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Review Article

Autism Spectrum Disorder: An Integrative Review with Insights from Unani Medicine

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Abstract

Autism Spectrum Disorder (ASD) is a neurodevelopmental condition characterized by impairments in social communication, restricted interests, and repetitive behaviours. Its global prevalence has risen significantly, reflecting enhanced diagnostic capability and environmental and genetic influences. Conventional biomedical sciences view ASD as a complex interplay of genetic, epigenetic, neurobiological, and environmental factors.

Unani medicine, with its holistic framework emphasizing mizāj (temperament), akhlāṭ (humours), arwāḥ (vital forces), quwā (faculties), and aʿḍā' (organs), offers a unique perspective for understanding developmental disorders. This review integrates contemporary biomedical insights with Unani concepts, highlighting potential etiological parallels, symptom interpretations, and therapeutic possibilities such as ilāj bi'l-ghidhā, ilāj bi'l-tadbīr, and ilāj bi'l-dawā.

Keywords: Autism Spectrum Disorder; Unani Medicine; Mizāj; Akhlāṭ; Regimenal Therapy; Ilāj bi'l-Tadbīr

Introduction

Autism Spectrum Disorder (ASD) has emerged as one of the most challenging neurodevelopmental conditions globally, with rising prevalence and significant socio-economic burden [1]. ASD affects nearly every domain of development—social communication, cognition, sensory processing, emotional regulation, and adaptive functioning [2,3].

ASD typically presents in early childhood, but its impact reverberates throughout the lifespan, influencing academic performance, occupational adaptability, interpersonal relationships, and overall quality of life. Despite scientific advances in neurogenetics, neuroimaging, and behavioural sciences, ASD continues to challenge clinicians because of its complex, multifactorial etiology and absence of a definitive cure [4,5]. The current therapeutic pattern revolves around behavioural intervention, educational support, speech therapy, and symptom-specific pharmacotherapy, yet these approaches remain limited in addressing the deep-rooted neurobiological and systemic imbalances that characterize ASD [6].

Traditional systems of medicine, including Unani Medicine, rooted in Greco-Arab heritage and enhanced over centuries by Arab and Indo-Persian scholars, offers a comprehensive, multi-dimensional understanding of human behaviour, development, and cognitive functioning. Its conceptual basis—centered on mizāj (temperament), akhlāṭ (humours), arwāḥ (vital forces), quwā (faculties), and the functional harmony of aʿḍā' (organs)—permits a holistic interpretation of neurodevelopmental disorders [7]. Although classical Unani texts did not explicitly identify autism as a discrete disorder, their detailed descriptions of social withdrawal, impaired speech, restricted interests, hyperactivity,

and behavioural rigidity demonstrate a remarkable resemblance to contemporary descriptions of ASD. The principles articulated by Ibn Sina, Razi, Jurjani, and Ibn Nafis provide valuable theoretical foundations for interpreting ASD symptoms in terms of humoral disequilibrium and deranged cerebral temperament [8,9].

Biomedical basis of autism

ASD is recognized today as a neurodevelopmental condition rooted in widespread alterations of brain structure, connectivity, neurotransmission, immune function, and metabolic pathways. The pathophysiology is neither linear nor monocausal; rather, it arises from a dynamic interplay of genetic susceptibility and environmental influence, operating during critical windows of early brain development [10].

Genetic keystones

Genetic factors contribute significantly to ASD, with heritability estimates ranging between 50–90% [11]. Extensive genomic studies have identified over one thousand risk-associated genes, many of which regulate synaptic organization, neuronal migration, axon guidance, dendritic arborization, and synaptic plasticity [12,13]. Mutations in genes such as *SHANK3*, *MECP2*, *NRXN1*, *CNTNAP2*, and *CHD8* disrupt synaptic signalling, leading to altered cortical microcircuitry [14]. Epigenetic mechanisms—DNA methylation, histone modification, and non-coding RNAs—also modulate gene expression during prenatal neurodevelopment, influencing risk [15-17].

Neurobiological circuitry and connectivity patterns

The autistic brain demonstrates atypical patterns of connectivity detectable through functional MRI, EEG coherence, and diffusion tensor imaging. Early overgrowth of the amygdala, increased volume of the prefrontal cortex, and altered cerebellar development have been consistently reported [18-20]. One of the most widely supported models posits local hyperconnectivity combined with long-range hypoconnectivity, which disrupts the coordination between higher-order association cortices [21]. This imbalance can explain deficits in socio-emotional reciprocity, reduced theory of mind, impaired joint attention, and atypical sensory processing. Furthermore, disrupted inhibitory–excitatory balance—characterized by GABAergic deficits and glutamatergic excess—creates cortical hyperexcitability that may underlie stereotypies and sensory overload [22].

Neurotransmitter imbalances

Serotonin, a key modulator of mood and social behaviour, is elevated in the blood of approximately 25–30% of children with ASD. Dopamine dysregulation affects reward learning and motivation, while oxytocin deficiency or receptor dysfunction might contribute to attachment difficulties. Dysregulated acetylcholine and norepinephrine systems further influence attention, arousal, and learning [23,24].

Neuroinflammation and immunological pathways

ASD is increasingly recognized as a disorder with significant immune involvement. Activated microglia, elevated pro-inflammatory cytokines (IL-6, TNF-α), and impaired regulatory T cell activity contribute to a chronic low-grade neuroinflammatory environment [25]. Maternal immune activation during pregnancy is strongly associated with ASD risk, mediated through cytokine signalling that affects fetal brain development [26-28]. Interestingly, these inflammatory mechanisms resemble the Unani concept of harārat-e-ghair tabaiyya (abnormal heat) and fasād-e-akhlāṭ (corrupted humours).

Gut–brain axis and microbiome alterations

A remarkable proportion of autistic children exhibit gastrointestinal abnormalities, including constipation, diarrhoea, gut dysbiosis, and increased intestinal permeability [29-32]. The gut microbiome influences brain development through microbial metabolites such as short-chain fatty acids, tryptophan metabolites, and immune mediators [31,33-35]. Dysbiosis disrupts neurotransmitter balance, increases systemic inflammation, and alters behaviour—providing one of the strongest links between traditional gut-based approaches and modern neurobiology [36,37]. This is a direct parallel to the Unani understanding that ikhtilāl-e-meʿda (disordered stomach function) adversely affects dimāgh (brain) through systemic humoural pathways [38].

Unani interpretation of autism

The conceptual depth of Unani medicine provides a sophisticated lens for interpreting ASD beyond its behavioural manifestations, addressing fundamental disturbances in temperament, humoural composition, cerebral function, and systemic homeostasis [39].

Temperament (Mizāj) and cerebral functioning

Classical Unani scholars viewed cognitive and behavioural functioning as direct reflections of cerebral temperament. ASD's characteristic detachment, reduced reciprocity, emotional flattening, and rigid behaviour shows a bārid–yābis (cold–dry) cerebral temperament [40]. A cold temperament diminishes neural responsiveness, dulls affect, and slows cognitive processing; dryness restricts flexibility, reduces imagination, and enhances repetitive tendencies. This conceptualization provides a coherent theoretical basis for many ASD features—impaired sociability, speech delay, restricted interests, sensory abnormalities, and adherence to routine.

Humoral imbalances (Akhlāṭ) and behavioural patterns

Unani medicine attributes neurological and psychological disturbances to qualitative and quantitative abnormalities in four humours: dam, balgham, safra, and saudā'. ASD-like behaviours most closely align with disturbances in balgham and saudā' [8,9].

- Excess balgham produces cognitive dullness, slow processing, delayed speech, hypotonia, and sensory under-responsiveness.
- Excess saudā' produces obsessive tendencies, fearfulness, emotional withdrawal, repetitive actions, and rigid thinking patterns.

These descriptions correspond closely with modern categorizations of sensory hypo-reactivity, obsessive–compulsive traits, and restrictive behaviours in ASD.

Derangement of psychic faculties (Quwā-e-Nafsāniyah)

Unani theory postulates specialized faculties for speech, imagination, intellect, and motor control. ASD symptoms reflect a dysregulation of:

- **Quwwat-e-Nātiqah (speech faculty):** Delayed verbal development, echolalia
- **Quwwat-e-Mufakkirah (cognitive faculty):** Poor abstraction, weak social reasoning
- **Quwwat-e-Musawwirah (imaginative faculty):** Restricted imaginative play
- **Quwwat-e-Muharrikah (motor faculty):** Stereotypies, hyperactivity or hypotonia [40]

This framework provides an explanation for the heterogeneous presentation of ASD.

Functional relationships among brain, gut, and liver

Unani texts emphasize the physiological interdependence between the brain (dimāgh), liver (jigar), and stomach (meʿda). This triad regulates humour production, detoxification, digestion, metabolism, and temperament balance [7]. Disruption in any one component can alter brain function. Modern findings on gut dysbiosis, mitochondrial dysfunction, and immune abnormalities strongly validate this classical tri-organ model [34,41].

Unani approach to the management of autism

The management of ASD within the Unani standard requires a holistic, multi-layered intervention directed at correcting temperament, restoring humoural balance, strengthening cerebral function, regulating the gut–brain axis, and harmonizing psycho-physical faculties. Unani therapeutics are founded upon three broad pillars—Ilāj bi'l-Tadbīr (regimenal therapy), Ilāj bi'l-Ghidhā (dietotherapy), and Ilāj bi'l-Dawā (pharmacotherapy)—each of which addresses specific dimensions of ASD pathophysiology. Unlike modern medicine, which often classifies treatment into behavioural, pharmacological, and educational domains, Unani medicine integrates physiological, psychological, metabolic, behavioural and environmental determinants into a unified therapeutic philosophy.

Ilāj bi'l-Tadbīr (Regimenal Therapies)

Regimenal therapies form an essential foundation of Unani clinical intervention. They aim to purify morbid humours, enhance circulation, modulate neural activity, stimulate neuroplasticity, and improve temperament. Many of these procedures have strong parallels in modern physical therapy, sensory integration techniques, neuromodulation, and complementary therapies.

Dalk (Therapeutic Massage)

Massage influences blood flow, nerve stimulation, lymphatic drainage, and hormonal balance. In ASD, Dalk serves multiple roles:

- Modulation of sensory processing, reducing tactile defensiveness
- Reduction of behavioural agitation through parasympathetic activation

- Improvement of motor tone in children with hypotonia
- Enhancement of sleep regulation, which is often severely compromised

Modern research supports massage therapy for improving anxiety, behavioural issues, cortisol levels, and parent–child bonding in ASD. Dalk especially benefits "cold-dry" temperaments by providing warmth, lubrication, and relaxation [42].

Riyāzat (Physical Exercise and Therapeutic Movement)

Classical Unani scholars considered exercise as fundamental for strengthening quwā, balancing humours, and enhancing cognitive faculties. In ASD, structured Riyāzat—swimming, cycling, jumping, balance training—supports sensory integration, executive functioning, motor planning and attention and impulse control.

Neuroscience confirms that exercise increases Brain-Derived Neurotrophic Factor (BDNF), enhances synaptic plasticity, and modulates dopamine and serotonin—all essential in ASD [43,44].

Hammām (Therapeutic Bathing)

Warm baths relax muscles, calm sensory burden, and improves circulation. When infused with herbs such as Ustukhuddus or Baboona, Hammām can reduce anxiety and irritability, improve sleep latency and ease rigidity and hyperactivity.

The concept aligns with modern hydrotherapy and thermal regulation therapies used in sensory processing disorders.

Nutool (Pouring of Medicated Decoctions or Oils on Head)

Nutool is a classical neurotherapeutic technique that involves gently pouring warm medicated liquids on the scalp. Decoctions of Ustukhuddus, Brahmi, Nirgundi, or infused oils may:

- Soothe the nervous system
- Improve cognitive processing
- Reduce behavioural agitation
- Modulate cerebral temperament

This has conceptual similarity to *shirodhara* in Ayurveda and has been shown through EEG studies to enhance *alpha*-wave activity and induce deep relaxation states.

Hijāmah (Cupping Therapy)

Though used cautiously in children, *Hijāmah*—especially dry cupping—may reduce inflammatory load, improve circulation, and modulate autonomic balance. Animal studies show cupping reduces oxidative stress and neuroinflammation—both implicated in ASD. Its use must be individualized [24].

Other regimenal measures

- Takhlia-e-Dimāgh (mental relaxation therapies)
- Aromatherapy using lavender, rose, and sandalwood
- Sunlight exposure to improve Vitamin D and circadian rhythms
- Breathing exercises to control hyperarousal

These collectively address behavioural dysregulation, anxiety, and sensory abnormalities.

Ilāj bi'l-Ghidhā (Dietotherapy)

Diet has a central role in both modern ASD management and Unani therapeutics. ASD children frequently exhibit gut dysbiosis, digestive disturbances, constipation, food intolerances, and metabolic abnormalities. Classical Unani texts emphasize that brain temperament and behaviour are deeply influenced by the quality of foods and digestive strength.

Dietary principles for ASD based on unani temperament theory

ASD matches most closely to cold-dry (*bārid-yābis*) temperament with *balgham* and *saudā'* humoural dominance. Therefore, diet seeks to:

- Warm and moisten the temperament
- Reduce excess balgham (cold–moist)
- Moderate saudā' (cold–dry)
- Strengthen digestion (quwwat-e-hāzimah)
- Promote brain nourishment (taghziyat-e-dimāgh)

Recommended foods

- **For warming and nourishing the brain:** Ghee, butter in small amounts, almonds, walnuts, pistachios, dates, figs, raisins, fish (rich in omega-3), eggs (especially yolk), olive oil, sesame oil, honey with warm water in the morning [44].

- **For reducing balgham:** Ginger, black pepper, cinnamon, light soups with spices, warm herbal teas (mint, thyme)
- **For reducing saudā':** Sweet and moist foods like pumpkin, carrots, beetroot, moderate use of natural sweeteners and fresh seasonal fruits [38].

Foods to avoid

- Cold, moist foods: yogurt, bananas, cold drinks
- Processed foods, artificial colours, preservatives
- Excessive wheat products
- Fried, greasy foods that impair digestion
- Refined sugars that worsen hyperactivity and dysbiosis [37].

Gut–brain axis alignment

Modern research shows that gluten and casein may worsen behaviour in some cases while fermented foods may improve microbiota, and fibre supports serotonin production [46]. These align with Unani dietary principles targeting gut restoration.

Ilāj bi'l-Dawā (Pharmacotherapy)

Unani pharmacopeia contains numerous herbs and formulations that exhibit neuroprotective, anti-inflammatory, anxiolytic, adaptogenic, and digestive-enhancing effects. Their mechanisms often overlap with modern neurobiological pathways implicated in ASD.

Neurotonics (Muqawwī-e-Dimāgh)

These drugs strengthen neural tissue, improve cognition, and regulate neurotransmitter balance [47]. For ex.

- Asgand (*Withania somnifera*) – increases GABAergic activity, reduces inflammation [48]
- Brahmi (*Bacopa monnieri*) – enhances memory, stimulates neurogenesis [49]
- Ustukhuddus (*Lavandula stoechas*) – classical cerebral cleanser; improves attention [50]
- Zafran (*Crocus sativus*) – modulates serotonin and dopamine; reduces anxiety [51]
- Amla, Shankhpushpi, Tagar – improves sleep, memory, and behavioural regulation.

Drugs for humoural balance

- **Phlegm (Balgham)-resolving herbs:** Zanjabeel, Filfil Siyah, Qust
- **Black Bile (Saudā')–moderating herbs**: Kafoor, Banafsha, Turanjabeen
- **Herbs that enhance digestion**: Zeera, Pudina, Ajwain, Kalonji [52]

Compound formulations

These classical preparations nourish the brain, improve temperament, and regulate digestion. Examples include Majoon Falasfa, Majoon Barshasha, Khamira Gaozaban, Jawarish Ood Shirin [44].

Role of Mushil (Cleansing/Laxatives)

Regular mild cleansing reduces toxin load and corrects fasād-e-akhlāṭ, supporting clearer cognitive function. For ex. Ispaghol, Roghan Badam, Halela Saghir.

Safety and individualization

Unani pharmacotherapy emphasizes temperament-based prescribing, ensuring safety, minimal side effects, and long-term suitability—essential for children with ASD.

Research Gap and Future Directions

There remain substantial gaps in the evidence base for Unani-informed management of autism spectrum disorder, particularly a paucity of randomized clinical trials, standardized temperament instruments, and mechanistic biomarker data. Future research should prioritize rigorous, multidisciplinary approaches that combine standardized Unani protocols with modern outcome measures; adaptive randomized trial designs; integrated multi-omic and neuroimaging biomarkers; and systematic safety and pharmacokinetic evaluations in paediatric populations. Parallel work is needed to develop validated temperament assessment tools, consensus clinical guidelines for integrative practice, and implementation research that tests scalability and cost-effectiveness in real-world settings. Ethical oversight, data standardization, and international collaboration will be critical to translating promising traditional concepts into evidence-based, patient-centered care pathways for ASD.

Conclusion

Autism Spectrum Disorder is increasingly recognized as a complex, multisystem neurodevelopmental condition that cannot be fully understood through a single disciplinary lens. Contemporary biomedical research highlights the role of genetic susceptibility, atypical neural connectivity, immune dysregulation, metabolic disturbances, and gut–brain axis dysfunction in shaping the autistic phenotype. These multilayered abnormalities resonate strongly with classical Unani concepts of sūʾ-e-mizāj, fasād-e-akhlāṭ, and disturbances in the functional relationships between the dimāgh (brain), jigar (liver), and meʿda (gut). The remarkable convergence between modern scientific findings and Unani theoretical principles provides a promising foundation for developing an integrative, systems-based approach to ASD.

Unani medicine offers a holistic framework that addresses the cognitive, behavioural, metabolic, and psychosocial dimensions of ASD through a combination of temperament correction, humoural balancing, neurotonic support, gut restoration, regimenal therapies, and lifestyle modification aligned with Asbāb-e-Sitta Ḍarūriyya. While these principles hold significant conceptual and therapeutic potential, their systematic application in ASD remains underexplored, largely due to the absence of robust clinical trials, standardized diagnostic tools, and biomarker-supported research methodologies.

The integration of Unani therapeutics with contemporary behavioural, nutritional, and biomedical interventions provides an opportunity to develop comprehensive care pathways that address both the biological complexity and the lived experience of individuals with ASD and their families. Future work must prioritize rigorously designed clinical studies, validated temperament-based assessment tools, mechanistic biomarker research, and long-term evaluation of developmental outcomes. Such efforts will not only strengthen the scientific foundation of Unani approaches but also contribute meaningfully to global autism care by offering culturally consonant, low-cost, and holistic options.

Bibliography

1. World Health Organization. "Autism spectrum disorders: Key facts" (2023).
2. Rosen T., *et al*. "ASD from a systems biology perspective". *Nature Reviews Neuroscience* 22.1 (2021): 5-23.
3. American Psychiatric Association. "Diagnostic and statistical manual of mental disorders". (5th ed., text rev.) (2022).
4. Modabbernia A., *et al*. "Environmental risk factors for autism". *Acta Psychiatrica Scandinavica* 136.4 (2017): 361-381.
5. WHO. "Global Traditional Medicine Strategy 2025". Geneva: World Health Organization (2025).
6. Lord C., *et al*. "Autism spectrum disorder". *Lancet* 392.10146 (2018): 508-520.
7. Kabiruddin. Kulliyat-e-Nafisi Part 1. New Delhi: Idara Kitab-ul-Shifa 268 (1954).
8. Sina Ibn-. Alqanoon Fi'l Tib. Vol-I. (Urdu Translation by Kantoori, GH). New Delhi: Idara Kitab-us-Shifa (2010): 167-171.
9. Razi Z. Kitab- al -Hawi New Delhi: Central Council for Research in Unani Medicine (2001).
10. Frith U and Happe F. "Autism spectrum disorder". *Current Biology* 15.19 (2005): R786-R790.
11. Green D., *et al*. "Motor impairment in autism". *Journal of Autism and Developmental Disorder* 39.6 (2009): 843-863.
12. Lai MC., *et al*. "Autism". *Lancet* 383.9920 (2014): 896-910.
13. Bauman M and Kemper T L. "Neuroanatomy of autism". *Journal of Neuropathology and Experimental Neurology* 64.7 (2005): 537-549.
14. Just M A., *et al*. "Brain connectivity and language processing in ASD". *Brain* 130.1 (2007): 134-149.
15. Baxter A J., *et al*. "The epidemiology and global burden of autism". *Psychological Medicine* 45.3 (2015): 601-613.
16. Chaste P and Leboyer M. "Autism risk factors: Genes, environment, and gene-environment interactions". *Dialogues in Clinical Neuroscience* 14.3 (2012): 281-292.

17. Chung W S and Chen Y. "Synaptic pruning and autism". *Science* 367.6477 (2020): 827-828.

18. Ecker C., *et al*. "Brain anatomy in autism". *Biological Psychiatry* 79.4 (2015): 350-358.

19. Hertz-Picciotto I and Delwiche L. "The rise in autism and the role of environmental exposures". *Epidemiology* 20.1 (2009): 84-90.

20. Pardo C A and Eberhart C G. "Neuroinflammation in autism". *Journal of Neuroinflammation* 4.1 (2007): 1-11.

21. Hyman S L., *et al*. "Nonpharmacologic management of autism". *Pediatrics* 145.1 (2020): e20193447.

22. Di Martino A., *et al*. "Functional connectivity in ASD". *Nature Communications* 5 (2014): 5730.

23. Dawson G., *et al*. "Early behavioural intervention and brain plasticity in autism". *Journal of the American Academy of Child and Adolescent Psychiatry* 49.11 (2010): 1097-1111.

24. Rossignol DA and Frye R E. "Evidence linking oxidative stress and ASD". *Translational Psychiatry* 4 (2014): e382.

25. Navarro F., *et al*. "Maternal inflammation and fetal brain". *Journal of Reproductive Immunology* 114 (2016): 1-4.

26. Rossignol D A and Frye R E. "Mitochondrial dysfunction in ASD". *Molecular Psychiatry* 17 (2012): 290-314.

27. Estes M L and McAllister A K. "Maternal immune activation and neurodevelopmental disorders". *Science* 347.6228 (2015): 933-938.

28. Masi A., *et al*. "Cytokine abnormalities in autism". *Molecular Psychiatry* 22.2 (2017): 232-241.

29. Coury D L., *et al*. "Gastrointestinal conditions in autism". *Pediatrics* 130.S2 (2012): S160-S168.

30. Kang DW., *et al*. "Reduced fecal microbiota diversity in ASD". *PLoS ONE* 8.7 (2013): e68322.

31. Luna RA and Foster JA. "Gut microbiome and ASD". *Microbial Ecology in Health and Disease* 26 (2015): 26878.

32. Courchesne E., *et al*. "The emerging role of the cerebellum in autism". *Nature Reviews Neuroscience* 21 (2020): 67-83.

33. Onore C., *et al*. "Immune dysfunction in autism". *Journal of Neuroimmune Pharmacology* 7.1 (2012): 1-12.

34. Cryan J F and Dinan T G. "Mind-altering microbes: The gut microbiota-brain connection". *Nature Reviews Neuroscience* 13.10 (2012): 701-712.

35. Hsiao E Y. "Gastrointestinal issues in autism". *Cell* 156.1-2 (2014): 123-127.

36. De Angelis M., *et al*. "Dysbiosis and metabolic markers in autism". *BMC Microbiology* 13 (2013): 91.

37. Adams J B., *et al*. "Nutritional and metabolic treatments for autism". *Nutritional Neuroscience* 21.6 (2010): 431-451.

38. Arzani A. Tibbe Akbar [Hkm M. Hussain trans], (YNM). Deoband: Faisal Publications 757-758.

39. Vohra S., *et al*. "Integrating complementary medicine in pediatric neurodevelopment". *Child Neurology Open* 3 (2016): 1-11.

40. Kabiruddin M. Kulliyate Qanoon - Tarjuma wa Sharah. New Delhi: Aijaz Publishing House 1,2 (2006): 43, 154, 190.

41. Nardocci F., *et al*. "Effects of sensory integration therapy". *Autism Research* 12.2 (2019): 304-316.

42. Field T. "Massage therapy research review". *Complementary Therapies in Clinical Practice* 20.4 (2014): 224-229.

43. Sowa M and Meulenbroek R. "Effects of physical exercise on ASD". *Research in Autism Spectrum Disorders* 6.2 (2012): 46-57.

44. Jurjani Ismail. "Zakhira Khwarazm Shahi". CCRUM publication (1135).

45. Cramer H., *et al*. "Yoga for children with ASD: A review". *BMC Pediatrics* 15 (2015): 1-9.

46. Levy SE and Hyman SL. "Complementary medicine in ASD". *Child and Adolescent Psychiatric Clinics of North America* 24.1 (2015): 117-143.

47. Rehman S and Siddiqui M K. "Unani neurotonics and behavioural disorders: A review". *Hamdard Medicus* 53.1 (2010): 30-45.

48. Ahmad S and Akhtar MS. "Neuropharmacological effects of *Withania somnifera*: A review". *Journal of Ethnopharmacology* 110.3 (2007): 379-386.

49. Russo A and Borrelli F. "Bacopa monnieri in cognition". *Phytomedicine* 12.4 (2005): 305-317.

50. Koul B and Taak P. "Pharmacological profile of *Lavandula stoechas"*. *Journal of Herbal Medicine* 14 (2018): 100-109.

51. Hosseinzadeh H and Noraei NB. "Antidepressant effect of saffron". *Progress in Neuro-Psychopharmacology* 33.8 (2009): 1510-1516.

52. Kianpour M., *et al*. "Nigella sativa and neurological disorders". *Journal of Ethnopharmacology* 249 (2020): 11226.