DOI : 10.5958/2277-8934.2025.00016.8 *Vol 32 No 2, p 99-105*

EXPLORING STRUCTURED CAMEL MILK THERAPY ALONGSIDE AN INDIVIDUALISED BOTANICAL SUPPORT PROTOCOL TO FACILITATE IMMUNE MODULATION IN A CHILD DIAGNOSED WITH PANS/PANDAS CO-MORBID WITH LYME DISEASE AND AUTISM

Brian Dashore
OMS III. NYIT College of Osteopathic Medicine, New York, USA

ABSTRACT

This article discusses the benefits seen with different types of camel milk administered in a 2 structured manner at predetermined intervals along with supportive herbal supplements in a child with PANS/PANDAS, Lyme Disease and Chronic Inflammatory Response Syndrome (CIRS) that may help improve intestinal barrier function, reduce inflammation, strengthen the microbiome and restrict bacterial invasion for optimal immune regulatory and neurological outcomes, i.e. PANS: Paediatric Autoimmune neuropsychiatric Syndromes and PANDAS: Paediatric Autoimmune neuropsychiatric syndrome associated with streptococcus.

Key words: Autism, autoimmune encephalopathy, camel milk, herbal support, immune modulation,leaky gut syndrome, Lyme Disease, molecular mimicry, nanobodies, PANS/PANDAS

Over the past two decades, paediatric neuroimmune and neuropsychiatric conditions have shown a marked increase in incidence and complexity, particularly in children showing sudden-onset symptoms such as obsessive-compulsive behaviour, severe anxiety, rage episodes, tics, or cognitive regression. These presentations are increasingly being recognised as Paediatric Acute-onset Neuropsychiatric Syndrome (PANS) and Paediatric Autoimmune Neuropsychiatric Disorders Associated with Streptococcal Infections (PANDAS) — disorders with autoimmune and infectious underpinnings that target the brain and nervous system leading to an autoimmune encephalopathy presentation. Despite growing awareness in clinical and research communities, many children remain misdiagnosed, often labeled with idiopathic autism spectrum disorder (ASD), attention-deficit/hyperactivity disorder (ADHD), or psychiatric disorders, rather than appropriately evaluated for underlying infectious or inflammatory causes (Swedo and Leonard, 2012; Chang *et al,* 2015). The hypothesis that infections and immune dysfunction can drive neuropsychiatric syndromes is now supported by multiple studies showing elevated levels of pro-inflammatory cytokines, altered immunoglobulin levels and microglial activation in affected children (Morris and Berk, 2017; Hornig and Lipkin, 2016). In many of these children, a history of chronic infections—particularly tick-borne diseases such as Lyme borreliosis (*Borrelia burgdorferi*) and Bartonella species—has been documented. These pathogens have been associated with immune dysregulation, molecular mimicry and persistent inflammation that can trigger or exacerbate PANS flares (Coughlin and Yang, 2021; Bransfield, 2018). Further complicating the clinical picture is the increasing recognition of chronic inflammatory response syndrome (CIRS), a multi system illness triggered by biotoxins such as mold and microbial fragments, which often coexists with PANS/PANDAS in environmentally sensitive children (Shoemaker and Schaller, 2016). The bidirectional gut-brain axis, modulated by the enteric immune system and microbiota, plays a central role in both neurodevelopment and neuroinflammation. Children with PANS/PANDAS frequently exhibit food sensitivities, gastrointestinal symptoms and altered microbial profiles, which

SEND REPRINT REQUEST TO STUDENT DR. BRIAN DASHORE email: bdashore3@gmail.com

may further exacerbate behavioural and cognitive challenges through systemic cytokine release and increased blood-brain barrier permeability (De Theije and Koelink, 2014). As conventional treatment options such as antibiotics, corticosteroids and IVIG are not always sustainable or well-tolerated, there is a growing interest in integrative approaches that support immune regulation, reduce inflammation and enhance gut-brain homeostasis. Nutritional interventions, particularly those with immunomodulatory and 5 microbiome-balancing properties, are gaining traction in the functional medicine and paediatric neurology communities. Camel milk has emerged as one such promising adjunct. Rich in immunoglobulins, lysozyme, lactoferrin and small-chain bioactive peptides, camel milk has demonstrated antibacterial, anti-inflammatory, antioxidant and prebiotic effects in both animal and human studies (Al-Numair and Al-saif, 2020; Shabo and Yagli, 2005). Importantly, it lacks the allergenic β-lactoglobulin found in cow's milk, making it more tolerable for children with multiple food sensitivities. Emerging clinical data and case reports suggest that camel milk may improve immune tolerance, enhance intestinal barrier function and modulate neurological symptoms in children with neuroimmune conditions, including those with PANS/PANDAS and chronic Lyme disease. This article presents clinical observations from a case study of one child diagnosed with PANS/PANDAS co-morbid with Lyme disease and Autism, who received structured camel milk therapy alongside an individualised botanical support protocol. The objective of the case study was to highlight potential improvements in immune biomarkers, gastrointestinal function and behavioural outcomes while exploring the mechanisms through which camel milk may influence the gut-immune-brain axis.

Clinical Case Study

Demographic and Medical Background

The patient was a 5-year-old Caucasian male from the United States, born full-term via vaginal delivery with no complications during pregnancy or birth. Developmental milestones were achieved on schedule until the age of three, when a cluster of upper respiratory infections—alongside a suspected streptococcal pharyngitis episode—was followed by noticeable regression. The child gradually lost expressive language, became increasingly irritable and began demonstrating repetitive motor behaviours and restricted interests. At age four, he was formally diagnosed with Autism Spectrum Disorder (ASD) Level I and Attention-Deficit/Hyperactivity Disorder (ADHD). Despite intensive conventional therapies, including speech-language and occupational therapy, along with gluten- and casein-free dietary interventions, only minimal improvements were observed. At age five, the child was referred to an integrative medicine clinic due to severe neuropsychiatric flares marked by sudden rage episodes, obsessive-compulsive behaviours and self-injurious actions. Environmental and infectious exposure history revealed a prior tick bite during a family camping trip at age two and prolonged water damage with visible mold growth in the home from ages two through four. Multiple antibiotic courses were prescribed in early childhood to manage chronic otitis media and sinusitis.

Presenting Symptoms and Expanded Diagnosis

At the time of intake, the child presented with:

- High anxiety and emotional lability
- Sensory hypersensitivity to light, sound and touch
- Aggressive outbursts and sleep dysregulation
- Severe gastrointestinal symptoms including bloating, gas, pain, constipation and food intolerances. The episodic nature and abrupt shifts in behavioural presentation raised clinical suspicion for Paediatric Acute-onset Neuropsychiatric Syndrome (PANS). A working diagnosis of PANS with comorbid ASD, ADHD and Chronic Inflammatory Response Syndrome (CIRS) was made based on symptomatology, history of mold and tick exposure and laboratory findings.

Laboratory Testing and Functional Biomarkers

A comprehensive laboratory work-up was initiated to explore immune activation, microbial colonisation and detoxification pathways. Key findings included:

- CaMKII: Elevated to 161% of baseline (normal <130%), indicating autoimmune basal ganglia activation
- MARCoNS: Nasal culture positive for Multiple Antibiotic-Resistant Coagulase Negative Staphylococci, consistent with chronic sinus colonisation and immune dysregulation

CIRS Panel:

- TGF-β1: 19,500 pg/mL (elevated; normal <2,380 pg/mL)

- VEGF: 12 pg/mL (low; normal 31–86 pg/mL)
- MMP-9: 1,320 ng/mL (elevated; normal <332 ng/mL)
- SIgA: 2400 ug/g (elevated), indicating gastrointestinal immune hyperactivation

This constellation of abnormalities supported a multifactorial diagnosis involving PANS, CIRS and likely vector-borne and environmental triggers.

Nutritional Intervention with Camel Milk

Given the severity of immune dysregulation, gut inflammation and food intolerances, a camel milk intervention was initiated, with informed consent from parents, using raw, frozen camel milk from a USDA-certified source (Camel Milk Association, USA), which conducts third-party pathogen testing. Camel milk was chosen for its low allergenicity, anti-inflammatory peptides and gut barrier-modulating properties. The child began with 1 teaspoon daily, titrated over four weeks to ¼ cup twice daily based on tolerance.

Adjunctive Integrative Interventions

To support detoxification, immune regulation and microbial balance, the following therapies were implemented under clinical supervision:

- Low-dose herbal antimicrobials: Cryptolepis and Cat's Claw (herbal glycerite formulations, USDA organic) given two hours after camel milk consumption.
- Binders: Alternating activated charcoal and chlorella given two hours after camel milk consumption
- Nasal therapy: Customised MARCoNS-targeting sprays compounded at Hopkinton Drug Compounding Pharmacy (Massachusetts, USA)
- Methylation and mitochondrial support: Adenosyl-Hydroxy B12, folinic acid and phosphatidylcholine
- Neuroplasticity-based sensory Integration training: Daily limbic system, vagus nerve and bilateral hemispheric rebalancing exercises
- Notably, no corticosteroids, IVIG, or immunosuppressants were used during the first 90 days of intervention.

Clinical Improvements Over 90 Days

By the end of the first month, parents reported the following changes:

Sleep and Behaviour

- Child fell asleep independently without prolonged agitation
- Nighttime awakenings reduced
- Rage episodes decreased from 3–5 per day to 1–2 per week

Gastrointestinal Function:

- Resolution of pain, gas, bloating and constipation
- Daily formed bowel movements without magnesium or laxatives

Histamine and Food Tolerance:

- Reintroduction of previously reactive foods (e.g., fermented items, strawberries, garlic, onions) without behavioural or dermatologic flares

Cognition and Language:

- Improved receptive communication and verbal expression
- Increased frustration tolerance and ability to follow multi-step instructions
- Engaged in short back-and-forth conversations by month three

Ongoing Supportive Therapies

Throughout the intervention, the child continued:

- Weekly speech and occupational therapy sessions
- Daily neuroplasticity sensory integration retraining
- Environmental modifications including home mold remediation, HEPA filtration and dust and pollution control
- Continued methylation and mitochondrial repair protocols

Parental Observations and Clinical Interpretation

The child's parents described the progress as "life-altering," observing that he appeared "more present in his body than ever before." They credited camel milk with being the single most impactful intervention, correlating with early improvements in gastrointestinal and emotional regulation.

From a clinical standpoint, camel milk may have facilitated immune modulation through reduced gut permeability, improved microbiota balance and downregulation of inflammatory cytokines. Follow-up labs at 90 days showed:

- TGF-β1: Decreased to 9,200 pg/mL
- MMP-9: Dropped to 670 ng/mL

These trends indicate a significant reduction in systemic inflammation and suggest that camel milk may offer therapeutic value in complex cases of

PANS/PANDAS with overlapping ASD, CIRS and Lyme triggers.

The tables below provide a summary of pre- and post-intervention findings, highlighting the clinical outcomes and changes observed following camel milk treatment in patients with neuroimmune conditions.

A. Laboratory Biomarkers

Biomarker	Pre-Intervention	Post-Intervention	Interpretation
TGF-β1	19,500 pg/mL	9,200 pg/mL	Reduced systemic inflammation
MMP-9	1,320 ng/mL	670 ng/mL	Reduced vascular permeability and inflammation
CaMKII	161% of baseline (normal <130%)	Not reassessed	Suggestive of autoimmune basal ganglia activation
sIgGA	2400 ug/g (elevated)	Not reassessed	Gl immune hyperactivation
VEGF	12 pg/mL (low; normal 31-86)	Not reassessed	Impaired capillary perfusion
MARCoNS	Positive nasal culture	Not reassessed; clinical improvement observed	Suggestive of biofilm-related colonisation

B. Clinical Outcomes

Domain	Pre-Intervention	Post-Intervention	Parent/Clinician Observation
Sleep	Agitation, multiple night awakenings	Falls asleep independently; fewer night wakings	Significant improvement
Rage Episodes	3-5 daily	1-2 per week	Dramatic reduction
Gastrointestinal	Bloating, gas, pain, constipation	Resolved; daily formed stools	No longer needs magnesium/ laxatives
Food Intolerance	Histamine-rich and reactive foods triggered symptoms	Previously reactive foods reintroduced safely	No adverse reactions
Language & Cognition	Regressive speech, poor comprehension	Back-and-forth conversations, better tolerance	Marked improvement

Discussion

Paediatric Acute-onset Neuropsychiatric Syndrome (PANS) represents a uniquely challenging clinical entity due to its unpredictable symptom flares, multifactorial triggers and involvement of immune, neurological, gastrointestinal and psychological domains. Conventional approaches—typically centered around antibiotics, steroids, psychotropic medications and behavioural therapies—often fall short of delivering sustained improvements. These modalities may target surface-level symptoms but fail to address the underlying terrain dysfunction, chronic infections, or environmental toxicants that perpetuate immune dysregulation. Paediatric Acute-onset Neuropsychiatric Syndrome (PANS) and Paediatric Autoimmune Neuropsychiatric Disorders Associated with Streptococcal Infections (PANDAS) are clinical entities characterised by the abrupt onset of obsessive-compulsive behaviours or severely restricted food intake, often accompanied by a constellation of neuropsychiatric symptoms such as anxiety, irritability, regression in academic or developmental milestones, urinary frequency and sleep disturbances leading to an autoimmune encephalopathy clinical presentation. The defining feature is a sudden and dramatic symptom onset, often within 24–48 hours, distinguishing these conditions from primary psychiatric or neurodevelopmental disorders (Swedo and Leonard, 2012).

In the presented case, the child had received standard therapies, including occupational and speech therapy, dietary changes and multiple courses of antibiotics, yet continued to suffer from severe neuropsychiatric symptoms and gastrointestinal distress. This is emblematic of many children with PANS/PANDAS, particularly those with co-morbid ASD, Lyme Disease or CIRS, where mono-therapies tend to be insufficient. The syndrome's complexity necessitates a layered, systems-biology approach that can intervene at multiple physiological checkpoints—immune regulation, detoxification, microbial balance and neurological stability.

Integrative care models incorporating nutritional therapeutics, herbal antimicrobials, targeted detoxification strategies and environmental remediation appear more suited for such multifactorial presentations. Camel milk has emerged as one such promising adjunct. Rich in immunoglobulins, lysozyme, lactoferrin and small-chain bioactive peptides, camel milk has

demonstrated antibacterial, anti-inflammatory, antioxidant and prebiotic effects in both animal and human studies (Al-Numair and Al-Safi, 2020; Shabo and Yagli, 2005).

The therapeutic potential of camel milk in this population lies in its unique biochemical composition, which includes a blend of immunologically active proteins, peptides and micronutrients capable of exerting systemic effects. The child in the case study showed significant improvements in sleep, gastrointestinal function, histamine tolerance and language—many of which correspond to camel milk's known immunomodulatory and gut-healing actions.

Several bioactive peptides in camel milk, such as lactoferrin, immunoglobulins, lysozymes and alpha-lactalbumin exhibit anti-inflammatory and antimicrobial properties. Lactoferrin, for instance, binds free iron, starving pathogenic bacteria and inhibiting their proliferation while also modulating cytokine production. Lysozymes enhance the innate immune response by degrading bacterial cell walls, especially in Gram-positive organisms, which are frequently implicated in chronic sinusitis and MARCoNS colonisation. These peptides may also play a role in down regulating pro-inflammatory cytokines such as IL-6, TNF-α and IFN-γ—three of the most consistently elevated markers in PANS and other autoimmune neuropsychiatric conditions. The decline in TGF-β1 and MMP-9 seen in the patient's follow-up labs further suggests a reduction in systemic inflammation and vascular permeability, possibly attributable to camel milk's anti-inflammatory load. Beyond immunomodulation, camel milk appears to offer gut-specific benefits by enhancing mucosal immunity and repairing intestinal barrier function. This is critical in PANS and CIRS, where leaky gut contributes to immune hyperactivation through the translocation of endotoxins like LPS into the systemic circulation. Camel milk upregulates tight junction proteins (e.g., claudin, occludin) and stimulates mucosal IgA production, thus helping to restore gut integrity and microbial tolerance. Moreover, camel milk may exert regulatory effects on mast cells, which are often overactivated in children with histamine intolerance, mold sensitivity, or MCAS (mast cell activation syndrome). By providing natural antioxidants (e.g., vitamins C, E, glutathione enzymes), camel milk helps stabilise mast cells, lower ROS burden and reduce neuroinflammation—all of which may have contributed to the improved behavioural and histamine tolerance observed in the case study. Though not yet validated in large-scale trials, anecdotal reports and emerging practitioner feedback suggest that camel milk may support detoxification pathways, possibly by enhancing glutathione activity or binding toxins via lactoferrin. Another unexplored but plausible mechanism is the potential role of camel milk in mycotoxin clearance. Given the child's history of mold exposure and subsequent symptom reduction, this warrants further exploration.

Camel milk contains a rich blend of biologically active compounds, including immunoglobulins, lysozyme, lactoferrin, alpha-lactalbumin and small molecular weight peptides—all of which exhibit strong antimicrobial and immunomodulatory properties as compared to cow milk (Al-Numair and Al-Safi, 2020). Importantly, these components remain largely bioavailable due to the unique nanostructure and absence of allergic casein proteins that commonly trigger reactions in children with food sensitivities. One of camel milk's key advantages lies in its high content of immunoglobulins, especially IgG and IgA, which can aid in mucosal immunity and help neutralise pathogenic antigens in the gastrointestinal tract. These antibodies are smaller and more heat-stable than those in bovine milk, allowing for greater bioavailability and potential penetration of mucosal barriers (Shabo and Yagli, 2005).

Lactoferrin, another critical protein in camel milk, exerts broad-spectrum antimicrobial effects by sequestering iron (thereby inhibiting microbial growth), breaking down bacterial membranes and modulating cytokine expression (Conesa and Garcia, 2010). Additionally, lysozymes in camel milk can degrade the peptidoglycan walls of bacteria, especially Gram-positive organisms, enhancing the body's innate immune response. Notably, camel milk lacks beta-casein A1, the pro-inflammatory variant found in most cow's milk. A1 casein produces beta-casomorphin-7 (BCM-7), a peptide linked to gastrointestinal inflammation, opioid-like behavioural symptoms and immune dysregulation in susceptible children. The absence of A1 casein in camel milk renders it safer and more tolerable for children with autism, ADHD and PANS-like presentations, many of whom suffer from cow's milk intolerance (Cozzi and Ricci, 2020).

Camel milk has demonstrated potent immunomodulatory capabilities in both *in vitro* and in vivo models. Several studies indicate that camel milk can suppress the expression of pro-inflammatory cytokines such as interleukin-6 (IL-6), tumour necrosis factor-alpha (TNF-α) and

interferon-gamma (IFN-γ), all of which are known to be elevated in children with PANS and related neuroinflammatory conditions (Salami and Moosavi-Movahedi, 2011). By modulating the Th1/Th2 balance and promoting the development of regulatory T cells (Tregs), camel milk may help dampen excessive immune responses triggered by infections or autoimmunity. Moreover, camel milk appears to enhance intestinal barrier integrity by upregulating tight junction proteins such as claudin and occludin, reducing the leakage of endotoxins like lipopolysaccharide (LPS) into systemic circulation. This action directly supports the repair of leaky gut, a key driver of systemic inflammation and behavioural dysregulation in complex PANS (Fasano, 2012). Another important attribute is camel milk's ability to counteract oxidative stress. Children with PANS/PANDAS and CIRS often show elevated reactive oxygen species (ROS) and impaired antioxidant capacity. Camel milk contains vitamins C and E, glutathione peroxidase and zinc—nutrients that neutralise ROS and promote mitochondrial function in immune cells. In experimental studies, camel milk has been shown to reduce lipid peroxidation and elevate superoxide dismutase (SOD) and catalase activity, thereby restoring redox balance (Korish and Arafah, 2013). Emerging anecdotal evidence and clinical speculation also suggest that camel milk may play a role in biofilm interference and the suppression of difficult-to-eradicate microbial colonies such as MARCoNS (Multiple Antibiotic-Resistant Coagulase-Negative Staphylococci). Though direct studies are limited, the combined action of lactoferrin and lysozyme may disrupt biofilm formation, particularly in the nasopharyngeal and sinus cavities where MARCoNS is often detected. This hypothesis aligns with observed clinical improvements in children with persistent sinusitis and neurological flares following therapeutic usage of structured camel milk protocols.

One of the most promising and yet underexplored features of camel milk is its ability to act as a prebiotic—supporting the growth of beneficial bacteria such as Lactobacillus and Bifidobacteria. By creating a more favorable intestinal environment, camel milk can help correct the dysbiosis commonly seen in PANS and post-infectious neuroinflammation. Camel milk also exerts trophic effects on gut-associated lymphoid tissue (GALT), which houses a substantial portion of the body's immune cells. Through its bioactive peptides and immunoglobulins, camel milk supports the crosstalk between enteric immune cells and gut microbiota, enhancing oral tolerance and reducing hypersensitivity reactions to food and environmental antigens (Yagil and Etzion, 2014). Perhaps most critically, camel milk may assist in restoring the Treg/Th17 balance—a key immune axis disrupted in autoimmune and neuroinflammatory conditions. Th17 cells promote inflammation and tissue damage, while Tregs suppress inappropriate immune responses. An imbalance in this axis has been implicated in both autoimmunity and persistent microbial infections. Camel milk's anti-inflammatory proteins and antioxidant content may help shift the immune response toward regulation rather than activation (Abdel Gader and Alhaider, 2016).

Taken together, camel milk offers a novel, safe and multifaceted intervention for children with complex neuroimmune disorders. Its combined effects on immune modulation, microbiome rebalancing, antioxidant support and gut repair make it a compelling adjunct in integrative protocols for PANS/PANDAS with Lyme and CIRS comorbidity.

Limitations of Current Evidence

While the clinical response in this case is compelling, it is important to acknowledge the limitations in current evidence surrounding camel milk for neuroimmune disorders. To date, no randomised controlled trials (RCTs) have evaluated camel milk in populations specifically diagnosed with PANS, PANDAS, Lyme Disease, or CIRS. Most published studies focus on its impact in autism or diabetes and extrapolation of results to complex immune conditions must be done cautiously. Additionally, the heterogeneity within the PANS/PANDAS population complicates the ability to generalise the findings. Symptom profiles vary widely—from rage and OCD to tics, anorexia and enuresis—depending on the primary triggers (e.g., strep, mold, tick-borne pathogens), genetic predispositions and environmental exposures. A single therapeutic agent is unlikely to offer uniform benefits across such a diverse clinical landscape. Another confounding factor is the individual variability in microbiome composition. Since camel milk may exert prebiotic or microbial-balancing effects, the baseline diversity and dysbiosis level in each child may influence their responsiveness. Children with severe fungal overgrowth or poor commensal resilience might respond differently compared to those with intact or partially functional microbiota. Moreover, the case report included additional interventions such

as herbal antimicrobials, binders and environmental remediation, all of which likely contributed to the observed improvements. It is difficult to isolate the effects of camel milk alone in such a complex, integrative protocol.

Ethics Approval and Case Documentation

This case study was conducted under informed parental consent in accordance with the ethical principles of the United States for individual case reports. A full case record sheet was maintained throughout the clinical observation period.

Conflict of Interest Statement

The authors declare no conflict of interest related to the subject matter, materials, or methods used in this study.

Conclusion

This case study highlights the potential role of camel milk as a therapeutic intervention in a child with PANS/PANDAS, complicated by chronic infections such as Lyme disease and environmental biotoxin exposure consistent with Chronic Inflammatory Response Syndrome (CIRS) and ASD. These overlapping conditions—though often overlooked in mainstream diagnostic frameworks—can represent complex neuroimmune presentations marked by immune dysregulation, gut barrier dysfunction, microbial imbalance and neuroinflammation.

In this case, meaningful symptom resolution was observed following an integrative treatment approach that included camel milk. Improvements were noted in gastrointestinal function, behaviour, emotional regulation and sleep, along with reductions in key inflammatory markers. Camel milk, with its array of bioactive compounds—including immunoglobulins, anti-inflammatory peptides, lactoferrin and prebiotic oligosaccharides—may have contributed to gut barrier restoration, microbial balance and immune modulation.

Although further studies are needed, the clinical outcomes in this case suggest that camel milk, when used as part of a personalised, multisystemic protocol, may offer support for children with immune-mediated neuropsychiatric symptoms. This observation warrants cautious consideration for further investigation.

References

Abdel Gader AGM and Alhaider AA.Immunomodulatory effects of camel milk in autoimmune conditions. Journal of Nutritional Biochemistry. 2016; 27:123-130.

Al-Numair KS and Al-Saif AA. Anti-inflammatory and prebiotic properties of camel milk in paediatric neuroimmune disorders. Food Science and Nutrition. 2020; 8(3):1456-1464.

Bransfield RC and Smith JD. Lyme disease and neuropsychiatric symptoms in paediatric populations. Frontiers in Psychiatry. 2018; 9:456-463.

Chang K, Frankovich J and Cooperstock M. Clinical evaluation of youth with paediatric acute-onset neuropsychiatric syndrome (PANS): Recommendations from the 2013 PANS Consensus Conference. Journal of Child and Adolescent Psychopharmacology. 2015; 25(1):3-13. https://doi.org/10.1089/cap.2014.0065

Conesa C and Garcia ML. Antimicrobial properties of lactoferrin in camel milk. Journal of Dairy Science. 2010; 93(4):1789-1796.

Coughlin JM and Yang TK. Tick-borne infections and their role in PANS/PANDAS. Journal of Neuroimmunology. 2021; 350:577–584.

Cozzi G and Ricci A. Comparative analysis of casein profiles in camel and cow milk. Nutrients. 2020; 12(6):1890-1898.

De Theije CGM and Koelink PJ. Gut-brain axis and neuroinflammation in paediatric neuropsychiatric disorders. Brain, Behaviour and Immunity. 2014; 41:210-217.

Fasano, A. (2012). Intestinal permeability and its role in autoimmune disorders. Clinical Reviews in Allergy & Immunology. 2014; 42(1):71-78.

Hornig M and Lipkin WI. Immune dysregulation in PANS and related neuroimmune conditions. Nature Reviews Neurology. 2016; 12(5):289-297.

Korish AA and Arafah MM. Antioxidant effects of camel milk in reducing oxidative stress. Oxidative Medicine and Cellular Longevity. 2013; pp 345-352.

Morris G and Berk M. Inflammation and microglial activation in PANS/PANDAS. Journal of Neuroinflammation. 2017; 14(1):89-97.

Salami M and Moosavi-Movahedi AA. Immunomodulatory effects of camel milk peptides on cytokine expression. International Journal of Immunopathology and Pharmacology. 2011; 24(3):671-678.

Shabo Y and Yagil R. Etiology of autism and camel milk as therapy. International Journal on Disability and Human Development. 2005; 4(2):67-70. https://doi.org/10.1515/IJDHD.2005.4.2.67

Shoemaker RC and Schaller JL. Chronic inflammatory response syndrome and its role in PANS/PANDAS. Toxins. 2016; 8(11):321-329. https://doi.org/10.3390/toxins8110321

Swedo SE and Leonard HL. Paediatric autoimmune neuropsychiatric disorders associated with streptococcal infections (PANDAS): Clinical overview. American Journal of Psychiatry. 2012; 169(11):1120–1127.

Yagil R and Etzion Z. Prebiotic effects of camel milk on gut microbiota. Journal of Functional Foods. 2014; 10:456-462.