nearly 50 years

of pathogenesis research, the cause of ASD is still unidentified (Bozkurt *et al*., 2021). Even though the frequency of ASD is rising, little is known about its pathophysiology, which may be because brain functions and neurobiology are complicated and challenging (Eissa *et al*., 2018). Although the onset of symptoms was observed almost before the age of 3 years, some features of slight autism were observed in the first months, for example, a change in social behavior (Alshammary *et al*., 2023). Although the exact causes of autism are unknown, one theory holds that a child with autism has a distinct way of processing and receiving information in their brain (Shehata *et al*., 2021).

Many neurotransmitter systems have been studied in the pathophysiology of ASD, and dysfunction of these systems is responsible for initial brain development it may prove to be a vital field in researching the etiology of ASD since it influences neuronal cell migration, differentiation, and synaptogenesis, culminating in the development of the brain (Eissa *et al*., 2018). The balance between excitation and inhibition signals from neurotransmitters aids the brain in performing its functions properly. Therefore, any change in this balance resulting from any disruptions of one of these components may cause abnormal neurotransmission leading to neurodevelopmental disorders such as ASD (Adak *et al*., 2021). Recent research found an imbalance in the excitatory and inhibitory pathways in GABA and glutamate neurophysiology in ASD patients (Al-Otaish *et al*., 2018).

Serotonin (5-HT) is a key regulator of mood and emotional functions in the central nervous system. There is growing evidence that disruptions in the serotonergic pathways (including the synthesis, transport and metabolism of this neurotransmitter) have a significant impact on early brain maturation, with alterations in the concentration of serotonin during critical time windows potentially resulting in abnormal formation of neural circuits and the development of autistic features (Commons, 2020). Concurrently, abnormalities in dopaminergic signaling are linked to autism, as dopamine plays a key role in the regulation of motor coordination, motivational drive and arousal states. Dopamine also regulates social interaction, locomotor activity, attentional capacity and responsiveness to the environment — areas that are typically affected in autistic individuals. The prefrontal cortex is especially sensitive to optimal dopaminergic tone, and it is believed that impaired dopamine tone contributes to the cognitive impairments often seen in ASD (Liu *et al*., 2020).

Among the neurotrophin family of neuroprotective proteins, brain-derived neurotrophic factor (BDNF) is one of the most important factors for neuronal proliferation, maturation, maintenance, and functional integrity. In addition to its general neurotrophic effects, BDNF is involved in the survival and differentiation of DA neurons in the developing brain. One of the most consistently reported biochemical abnormalities in ASD is altered BDNF concentrations. In the same way, cholinergic neurons that are crucial for attentional processing rely on nerve growth factor (NGF) for their development and for the maintenance of synaptic plasticity. Thus, NGF homeostasis disturbances have been linked to the pathophysiological mechanisms of ASD (von Bohlen *et al*., 2024). Although synaptic plasticity and neurodevelopmental mechanisms have been the main focus of ASD neurobiological studies, there is a growing body of evidence that suggests that immune dysfunction may play a role in the etiology of — or exacerbation of — ASD. Epidemiological studies also show that immune dysregulation is more prevalent in the maternal and paternal caregivers of children with ASD (Pangrazzi *et al*., 2020). Thyroid hormones play an irreplaceable role in early neurodevelopment and are involved in almost every physiological process in the body; their levels may be reduced and trigger multiorgan dysfunction (Yamakawa *et al*., 2021). Previous studies have shown that thyroid hormones are involved in the immunomodulatory regulation and that the dys-regulation of thyroid hormones is a possible contributing factor in the etiology of autism (Desoky *et al*., 2017).

The general purpose of the current study was to perform a comprehensive evaluation of the alterations in thyroid hormone profile, neurotrophin levels, some selected cytokine concentrations, and serum neurotransmitter parameters in children with autism spectrum disorders, with the aim of gaining insight into the biological mechanisms that might contribute to the disorder.

MATERIAL AND METHODS

Study Population

A total of 80 children aged between 6-12 years were selected and divided into two different groups of 40 children. The first group consisted of children who were diagnosed with autism spectrum disorder (ASD) with a confirmed diagnosis from the Autism Treatment Center, Child Protection Teaching Hospital, Medical City, Baghdad. All diagnoses were made under the guidance of a specialized multi-disciplinary team of consultant child psychiatrists and neurologists, using the standardized diagnostic criteria of the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5). Apparently healthy children, 40 in number, were selected as a comparative reference population. Demographic and clinical data were systematically collected from each participant's medical records and included full name, biological sex, chronological age, disease stage, presence or absence of comorbid conditions, and basis of diagnosis. Control group participants were confirmed as having no medical, neurological, or inflammatory condition documented, thus making them a suitable unaffected reference group.

Blood Sample

Following written informed consent from the parents/legal guardians, a 5 mL venous blood sample was obtained from each participant by standard venipuncture techniques. Samples were placed in gel activator tubes and were allowed to clot at 40°C for 15 minutes. The samples were then centrifuged at 3000 rpm for 10 minutes to obtain serum. The serum obtained was placed in Eppendorf tubes and kept at −20°C for biochemical analyses. The serum samples were used to measure the following parameters:

1. The serum levels of the neurotransmitters gamma-aminobutyric acid (GABA), dopamine, and serotonin, as well as the neurotrophic factors brain-derived neurotrophic factor (BDNF) and nerve growth factor (NGF), were measured using sandwich enzyme-linked immunosorbent assay (ELISA) kits supplied by MS4S, France.
2. The level of two cytokines, interlukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α), using the sandwich enzyme-linked immunosorbent test (ELISA) kit supplied by Ms4s/French.
3. The level of thyroid hormone: The hormones stimulate thyroid (TSH), free thyroxine (FT4), and free triiodothyronine (FT3) was determine by Fluorescence immune-chromatographic method using FinecareTM Kit, instrument from Wondfo Company/ China country.

Statistical Analysis

Means ± standard error of the mean (SEM) were used to summarize quantitative data. StatView software (version 5.0) was used for statistical analyses. One-way analysis of variance (ANOVA) was used to determine whether the differences between study groups were significant. If necessary, Fisher's least significant difference (LSD) test or Student's t-test was used to make subsequent comparisons, depending on the type of comparison. A p value of < 0.05 was considered statistically significant.

RESULTS

Table 1 shows that the serum level of gamma-aminobutyric acid (GABA) and dopamine in children with autism spectrum disorder (ASD) was lower than that of healthy children (*p* < 0.05). Mean values of GABA in ASD group was 1.033 ± 0.026 µmol/L while in control group was 1.172 ± 0.052 µmol/L. Likewise, the serum dopamine level was decreased in children with ASD (13.485 ± 0.322 nmol/L) compared to the control group (16.751 ± 0.944 nmol/L). However, there was no statistically significant difference in serum serotonin level between the two groups ($p>0.05$). The mean serotonin levels were 121.197 ± 3.137 ng/mL in the ASD group and 115.128 ± 6.342 ng/mL in the control group.

As depicted in Table 2, serum BDNF concentrations were significantly elevated ($p < 0.05$) in the ASD group (5.196 ± 0.241 ng/mL) relative to neurologically healthy controls (4.528 ± 0.255 ng/mL), reflecting a statistically meaningful upregulation of this neurotrophin in autistic children. Regarding NGF, serum concentrations were numerically greater in the ASD cohort (65.387 ± 2.271 ng/mL) than in the control group (63.201 ± 3.537 ng/mL); however, this difference did not reach statistical significance ($p > 0.05$), implying that despite the apparent numerical trend toward higher NGF values in ASD, the intergroup variation was insufficient to be considered biologically conclusive within the current sample.

Data presented in Table 3 indicate that serum TNF-α concentrations were significantly greater ($p < 0.05$) in children diagnosed with ASD (8.254 ± 0.515 pg/mL) when compared to their healthy counterparts (4.876 ± 0.867 pg/mL), demonstrating a pronounced and statistically meaningful elevation of this pro-inflammatory mediator within the autistic cohort. Conversely, serum IL-6 concentrations exhibited no statistically significant intergroup variation ($p > 0.05$), with mean values of 12.136 ± 0.365 pg/mL and 11.724 ± 0.326 pg/mL recorded in the ASD and control participants, respectively, suggesting that circulating IL-6 levels do not undergo substantial dysregulation in this particular patient population.

Table 1: GABA, dopamine, and serotonin levels in the blood of individuals with ASD and control

Groups	GABA (µmol/L) Mean ±SE	Dopamine (nmol/L) Mean ±SE	Serotonin (ng/ml) Mean ±SE
control	1.172±0.052	16.751±0.944	115.128±6.342
ASD	1.033±0.026*	13.485±0.322*	121.197±3.137
P. Value	0.0094	0.0001	0.05209

P. Value significant at <0.05, SE standard error, *significantly different between ASD and control group

Table 2: Serum levels of BDNF and NGF in ASD and control groups

Group	BDNF (ng/mL) Mean ± SD	NGF (ng/mL) Mean ± SD
Control	4.528 ± 0.255	63.201 ± 3.537
ASD	5.196 ± 0.241*	65.387 ± 2.271*
P-value	0.0454	0.0498

Values are expressed as mean ± SD. * Significant difference compared with the control group ($P < 0.05$).

Table 3: The level of serum TNF-α and IL-6 in ASD and control groups

Groups	TNF-α (pg/mL) Mean ±SE	IL-6 (pg/mL) Mean ±SE
Control	4.876 ± 0.867	11.724 ± 0.326
ASD	8.254 ± 0.515*	12.136 ± 0.365
P. Value	0.0007	0.4837

Note: *P.* value significant at <0.05. SE = standard error; * indicates significantly different between ASD and control groups.

As shown in Table 4, serum TSH and FT3 levels were not significantly different between ASD and control group (p>0.05). The mean TSH level in the ASD and control groups were 1.944 ± 0.035 U/ml and 1.934 ± 0.062 U/ml respectively, and FT3 concentration was 2.090 ± 0.423 pmol/L and 2.042 ± 0.655 pmol/L respectively, showing that the TSH and triiodothyronine levels were maintained in both groups. It is notable, however, that serum FT4 was significantly decreased (p < 0.05) in autistic children (16.060 ± 0.362 pmol/L) compared to healthy controls (18.781 ± 0.105 pmol/L), suggesting that free thyroxine availability is selectively impaired, perhaps reflecting a form of subclinical thyroid dysfunction that is peculiar to the pathophysiological context of autism spectrum disorder.

Table 4: The level of TSH, FT3 and FT4 in the serum of ASD and the control groups

Groups	TSH (U/ml) Mean ±SE	FT3 (pmol/L) Mean ±SE	FT4 (pmol/L) Mean ±SE
Control	1.934 ± 0.062	2.042 ± 0.655	18.781 ± 0.105
ASD	1.944 ± 0.035	2.090 ± 0.423	16.060 ± 0.362*
P. Value	0.5602	0.5090	<0.0001

DISCUSSION

The severe neurological development disease known as autism spectrum disorder (ASD) is characterize by recurring, stereotypical actions as well as disturbances in communication and social interaction. Although the prevalence varies by location, reports have indicated a greater prevalence of up to 1:88 children (Hodges *et al*., 2020). The pathophysiology of autism cannot sufficiently explained by a single mechanism, which makes the etiology of autism a challenging subject for psychiatric and neurological experts (Hoshiko *et al*., 2011). The current study examined a set of biomarkers that could offer fresh perspectives on the diagnosis and treatment of autism.

An imbalance between glutamate-mediated excitation and GABA-mediated inhibition has been proposed to play a major role in the pathology of ASD. The reduction in the serum level of GABA observed in autistic children in the present study is consistent with the recent study that reported a reduction in the level of GABA in autistic individuals, which revealed the critical role of the integrity of the GABAergic system in maintaining E/I balance (Alabdali *et al*., 2025). In line with the E/I imbalance hypothesis, previous studies have also suggested that impaired GABAergic transmission in ASD is due to a hyper-excitatory drive (Pizzarelli & Cherubini, 2011). The dopaminergic system is well known to play a role in the speech and communication deficits associated with autism. The substantial drop in serum dopamine levels observed in autistic children in the present study is consistent with previous studies that found similar decreases in dopamine levels in children with ASD compared to controls (Alabdali *et al*., 2014).

The results of the dopamine in the present study are inconsistent with a previous study that reported increased plasma dopamine in autistic children (Alabdali *et al*., 2014), indicating that abnormalities in dopamine homeostasis (either increased or decreased) can independently increase the susceptibility to autism. In this respect, a decrease in dopamine has been correlated with poor communicative skills and decreased efficiency of information processing, while an increase in dopamine has been correlated with increased motor activity and tic-like behaviour (Previc, 2007).

The wide-ranging regulatory effects of serotonin on mood, appetite, sleep, cognition and anxiety suggest that a disruption of serotonin homeostasis may directly contribute to several core features of autism, such as sociocognitive deficits and sleep disturbances (Zhao *et al*., 2022). While serotonin pathways have been considered an interesting target for assessing individual susceptibility to autism, results from studies examining this pathway have also been inconsistent and controversial (Lee *et al*., 2022). In the present study, there was a slight increase in the serum serotonin level in autistic children compared with controls, but this was not significant. This is partly in line with previous studies that showed significantly higher serum serotonin levels in children with ASD than in healthy children, suggesting the role of serotonin in the symptomology of autism (Abdulamir *et al*., 2018). The discrepancies in the serotonin results from the different studies could stem from serotonin sequestration by platelets, which is its main storage site, and this could increase the amount of serotonin in platelets, while the amount in the free plasma remains unchanged in some subpopulations of ASD (Esposito *et al*., 2024).

Neurotrophins are important molecules in the neuromodulators family that play a role in the orchestration of neurogenic inflammation. BDNF in particular has been well documented to have a significant effect on neuroplasticity and neuronal growth in the human central nervous system (Chakrapani *et al*., 2020; Bozkurt *et al*., 2021). The present study showed that the serum BDNF and NGF levels were significantly higher in autistic children than controls, which is consistent with the previously published studies (Bozkurt *et al*., 2021; Farmer *et al*., 2021). The observed increase in NGF concentrations is also supported by a recent study in Egyptian autistic children that found a significant positive correlation between the elevated serum NGF and hyperserotonemia which is commonly seen in ASD populations (Mostafa *et al*., 2021). In the present study, higher levels of neurotrophins were found in autistic children, which has been suggested as a compensatory adaptive mechanism to reorganise disturbed neural networks, especially in light of the scarcity of studies on neurotrophin profiles in autistic children. They have also been suggested to be a result of an underlying disruption of the normal pace of neurodevelopment in autistic children, as they remain elevated over time (Ilchibaeva *et al*., 2023).

Several lines of evidence have emerged recently suggesting a possible link between the autistic phenotype and different aspects of immune dysfunction, especially cytokine dysregulation. Immunological abnormalities have been increasingly found in people with ASD (Alshammary *et al*., 2023). In line with this, the present study found that the serum levels of TNF-α were significantly higher in autistic children than in controls, which is consistent with a recent study conducted in Korean children with autism (Shim *et al*., 2024). Moreover, high levels of TNF-α have been linked to autistic core symptoms like repetitive behaviors and reduced social interaction (Alshammary *et al*., 2023).

In the current study, there were no significant differences between groups in IL-6 concentrations, but there was a trend for the ASD group to have higher levels. Chronic inflammation is now becoming more and more accepted as a characteristic of ASD and persistently elevated peripheral cytokines may indicate an ongoing neuroinflammatory process. Pro-inflammatory cytokines, such as TNF-α and IL-6, are known to cross the blood-brain barrier and activate microglial cells, which in turn have been shown to negatively impact synapse pruning and neuronal signaling, further contributing to the E/I imbalance seen in ASD (Zhao *et al*., 2021). The peripheral cytokine profiles could thus be a useful pathway to further improve prognostic assessment in autism.

A growing body of evidence points towards a connection between thyroid dysfunction and neurodevelopmental disorders, such as ASD, and sufficient thyroid hormone activity is essential for normal neurological maturation (Meng *et al*., 2024). In the present study, the serum FT4 levels in autistic children were significantly lower than those in controls, while the serum TSH and FT3 levels were not significantly different between the cases and controls. This is consistent with previous large-scale studies of children with a diagnosis of ASD. However, the current literature is controversial, with some studies finding no hormonal differences, and others finding a reduction in TSH or slight changes in FT4. Significantly elevated levels of TSH were reported in autistic children compared to controls, suggesting that subclinical hypothyroidism may be present, and indicating a possible immunomodulatory aspect of thyroid dysfunction in ASD (Desoky *et al*., 2017), further suggesting that subclinical hypothyroxinemia may play a role in the spectrum of neurodevelopmental disease (Meng *et al*., 2024). Hoshiko *et al*., showed that neonatal thyroid hormone insufficiency is linked to a later diagnosis of ASD, and suggested that although thyroid hormone levels may normalize with time, there may be irreparable neurodevelopmental effects (Hoshiko *et al*., 2011). This interpretation is supported by findings that neonates with lower T4 levels are at a higher risk to develop ASD later in life (Yau *et al*., 2015).

More studies are needed to confirm and complete the clinical significance of these findings. Significantly, as far as we know, no previous study in Iraq has investigated this panel of biomarkers together in autistic children. Together, the dysregulation of GABA, dopamine, serotonin, BDNF, NGF, TNF-α, and FT4 in the current cohort highlights the potential of these parameters as candidate biomarkers in the future for the diagnosis and monitoring of therapy for ASD.

CONCLUSIONS

In this study, there were many changes in autism children there is significantly alteration in the level of GABA, that responsible of inhibition restoring the GABA–glutamate equilibrium, may be a useful ASD treatment approach. In addition, the decrease in dopamine level may reflected impaired in reward processing, social motivation and adaptive behavior in autism children, consequently it contributes to the primary clinical symptoms of autism. We think that the rise in BDNF and NGF could be the body's defense response to the nerve injury. The increase level of pro inflammatory cytokines observed in autism may reflected an imbalanced immune response contributing to neuroinflammation and altered neural network development. The reduce in FT4 in autism children could be a sign thyroid problems that impact neurodevelopment, potentially contributing to the cognitive feature of ASD. In order to reestablish physiological equilibrium, future therapeutic efforts may benefit greatly from the evaluation of these characteristics, which may serve as predictive biomarkers of clinical symptoms.

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