Saying Goodbye to *CANDJ*

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As most of our readers will know by the time this edition goes to press, this is my last edition as Editor in Chief of *CAND Journal*. It's certainly been an eventful seven and a half years; during this time, our print publication has been transformed to a fully digital, open access, peer reviewed and indexed scientific/medical publication, reflected in its new name, the *Canadian Association of Naturopathic Doctors Journal (CANDJ),* which evolved from its print predecessor, *Vital Link*.

It's been no small amount of labour to get us here, certainly, and there's no question that we would not have succeeded without the participation and support of the editors and editorial board (current and prior), and many people in senior naturopathic leadership and research who were willing to submit their clinical studies to us before we were indexed at all, as well as the numerous other colleagues who worked independently to send us case summaries, clinical reviews and commentaries on a wide variety of topics. As a scholarly journal in health care, we do not pay for content, nor do we publish interviews or book reviews, so we have relied on these colleagues to send us the results of their ongoing work for the betterment of our knowledge base, as well as the credibility of the profession as a whole. From my point of view, one of the most rewarding aspects of this work has been seeing the journal evolve into a platform that can support the continued development of naturopathic scholarship and best practices in Canada. The structures we have put in place, including open access publication, rigorous peer review, and a growing editorial community, provide a strong foundation for the next stage of the journal's growth.

While it can be easy to assume this process has a certain forward momentum, this is only because there have been many hands behind the scenes reaching out to colleagues and co-writers, making sure that the independent, credible and science-foreword voice of *CANDJ* remains so, while maintaining our unique voice championing traditional, complementary and integrative medicines (TCIM) as well as planetary health. A journal is ultimately a community effort, and I remain deeply grateful to the many authors, reviewers, editors and contributors who have given their time and expertise to help sustain this work.

Overall, running a quarterly journal for a relatively small association like ours means you are always thinking about it, always thinking ahead to the next edition, worried about late reviews, late revisions, and whether you will have enough editorial and science-based articles to interest the readers. We have worked hard to maintain a rigorous and objective, yet also positive and supportive, peer-review process, particularly for early-career authors. This can be a challenging balance to negotiate, but we've been well supported by our growing pool of reviewers, as well as the skilled professionals on our production team at SG Publishing, who are essential in helping the editors maintain our quarterly production and publication dates. As Editor in Chief, it was also my job to make sure that no copyediting or proofreading errors (including errors in tables) slipped through to publication, and I felt blessed to have such a capable staff at SG Publishing making sure that even last-minute changes to either our content or layout were accommodated quickly and seamlessly.

It's important to note that since our digital transition in 2021, we have consistently received strong, supportive feedback (both inside and outside the profession) about the high quality of our content, which, although it is not quite at the level of being ready for the most competitive medical indexing platforms, is well along that road. That says a lot for a profession that contains fewer than 5000 members nationally, and a journal that is still in its start-up phase. Although we have room to improve, I think we can be justifiably proud of our accomplishments, particularly our themed special editions in 2023 and 2025, and the creation of a comprehensive set of guidelines for the citation and use of Indigenous knowledge and Knowledge Keepers, completed in 2022.

I think that while we have challenges ahead of us, we can look back over the last 5 years in particular and be proud of what we have accomplished at *CANDJ*. And as I transition out of editorial work to focus more on writing and inter-professional collaborations (especially in the planetary health space), I look forward to seeing how the journal continues to evolve with new leadership and with new voices contributing to its pages.

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Please enjoy our contributions for this edition, and consider submitting in the future.

Warm regards,
Marianne Trevorrow, MA ND MSCP

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The Effect of Nutraceuticals on Incidence and Severity of Chemotherapy-Induced Peripheral Neuropathy: A Narrative Review

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ABSTRACT

Introduction: Chemotherapy-induced peripheral neuropathy (CIPN) is a common and debilitating side effect of cancer treatments involving platinum and taxane agents such as paclitaxel, cisplatin, and oxaliplatin. This review evaluates the efficacy of three natural health products (NHPs)—omega-3 fatty acids, l-glutamine, and melatonin—on the incidence and severity of CIPN symptoms.

Methods: A search of PubMed was conducted from inception to October 2024. Inclusion criteria were human trials assessing the effect of either omega-3s, l-glutamine or melatonin on the incidence or severity of CIPN as a primary outcome, caused by platinum and taxane chemotherapy, with outcomes measured by clinical evaluations and scoring tools.

Results: Six clinical trials met the inclusion criteria. Of the included studies, three randomized controlled trials (RCTs) examined the effect of omega-3s, one RCT and one non-randomized trial examined l-glutamine, and one open-label pilot trial assessed melatonin. The participants enrolled in the studies did not have CIPN at baseline, with NHP administration starting during chemotherapy with some extending shortly after. Therefore, studies evaluated the effect of the NHPs on the incidence or severity of CIPN symptoms. Supplementation with omega-3 fatty acids and l-glutamine demonstrated statistically significant reductions in the incidence and severity of CIPN across multiple studies involving patients on paclitaxel and oxaliplatin regimens. In a small single-arm pilot study, melatonin use was associated with a lower neuropathy incidence, compared with historical incidence rates.

Discussion: Omega-3 fatty acids and l-glutamine, when used individually, may reduce the incidence and severity of neuropathic pain while supporting nerve function. However, a small number of relevant studies were identified, including small sample sizes and methodological limitations. Future research should include more human trials and further explore supplement safety, efficacy, and underlying mechanisms to establish integrative protocols for reducing the risk and severity of chemotherapy-induced neuropathy in cancer patients.

Key Words CIPN, natural health products, orthomolecular, l-glutamine, omega-3 fatty acids, melatonin

INTRODUCTION

Chemotherapy-induced peripheral neuropathy (CIPN) is a prevalent and often debilitating side effect of various chemotherapeutic agents, particularly platinum compounds (e.g., cisplatin, oxaliplatin) and taxanes (e.g., paclitaxel, docetaxel).[1] The symptoms include sensory disturbances, such as numbness, tingling, and burning pain.[1,2] In severe cases, CIPN can also affect motor function and balance, significantly impairing quality of life and leading to chemotherapy dose reductions or discontinuation.[1] CIPN is a dose-limiting toxicity, as its development can necessitate treatment delays, dose reductions, or discontinuation, which can compromise the overall effectiveness of cancer therapy.[1] The mechanisms underlying CIPN are multifactorial and include direct neuronal toxicity, mitochondrial dysfunction, oxidative stress, and inflammation targeting the dorsal root ganglia and peripheral nerves.[2] It is estimated that 30% of patients have peripheral neuropathy a year or more after finishing chemotherapy.[1]

To mitigate symptoms of peripheral neuropathy, several neuromodulatory agents such as calcium-magnesium infusions, antiepileptic drugs like carbamazepine and gabapentin, amifostine, and glutathione have demonstrated some activity in the prophylaxis and treatment of oxaliplatin-induced acute neuropathy.[2]

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Duloxetine is commonly used off-label in oncology settings because it has been shown to reduce neuropathic pain intensity and improve quality of life in patients who already have CIPN. CIPN treatment is an off-label indication for duloxetine; however, it has not been shown to reduce the incidence or severity of neuropathy in patients undergoing chemotherapy who do not yet have symptoms.[3] Despite its high incidence, there are currently no Health Canada-approved agents for the prevention of CIPN, highlighting the need for safe and effective therapeutic options.[4] There are ongoing studies that are looking into the prevention and efficacy potential of pharmaceutical drugs.[5,6]

L-glutamine is an abundant amino acid in the human plasma and skeletal muscle and serves key metabolic and physiological functions in the body.[7] Although considered conditionally essential, its demand increases substantially under stress conditions, including cancer and chemotherapy. Glutamine is a primary energy substrate for rapidly proliferating cells, particularly enterocytes and immune cells, and supports protein synthesis, nitrogen transport, and glutathione production involved in cellular antioxidant defense.[6] In oncology care, glutamine depletion can contribute to gut mucosal atrophy, immune suppression, and treatment-related toxicity.[7] As such, glutamine supplementation has been investigated for its potential neuroprotective properties in CIPN, with proposed mechanisms including enhanced neuronal energy metabolism and protection from oxidative stress.[7] It may reduce oxidative stress by supporting glutathione synthesis and modulating inflammation through cytokine regulation.[8] Additionally, l-glutamine has been shown to preserve the structure and function of peripheral nerves by mitigating axonal damage and supporting neuronal regeneration.[8]

Omega-3 polyunsaturated fatty acids (PUFAs), primarily eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), are essential fats known for their potent anti-inflammatory and neuroprotective properties. Found abundantly in fish, omega-3s modulate the production of pro-inflammatory cytokines and are known to influence membrane fluidity and neuronal signalling.[9] In the setting of CIPN, omega-3s may attenuate neuronal damage by stabilizing nerve membranes, reducing neuroinflammation, and downregulating pathways that are implicated in neuropathic pain.[9] Oxaliplatin-induced nerve injury is driven by mitochondrial dysfunction, oxidative stress, neuroinflammation, and disruptions in neuronal membrane integrity. Omega-3s help counter these processes by incorporating into neuronal cell membranes to improve membrane stability and fluidity, reducing the production of pro-inflammatory cytokines, and supporting mitochondrial energy metabolism.[10] They also enhance the generation of specialized pro-resolving mediators that actively downregulate inflammatory signalling implicated in neuropathic pain.[10]

Melatonin is an endogenously produced hormone synthesized by the pineal gland, best known for regulating circadian rhythms and promoting sleep. Beyond its chronobiotic effects, melatonin possesses robust antioxidant and anti-inflammatory properties, making it a compelling candidate for neuroprotection.[11] It readily crosses the blood-brain barrier and has been shown to scavenge reactive oxygen species (ROS), inhibit nitric oxide production, and modulate pro-inflammatory cytokine release. In the context of CIPN, melatonin may protect peripheral nerves from oxidative and inflammatory damage induced by chemotherapeutic agents, particularly taxanes.[11]

The purpose of this review is to evaluate the efficacy of l-glutamine, melatonin and omega-3 fatty acids on the incidence and severity of CIPN in patients receiving platinum- or taxane-based agents. While several published studies have explored a wide range of NHP interventions for CIPN, this narrative review focuses specifically on these three NHPs and their clinical impact in these commonly used chemotherapy classes.[12,13,14]

METHODS

A literature search was conducted to identify relevant clinical studies evaluating the effects of omega-3 fatty acids, glutamine, and melatonin on the incidence and severity of CIPN. Databases searched included PubMed to identify clinical studies evaluating the efficacy of identified NHPs on the incidence or severity of CIPN. Participants in the included studies did not have CIPN at baseline. In this review, we selected omega-3s, glutamine, and melatonin as the interventions of interest because, after preliminary searching, they were deemed to have limited safety risk and preliminary clinical evidence in CIPN management.

Boolean operators (AND/OR) were used to combine keywords related to CIPN, specific chemotherapeutic agents (e.g., paclitaxel, oxaliplatin), and interventions (e.g., omega-3, glutamine, melatonin). Additional articles were identified via manual review of reference lists in eligible studies and relevant reviews. Data search was conducted in November 2024, with no restrictions based on the date of publication. Studies were included in this review if they met the following criteria:

1. Published in English in a peer-reviewed journal.
2. Conducted with human participants undergoing chemotherapy with either taxane- or platinum-based agents.
3. Evaluated the effects of omega-3 fatty acids, l-glutamine, or melatonin (any route of administration)
4. Incidence or severity of peripheral neuropathy was a primary outcome.
5. Employed validated clinical tools for evaluating neuropathy incidence or severity, such as reduced Total Neuropathy Score (rTNS), National Cancer Institute Common.
6. Clinical trials of any design.

All studies that met these criteria are included in the present review.

Studies were excluded if they were non-clinical in nature, including animal models, in vitro research, or narrative reviews. Observational studies, case reports, editorials, or studies not directly evaluating CIPN incidence or severity as a primary outcome were also omitted. Studies that involved other chemotherapy agents besides taxane- and platinum-based drugs were excluded. However, in colon cancer, oxaliplatin is generally not used as monotherapy, so studies involving chemotherapy for colon cancer include oxaliplatin in combination with capecitabine.

Studies were screened independently, in duplicate. Extracted data were analyzed qualitatively to assess for trends, patterns and gaps. A formal risk of bias assessment was not conducted.

RESULTS

A total of 94 studies were found in initial database searches. After applying the inclusion and exclusion criteria, 6 studies were included in the final analysis (see Table 1).

Three RCTs assessed the impact of omega-3 fatty acids on CIPN. A double-blind RCT involved 57 female participants with breast cancer, on paclitaxel chemotherapy, who were supplemented with either 640 mg of omega-3 fatty acid capsules (54% DHA, 10% EPA) or a placebo of sunflower oil soft gelatin capsules three times daily during chemotherapy, and for 1 month at the end of therapy.[9] 70% of patients (21/30) did not develop CIPN in the omega-3 fatty acids supplemented group, while 40.7% (11/27) did not develop CIPN in the placebo group. This was statistically significant with a *p* value of 0.029. In terms of severity, 13.3% of patients (4/30) developed mild peripheral neuropathy, 16.7% (5/30) developed moderate peripheral neuropathy, and 0% developed severe peripheral neuropathy in the omega-3 group. In the placebo group, 37% (10/27) developed mild peripheral neuropathy, 18.5% (n=5/27) developed moderate peripheral neuropathy, and 3.7% (1/27) developed severe peripheral neuropathy. There was no statistically significant difference in the severity of peripheral neuropathy between groups (p=0.054).[9] A double-blind RCT involving 71 patients, male and female, with stage 3 colon cancer, on oxaliplatin and capecitabine chemotherapy, were supplemented with either 640 mg of omega-3 fatty acid capsules (54% DHA, 10% EPA) or a placebo of sunflower oil capsules three times daily during chemotherapy, and for 1 month after the end of therapy.[10] 47% of patients (17/36) did not develop CIPN while supplemented with omega-3 fatty acids. In contrast, 11% of patients (4/35) in the placebo group did not develop CIPN. This difference is statistically significant, with a *p* value of 0.002. In terms of the severity of CIPN developed, in the omega-3 group, mild peripheral neuropathy was seen in 42% of patients, moderate in 3% and severe in 8%. In the placebo group, mild peripheral neuropathy was seen in 57% of patients, moderate in 20% and severe in 12%. The difference in severity of CIPN is statistically significant between groups, with a *p* value of 0.001.[10] A double-blind RCT involving 179 patients, male and female, with stage 3 or 4 colon cancer, on oxaliplatin combined with capecitabine, were supplemented with either 640 mg of omega-3 PUFAs (54% DHA, 10% EPA) or placebo capsules, three times a day during chemotherapy, and for 1 month after the end of therapy.[15] When supplemented with omega-3 fatty acids, 47.8% of patients (43/90) did not develop peripheral neuropathy. When supplemented with placebo, 30.3% of patients (27/89) did not develop peripheral neuropathy. In terms of severity, 27.8% (25/90) developed stage 1, 12.2% (11/90) developed stage 2, 6.7% (6/90) developed stage 3 and 5.6% (5/90) developed stage 4 peripheral neuropathy, when supplemented with omega-3 PUFAs. In the placebo group, 24.7% (22/89) developed stage 1, 12.4% (11/90) developed stage 2, 19.1% (17/90) developed stage 3, and 13.5% (12/90) developed stage 4 peripheral neuropathy. The difference in both incidence and severity between groups is statistically significant, with both *p* values of 0.017. There was also a significant difference in quality-of-life scores in the omega-3 PUFA group compared with the placebo group (p=0.046), and an increase in appetite loss in the placebo group (p=0.025).[15]

One RCT and one non-randomized study assessed the impact of l-glutamine supplementation on CIPN. An RCT included 86 participants with colorectal cancer undergoing chemotherapy with oxaliplatin.[7] Participants were randomized to obtain or not obtain 15 g of l-glutamine twice daily for 7 consecutive days every 2 weeks, beginning on the day of chemotherapy infusion. When supplemented with 15 g twice daily of l-glutamine, compared with the placebo, a lower percentage of grade 1–2 peripheral neuropathy was observed in the l-glutamine group (16.7% vs 38.6%, p=0.04) after two cycles of oxaliplatin treatment, and a significantly lower incidence of grade 3–4 neuropathy was noted in the l-glutamine group after four cycles (4.8% vs 18.2%, p=0.05) and six cycles (11.9% vs 31.8%, p=0.04). Additionally, interference with daily activities due to neuropathy was reported by 16.7% of patients in the l-glutamine group, compared with 40.9% in the control group (p=0.017). Oxaliplatin dose reductions due to neuropathy were necessary in 7.1% of patients in the l-glutamine group, compared with 27.3% in the control group (p=0.024).[7] In a non-randomized unblinded study, 46 patients with breast cancer on high-dose paclitaxel chemotherapy were given either 10 g of l-glutamine, three times daily, for 4 days, starting 24 hours after beginning paclitaxel treatment (n=17) or no l-glutamine supplementation (n=29).[16] L-glutamine supplementation caused significant changes in weakness, vibratory sensation and toe numbness parameters. Patients who received l-glutamine experienced significantly less weakness compared with those in the control group (p=0.02). The l-glutamine group had a significantly lower incidence of vibratory sensation loss compared with the control group (p=0.04). Lastly, patients receiving l-glutamine reported significantly less toe numbness than those who did not receive l-glutamine (p=0.004).[16]

One study assessed the impact of melatonin supplementation on CIPN. In a non-controlled, open-label, phase 2 pilot trial, 22 participants with breast cancer being treated with either paclitaxel or docetaxel were enrolled. Patients were supplemented with 21 mg of melatonin every day for the duration of taxane chemotherapy and for 28 days following completion of treatment. When supplemented with melatonin, 45% of patients (10/22) developed neuropathy. This included 23% with grade 1 neuropathy and 22% with grade 2 neuropathy; no patients developed grade 3 neuropathy. 55% of patients (12/22) reported no neuropathy. The median score on the FACT-Taxane quality-of-life assessment remained stable, with only a 0.5-point median decline from baseline to the end of the study, suggesting that melatonin did not adversely affect quality of life. The authors note a historical incidence rate of CIPN around 60%. Although this study found a lower incidence rate, given the small and uncontrolled nature, no conclusion can be drawn.[11]

Safety reporting across the included studies was limited. The melatonin study reported on serious adverse effects, which seem to

TABLE 1 Studies included in the narrative review

Authors	Sample Size and Study Design	Interventions	Comparison	Outcome Measures	Results
Ghoreishi et al., 2012	N=57 Randomized double blind placebo-controlled trial	Participants received omega-3 fatty acid capsules, 640 mg 3 times daily (totaling 1,920 mg per day), during chemotherapy and for 1 month after completing paclitaxel treatment.	Participants received placebo capsules following the same schedule.	Evaluate the presence and severity of paclitaxel-induced peripheral neuropathy based on rTNS score	**Incidence:** The study found that 70% of patients in the omega-3 group did not develop peripheral neuropathy, compared with 40.7% in the placebo group. This difference was statistically significant (p=0.029). **Severity:** There was no statistically significant difference in PN severity between the groups.
Esfahani et al., 2016	N=71 Randomized double-blind placebo-controlled trial	Patients received 640 mg of omega-3 fatty acids 3 times daily (totaling 1,920 mg per day) during chemotherapy and for 1 month after completing oxaliplatin and capecitabine treatment.	Patients received placebo capsules following the same schedule.	Incidence of oxaliplatin-induced peripheral neuropathy based on rTNS	**Incidence:** 47% of patients in the omega-3 group did not develop CIPN, compared with 11% of patients in the placebo group. This difference was statistically significant (p=0.002). **Severity:** The severity of neuropathy, assessed using the rTNS, was significantly lower in the omega-3 group compared with the placebo group (p=0.001).
Zhang et al., 2020	N=179 Double-blind RCT	Patients received 640 mg of omega-3 fatty acids 3 times daily (totaling 1,920 mg per day) during chemotherapy and for 1 month after completing oxaliplatin and capecitabine treatment.	Patients received placebo capsules following the same schedule.	Nerve conduction studies measured with a Nicolet/ VIASYS Viking Quest EMG Machine	**Incidence:** 47.8% of patients did not develop PN when supplemented with omega-3 fatty acids. 30.3% of patients did not develop PN in placebo group. This difference is statistically significant (p=0.017). **Severity:** The severity of neuropathy was significantly lower in the omega-3 group compared with placebo (p=0.017).
Wang et al., 2007	N=86 Pilot study	Patients received 15 g of oral glutamine twice daily (totaling 30 g per day) for 7 consecutive days, starting on the day of each oxaliplatin infusion.	Participants did not receive glutamine supplement.	Incidence and severity of oxaliplatin-induced peripheral neuropathy using NCI-CTC–based sensory neuropathy grades	**Incidence:** A lower percentage of grade 1–2 peripheral neuropathy was observed in the l-glutamine group (16.7% vs 38.6%, p=0.04) after 2 cycles of oxaliplatin treatment, and a significantly lower incidence of grade 3–4 neuropathy was noted in the l-glutamine group after 4 cycles (4.8% vs 18.2%, p=0.05) and 6 cycles (11.9% vs 31.8%, p=0.04). **Impact on Daily Activities**: Interference with daily activities due to neuropathy was reported by 16.7% of patients in the glutamine group, compared with 40.9% in the control group. (p=0.017). **Oxaliplatin Dose Reduction:** Dose reductions due to neuropathy were necessary in 7.1% of patients in the glutamine group, compared with 27.3% in the control group (p=0.024).
Stubblefield et al., 2005	N=46 Non-randomized study	Participants received 10 g of glutamine 3 times daily for 4 days starting 24 hours after completing paclitaxel treatment.	Participants did not receive glutamine supplement.	Severity of paclitaxel-induced peripheral neuropathy using clinical neurologic examination and patient-reported symptoms	**Weakness:** Patients who received glutamine experienced significantly less weakness compared with those in the control group (p=0.02). **Loss of Vibratory Sensation:** The glutamine group had a significantly lower incidence of vibratory sensation loss compared with the control group (p=0.04). **Toe Numbness:** Patients receiving glutamine reported significantly less toe numbness than those who did not receive glutamine (p=0.004).
Nahleh et al., 2010	N=22 Open-label, phase II pilot clinical trial	All participants received 21 mg of melatonin daily at bedtime throughout their taxane chemotherapy regimen and continued for an additional 28 days post-chemotherapy.	This was a single-arm study without a placebo or control group. The outcomes were compared with historical data on the incidence of taxane-induced neuropathy.	Incidence and severity of taxane-induced PN using NCI-CTC as well as neuropathy grade	**Incidence of Neuropathy:** 45% of patients (10 out of 22) developed neuropathy: Grade 1: 23% (5 patients) Grade 2: 23% (5 patients) No cases of Grade 3 or higher neuropathy were reported. **No Neuropathy:** 55% of patients (12 out of 22) did not experience any neuropathy. **Quality of Life:** The median score on the FACT-Taxane quality-of-life assessment remained stable, with only a 0.5-point median decline from baseline to the end of the study, suggesting that melatonin did not adversely affect quality of life.

rTNS = reduced total neuropathy score; NCI-CTC = National Cancer Institute common toxicity criteria; PN = peripheral neuropathy; CIPN = chemotherapy-induced peripheral neuropathy; FACT = functional assessment of cancer therapy.

be attributed to the chemotherapy treatment itself. This included grade 3 nausea/vomiting (n=3) and grade 3 fatigue (n=1). Adverse effects plausibly attributed to melatonin include mild nighttime sedation in two individuals, with no daytime sedation reported.[11] One of the included l-glutamine studies reported that all patients completed 6 cycles of chemotherapy, and glutamine supplementation did not adversely affect treatment response or survival, with similar chemotherapy response rates and comparable median survival times between groups. Additionally, no significant differences were observed in non-neurologic toxicities, including grade 3–4 leukopenia, thrombocytopenia, and hepatic or renal function impairment (p=0.76), indicating that oral glutamine was well tolerated.[7] No other studies reported safety outcomes of the agents used, commented on impacts on chemotherapy effectiveness or evaluated long-term efficacy beyond the active chemotherapy period.

DISCUSSION

Supplementation with omega-3 fatty acids and l-glutamine demonstrated statistically significant reductions in the incidence and severity of CIPN across multiple RCTs involving patients on paclitaxel and oxaliplatin regimens, while data for melatonin is too preliminary for reliable interpretation.

The three omega-3 RCTs included patients receiving either paclitaxel for breast cancer or oxaliplatin-based regimens for colorectal cancer, indicating that effects may extend across major neurotoxic chemotherapies. All studies used an omega-3 dose of 640 mg 3 times daily, which is a relatively low dose compared with the 2–4 g/day commonly used in clinical practice, potentially limiting the magnitude of observed effects. Across the studies, omega-3 supplementation reduced the proportion of patients developing CIPN and, in the oxaliplatin trials, reduced severity and improved quality-of-life scores. However, 2 of the oxaliplatin studies shared similar sample sizes, dosing, follow-up periods, and study protocols, raising the possibility of overlapping patient populations and limiting the independence of the evidence.[9,10,15]

For l-glutamine, evidence comes from one RCT in oxaliplatin-treated colorectal cancer patients and one non-randomized study in high-dose paclitaxel-treated breast cancer patients. The RCT demonstrated reductions in CIPN incidence and severity, decreased interference with daily activities and fewer chemotherapy dose reductions. The non-randomized, unblinded trial focused on specific signs and symptoms of CIPN, noting that l-glutamine–supplemented patients experienced less lower extremity weakness, loss of vibration and toe numbness. Dosing regimens differed between studies (15 g twice daily for 7 days every 2 weeks versus 10 g three times daily for 4 days per cycle) as well as study design, making direct comparisons challenging. Sample sizes were modest, and follow-up was limited to the active chemotherapy period, leaving long-term outcomes and optimal dosing unclear.[7,16]

The evidence for melatonin is limited. A single small, single-arm pilot study reported that over half the participants did not develop neuropathy; however, without a control group, no conclusions can be drawn regarding efficacy.[11]

Possible Mechanism of Action

Several proposed mechanisms might mediate the impact of omega-3 fatty acids on CIPN, particularly through their influence on neuronal structure, function, and inflammatory signaling. Omega-3 fatty acids integrate into neuronal cell membranes, influencing signal transduction, ion transport, receptor function, and neurotransmission. Omega-3-fatty acids, particularly DHA, help reduce neuropathic pain by directly modulating nerve activity and inhibiting proinflammatory cytokines such as IL-1β, IL-6 and TNF-α.[17] Neuroprotectin D1 (NPD1) is created from DHA, and is a neuroprotective agent. NDP1 promotes axonal regeneration and reduces expression of pro-inflammatory cytokines.[9,10,14] Omega-3 fatty acids also prevent the slowing down of nerve conduction velocity, through improving Na+/K+ ATPase activity.[9,10] Mice enriched with genes that increase omega-3 fatty acids have exhibited a greater recovery to peripheral nerve injury and reduced neuronal cell death.[17] Omega-3 fatty acid metabolites have also been shown to promote neuronal growth and function.[17] DHA contributes to greater flexibility and fluidity in cell membranes, which could allow for faster production of synaptic vesicles that deliver neurotransmitters, therefore enhancing neuronal signaling.[18] RCTs have shown that patients supplemented with omega-3 fatty acids during taxane- or platinum-based chemotherapy experienced significantly lower incidence and severity of peripheral neuropathy compared with placebo groups.[9,10] These promising outcomes suggest omega-3 supplementation may serve as a safe and effective intervention in reducing the risk and severity of CIPN and improve chemotherapy tolerance.

The mechanism of neuroprotection by l-glutamine is unclear, yet research suggests it may offer neuroprotection by upregulating nerve growth factor (NGF), which declines as neuropathy worsens. NGF administration prevents paclitaxel-induced neuropathy in mice, and l-glutamine has been shown to increase NGF mRNA in animal models.[8] Additionally, high systemic l-glutamine may reduce glutamate conversion, potentially contributing to symptom relief.[16] A common fear with l-glutamine supplementation is that it will protect tumour cells from the cytotoxic effects of chemotherapy.[7] However, several studies have refuted this claim since there were no between-group differences in chemotherapy response rates.[7,16] Some studies have shown an increased immune response with l-glutamine supplementation, hence decreased tumour growth.[16]

Melatonin may provide neuroprotection during chemotherapy through multiple mechanisms, including reducing oxidative stress, regulating mitochondrial function, and suppressing the formation of free radicals that contribute to neurotoxicity.[11] In addition to its potential role in CIPN, melatonin is widely used in oncology supportive care due to its demonstrated effects on improving treatment tolerance. Research has shown that melatonin supplementation is associated with lower rates of thrombocytopenia, malaise, asthenia, stomatitis, and cancer-related cachexia, collectively supporting improved quality of life during chemotherapy.[19] Importantly, melatonin does not appear

to interfere with the antitumour efficacy of chemotherapy agents, as clinical data have shown no reduction in response rates among patients receiving melatonin as an adjunct therapy.[11] These findings suggest that melatonin may offer a safe and biologically plausible strategy to reduce chemotherapy-related toxicities, including neuropathy.

Strengths and Limitations

A notable strength of this review is its narrowed focus on three specific NHPs, which allowed for a more detailed exploration of the evidence and proposed mechanisms of action, compared with broader reviews that evaluate broader topics. In addition, we developed a priority eligibility criterion in order to minimize selection bias.

Several limitations of this review and the available literature should be considered when interpreting the findings. First, a key limitation of this paper is its narrative review format, which lacks the methodological rigour and comprehensive search strategy of a systematic review. Second, the literature search was conducted using a single database. This raises the possibility that relevant studies may have been missed, and the findings may not fully represent the complete body of evidence on NHPs for CIPN. Third, no formal risk of bias assessment was performed to evaluate the quality of the included studies. As a result, confidence in the reported findings is limited, and methodological shortcomings in the original studies may have influenced the results. Fourth, there is a lack of human trials investigating NHPs for CIPN. Additionally, there is a lack of quality studies among those available. Many of the included studies have a small sample size, and had design flaws such as a lack of randomization or blinding, or the absence of a control or placebo group. Fifth, the studies mentioned a lack of long-term follow-up. Because of this, the influence of these NHPs on chemotherapy efficacy, survival outcomes, and sustained neuroprotective effects for CIPN remains unclear. While the available studies did not indicate any negative impact on chemotherapy dosing or response, formal assessment of long-term treatment outcomes was not performed, highlighting the need for future trials to evaluate both neuropathy prevention and potential interactions with antineoplastic therapy over time. Sixth, there was minimal reporting in the included studies on direct safety or adverse effects of the interventions themselves, limiting conclusions about their tolerability. Seventh, given the small body of evidence on this topic, there is a small number of studies included. The studies included involved patients receiving treatment for several different types of cancer with limited overlap. Furthermore, as the included studies primarily involved patients with breast and colorectal cancers, the findings may not be generalizable to other cancer types treated with taxane- or platinum-based chemotherapy. While CIPN is largely driven by the neurotoxic mechanisms of these agents rather than the underlying malignancy, variations in chemotherapy protocols, dosing schedules, and patient characteristics across cancer types may influence outcomes. Therefore, extrapolation of these results to other oncology populations should be made with caution. Lastly, we acknowledge that the included trials utilized different neuropathy assessment scales and chemotherapy regimens, which may have contributed to variability in the reported outcomes.

Further Research

While current evidence is promising, several limitations in the literature highlight important directions for future research to strengthen the clinical application of nutraceutical use in CIPN. These include higher-quality studies, with larger sample sizes and longer follow-up. Further studies can include research on the mechanism of action of these NHPs to more deeply understand their neuroprotective potential. Additionally, trials comparing these products to one another using the same study methods would allow for accurate comparison, permitting the establishment of the superiority of one therapy. A greater number of well-designed melatonin studies could also allow for a comprehensive understanding of its effectiveness and mechanism of action. Future studies are warranted to investigate NHPs across similar cancer types to strengthen comparability and clinical relevance as well. Beyond individual efficacy, these NHPs may offer additive or synergistic effects when used in combination, given their distinct neuroprotective mechanisms. Omega-3 fatty acids reduce neuroinflammation and modulate ion channels, l-glutamine supports neuronal regeneration and nerve growth factor expression, and melatonin scavenges free radicals and protects against oxidative injury. No current clinical trials have investigated combined supplementation; however, future research should explore multi-agent protocols to determine whether concurrent use provides superior protection against CIPN. Another key consideration is the variability in trial design across studies reviewed, including differences in sample sizes, dosages, treatment durations, and outcome measures, which limits direct comparison and generalizability. While these supplements are generally safe, accessible, and cost-effective, especially compared with pharmacologic agents like duloxetine, their integration into standard oncology practice faces challenges due to regulatory limitations and a lack of widespread education in integrative care. Patient-reported outcomes such as functional impairment, pain interference, and quality of life also remain underreported and should be prioritized in future trials. Including more outcome measures in further research, such as CIPN incidence, severity, and specific nerve conduction parameters, would allow for a more holistic view of the NHPs' effect.

Additionally, longer-term follow-up data are lacking, which impairs our understanding of whether these agents merely delay neuropathy onset or offer lasting neuroprotection post-chemotherapy. Future research should include more large-scale randomized human trials and further explore NHP safety, efficacy, and underlying mechanisms. Expanding research in these areas will help solidify the role of NHPs in reducing the incidence and severity of CIPN and support the development of evidence-based integrative protocols.

Clinical Application

Omega-3 fatty acids and l-glutamine show promise for reducing the incidence and severity of CIPN in patients undergoing

platinum- and/or taxane-based chemotherapy. CIPN can be a debilitating adverse effect that may necessitate a reduction in chemotherapy dose or early discontinuation. One RCT assessing l-glutamine demonstrated a lower incidence of required oxaliplatin dose reductions due to CIPN in the l-glutamine group compared with control.[7] However, this finding is limited to a single study, and long-term safety data were not assessed. Current evidence remains insufficient to conclude that supplementation with l-glutamine or omega-3 fatty acids reliably prevents chemotherapy dose reductions or interruptions. Larger, more robust clinical trials with long-term safety monitoring are required before making definitive clinical recommendations or conclusions regarding long-term outcomes.

CONCLUSION

Omega-3 fatty acids and l-glutamine, when used individually, may reduce the severity and incidence of chemotherapy-induced neuropathic pain, while supporting nerve function. Future human studies with greater sample sizes and rigorous study designs can allow for a more comprehensive analysis of these NHPs, holding the potential for the development of integrative protocols to address CIPN. Expanding available CIPN management tools has the potential to improve patient outcomes by enhancing the efficacy and tolerability of chemotherapy.

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Multimodal Naturopathic Approach to Managing Musical Tinnitus: A Case Report

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ABSTRACT

Tinnitus, including the subtype musical tinnitus, can significantly impact quality of life. Musical tinnitus involves the perception of structured musical sounds without an external source and is often more distressing than typical tinnitus due to its intrusive nature. A 73-year-old female with a 12-year history of bilateral tinnitus presented with the recent onset of musical tinnitus in her left ear. The symptom was associated with significant emotional distress, insomnia, anxiety, and nausea. A multimodal treatment plan was implemented that included natural health products containing magnesium threonate, Ginkgo biloba, ginger, and other ingredients, in combination with acupuncture and homeopathy. After 6 weeks of treatment, the patient reported substantial improvement in musical tinnitus severity, sleep quality, anxiety, and nausea. This report documents a case of musical tinnitus where naturopathic medicine provided meaningful symptom relief. Further research is needed to explore the potential role of naturopathic medicine therapies in tinnitus management.

Key Words Naturopathic medicine, acupuncture, Ginkgo biloba, magnesium threonate, GABA, case report

INTRODUCTION

Tinnitus is the perception of sound without an external auditory stimulus, commonly described as ringing, buzzing, or hissing in the ears.[1] It affects approximately 10–15% of the adult population, with about 1–2% experiencing severe distress that impacts daily functioning.[1] Musical tinnitus, also known as musical hallucination, is a less common subtype characterized by the perception of structured musical sounds without an external source. This form of tinnitus is often more distressing due to its intrusive nature and the complexity of the perceived sounds.[2]

The pathophysiology of tinnitus is complex and multifactorial, involving both peripheral and central auditory pathways. Proposed mechanisms include abnormal spontaneous activity in the auditory cortex, reduced cochlear blood flow, maladaptive neuroplasticity, and neurotransmitter imbalances.[3] Specifically, tinnitus has been associated with alterations in glutamate, gamma-aminobutyric acid (GABA), serotonin, and dopamine.[3] Excess glutamatergic activity and reduced GABAergic inhibition may contribute to hyperexcitability in the auditory cortex, while serotonergic and dopaminergic dysregulation may influence emotional distress, attention, and the salience of tinnitus.[3]

Reduced cochlear blood flow has also been implicated, as inadequate inner ear perfusion may impair oxygen and nutrient delivery to the cochlea, disrupt ion homeostasis in hair cells, and ultimately lead to auditory nerve dysfunction.[3] These vascular changes can contribute to auditory deafferentation (such as hearing loss), which then triggers central compensatory mechanisms involved in tinnitus perception.

Maladaptive neuroplasticity refers to the brain's attempt to reorganize neural pathways in response to auditory deafferentation.[3,4] In tinnitus, this reorganization is thought to cause increased synchronous firing and aberrant connectivity within auditory and non-auditory regions, including limbic and attentional networks.[3,4] Such maladaptive cortical changes heighten the persistence and intrusiveness of tinnitus perception and may explain why tinnitus often worsens with stress, anxiety, or depression.[3,4] Contributing risk factors include age-related hearing loss, social isolation, anxiety, depression, and various neurologic or otologic comorbidities.[4]

The standard treatment approach for tinnitus typically includes cognitive behavioural therapy (CBT), sound therapy, and, occasionally, pharmacological interventions. Although CBT remains the most evidence-based strategy for improving coping and quality of life, a significant proportion of individuals, an estimated 20–40%, do not achieve meaningful symptom relief and continue to experience chronic, intrusive tinnitus.[5] This treatment resistance underscores the importance of exploring novel, patient-centered treatment strategies.

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Naturopathic medicine represents one such integrative approach. Naturopathic doctors (NDs) are trained as primary care providers in accredited 4-year, graduate-level programs. Their education includes biomedical sciences, clinical diagnosis, and a broad array of therapeutic modalities, including clinical nutrition, botanical medicine, acupuncture, homeopathy, hydrotherapy, and lifestyle counseling. Naturopathic practice is guided by principles such as treating the whole person, identifying and addressing the root cause of illness, and using the least force necessary to restore health.[6,7]

Despite growing public interest in and clinical use of naturopathy, there is a notable absence of published clinical trials assessing the impact of multimodal naturopathic interventions for tinnitus, particularly in rare subtypes such as musical tinnitus. This report aims to address that gap by presenting a case of a patient with musical tinnitus who underwent a naturopathic treatment plan involving nutraceuticals, acupuncture, and homeopathy. The case highlights changes in tinnitus severity, as well as related symptoms such as sleep disturbance, anxiety, and nausea, offering preliminary insight into the potential role of naturopathic care in managing complex tinnitus presentations.

CASE PRESENTATION

Patient Information

The patient is a 73-year-old female who presented with a 12-year history of bilateral tinnitus with a recent onset of musical tinnitus in her left ear, starting in November 2024. Written informed consent was obtained from the patient for publication of this case report and any accompanying information.

Primary Concerns and Symptoms

The patient's primary concern was the sudden onset of musical tinnitus in her left ear, beginning in November 2024, after a 12-year history of stable bilateral tinnitus. The onset of the musical tinnitus was spontaneous. She reported hearing melodies that were not externally present, which led to significant emotional distress, insomnia, and interference with daily activities. The previously stable bilateral tinnitus became secondary to the new, more intrusive symptom. The patient also described experiencing poor sleep quality, averaging 3 to 4 hours per night, accompanied by anxiety, fatigue, and nausea. She reported that these symptoms were exacerbated by the emotional distress caused by the onset of musical tinnitus, further impacting her quality of life.

Medical History

The patient has experienced chronic bilateral tinnitus for the past 12 years, accompanied by mild sensorineural hearing loss. Her medical history includes unmanaged hypertension and depression that is currently being managed with psychotherapy. She has no history of noise exposure or the use of ototoxic medications, and she does not report any known allergies or other significant comorbidities. Her family history is unremarkable for conditions related to tinnitus or hearing loss. While she benefits from a supportive family structure, the emotional distress caused by her tinnitus symptoms has led to some degree of social withdrawal.

Relevant Past Interventions with Outcomes

Sound therapy was previously attempted to manage the patient's chronic bilateral tinnitus; however, it was unsuccessful in providing symptom relief. No other treatments were pursued specifically for the onset of musical tinnitus. Additionally, the patient's hypertension remains unmanaged, as she has chosen not to pursue treatment at this time.

Diagnostic Assessment

The patient was diagnosed with musical tinnitus in her left ear following a thorough clinical evaluation. She had a longstanding history of bilateral tinnitus, with the recent onset of musical tinnitus specifically affecting the left ear. An audiological evaluation revealed mild bilateral sensorineural hearing loss, which was consistent with her history, and no acute changes in hearing were detected. Otoscopic examination showed no abnormalities, and the neurological examination revealed no focal deficits, although the patient did display signs of anxiety and distress associated with her tinnitus. Imaging studies were not performed, as there were no red flags indicating the need for neuroimaging.

Therapeutic Interventions

The patient was prescribed a multimodal naturopathic treatment plan aimed at reducing the severity of musical tinnitus and alleviating associated symptoms, such as nausea and anxiety (Table 1).

The naturopathic interventions were prescribed based on a range of desired therapeutic effects. Magnesium threonate supports neural signaling, brain plasticity, and stress reduction by modulating N-methyl-D-aspartic acid (NMDA) receptor activity, which has been implicated in tinnitus pathophysiology.[8] Magnesium threonate crosses the blood–brain barrier more efficiently than other forms, increasing brain magnesium levels.[8] Magnesium acts as a natural NMDA receptor antagonist, reducing excitotoxic glutamatergic signaling and cortical hyperexcitability, which are

TABLE 1 Summary of Naturopathic Treatment Recommendations

Intervention	Ingredients/Details & Dose	Instructions
Magnesium Threonate	Magnesium Threonate: 50 mg per capsule	Take 2 capsules before bed
Combination natural health product for cognition	ALA: 125 mg Ginkgo biloba: 100 mg Bacopa extract: 37.5 mg CPC: 25 mg Vitamin B6: 6.25 mg B12: 250 mcg Folate: 200 mcg	Take 1 capsule in the morning
Ginger	Ginger rhizome extract: 250 mg Ginger rhizome: 50 mg	Take 1 capsule, as needed
GABA/ L-theanine	GABA: 300 mg L-Theanine: 150 mg	Take 2 capsules before bed (up to 4 if well tolerated)
Acupuncture	Local points: GB2, SI19, TW21 Calming points: Yintang, HT7	Twice/week for 4 weeks, then once/week ongoing
Homeopathy: Cinchona officinalis 30C	Single dose of Cinchona officinalis 30C	Administered in-office, one-time dose

key drivers in tinnitus generation.[8] It also stabilizes neuronal firing and supports synaptic plasticity, potentially counteracting maladaptive neuroplastic changes in auditory pathways.[8] In addition, a multi-nutrient cognition supplement was recommended that includes ingredients that support neurovascular function and cochlear circulation and reduce tinnitus severity.[9] Alpha-lipoic acid (ALA) reduces oxidative stress in cochlear hair cells and auditory neurons.[9] Ginkgo biloba enhances cochlear and cerebral microcirculation, improves mitochondrial function, and has neuroprotective effects.[9] Bacopa supports synaptic communication and reduces neuronal oxidative damage.[9] B-vitamins (B6, B12, folate) regulate homocysteine metabolism, support methylation-dependent neurotransmitter synthesis (serotonin, dopamine, GABA), and improve neural repair.[9] Together, these ingredients target oxidative stress, impaired circulation, and neurotransmitter dysregulation—all implicated in tinnitus pathophysiology.[9] Furthermore, a ginger supplement was given for nausea relief via serotonin (5-HT3) receptor modulation and anti-inflammatory effects, which may also help regulate auditory neurotransmission and reduce tinnitus severity.[10] Ginger's bioactive compounds (gingerols, shogaols) act as antagonists at 5-HT3 serotonin receptors, reducing nausea and abnormal serotonergic signaling.[10] Since serotonin plays a role in tinnitus perception and limbic activation, ginger may modulate serotonergic pathways that influence auditory processing.[10] Its anti-inflammatory effects further protect cochlear and neural tissues from inflammatory injury, indirectly reducing tinnitus severity.[10] GABA and L-theanine were also given to promote relaxation and cochlear microcirculation, reducing stress-induced tinnitus exacerbations by influencing the limbic and auditory cortex interactions.[11,12]

In addition to the natural health products, acupuncture was performed to improve cochlear circulation and promote parasympathetic activity and to reduce tinnitus intrusiveness.[13] Local ear points included GB2, SI19, TW21, which help stimulate blood flow and neural signaling in the auditory system. Central calming points (Yintang, HT7) activate parasympathetic pathways and modulate limbic system overactivity, which contributes to tinnitus distress.[13] Functional magnetic resonance imaging (MRI) studies suggest acupuncture influences auditory cortex activity and restores autonomic balance, thereby reducing the perception and intrusiveness of tinnitus.[13]

Lastly, a homeopathic, *Cinchona officinalis* 30C, was given. In homeopathy, *Cinchona officinalis* is classically indicated for high-pitched auditory disturbances, weakness, and nervous exhaustion.[14] While the exact mechanism is not pharmacologically established, it is hypothesized to act via the homeopathic principle of symptom resonance, potentially influencing central auditory processing or stress-related amplification of tinnitus symptoms.[14]

Follow-Up and Outcomes

At 3 weeks, the patient reported improvement in tinnitus symptoms, sleep, blood pressure, and distress as well as resolution of nausea. Because symptoms had not fully resolved, the homeopathic remedy was prescribed. At 6 weeks, the patient reported continued improvements in all of the presenting symptoms, including the severity of tinnitus, sleep, anxiety, appetite, and blood pressure (Table 2). She reported that the reduction in musical tinnitus allowed her to engage in social interactions without distress, enjoy meals again as her appetite and nausea improved, and experience restful sleep, indicating a broad enhancement in daily functioning and overall quality of life. Table 3 presents a timeline of the interventions and outcomes. The provider queried the patient about compliance at follow-up appointments. The patient reported that she took the supplements as prescribed. The patient was asked about adverse or unanticipated events; none were reported.

DISCUSSION

This report describes a case of musical tinnitus which improved following a multimodal naturopathic treatment. While the patient's bilateral tinnitus had been manageable for years, the onset of musical tinnitus in her left ear created significant distress. The treatment, including nutraceuticals, acupuncture, and homeopathy, addressed both the auditory and systemic components of the condition.

No single treatment modality has demonstrated consistent, robust effectiveness for musical tinnitus. There is preliminary evidence that nutraceuticals such as the combination product for cognition, which contains Ginkgo biloba among other neuroprotective and vasodilatory herbs, magnesium threonate, and acupuncture may offer partial benefit individually.[1,15] However,

TABLE 2 Summary of Outcomes

Outcome Measure	Before Intervention	After Intervention
Musical Tinnitus Severity	10/10	3/10
Sleep Quality	Poor; frequent awakenings. Unrefreshed sleep.	6–7 hours per night; only waking up once to urinate. Feeling refreshed upon awakening
Anxiety	High distress related to tinnitus	Reduced distress; less anxiety reported
Nausea	Daily episodes of nausea	Sustained absence of any nausea symptoms
Appetite	Reduced due to nausea and distress. Only eating one small meal a day, such as porridge, which is also difficult for her to eat.	Normal appetite.
Blood Pressure	Systolic blood pressure was consistently elevated, ranging from 140-160.	Systolic blood pressure was reduced, ranging from 120-140. Fluctuated based on her psychological stress levels.
Quality of Life	Impaired daily functioning. Isolating from everyone, unable to engage in regular activities, like walking.	Marked improvement. Socializing more with friends, going out for regular walks and exercising.

TABLE 3 Timeline

Date	Event
>12 years ago	Onset of bilateral non-musical tinnitus
November 2024	Onset of musical tinnitus in **left ear**
December 7, 2024	Intake appointment and complete physical exam: Assessment of medical history and chief complaint. Patient seeks help due to distress from musical tinnitus.
December 14, 2024	Treatment initiation of acupuncture and supplements.
January 11, 2025	3-week follow-up: Patient reports significant improvement in tinnitus symptoms, sleep quality, and blood pressure (126/72 mmHg LAS), with continued reduction in distress. Nausea symptoms resolved completely. Prescribed in-house dose of homeopathic remedy.
February 8, 2025	6-week follow-up: Patient reports continued improvement in tinnitus symptoms, sleep quality, and blood pressure (124/78 mmHg LAS), with continued reduction in distress.
March 8, 2025	Continued improvement in tinnitus symptoms, sleep quality with continued reduction in anxiety and distress. Blood pressure was 148/74 mmHg LAS due to psychological stress.
April 19, 2025	Final visit before leaving for her trip to London. Patient reports no stress, continued improvements in sleep (only waking up once to use the bathroom), and increased socialization with friends.

in combination, these therapies may offer synergistic support by acting on multiple physiological targets, including neurovascular, psychological, and auditory mechanisms, improving the likelihood of a clinically meaningful response.[1] The cognition formula may help improve cochlear blood flow and reduce oxidative stress, which could contribute to mitigating tinnitus symptoms by enhancing the vascular health of the inner ear and potentially normalizing disrupted auditory processing pathways.[1]

Magnesium is involved in neuromodulation and synaptic plasticity. Magnesium threonate, in particular, crosses the blood–brain barrier, potentially stabilizing neural excitability and addressing central auditory processing abnormalities commonly seen in tinnitus.[4] Given that the patient experienced reductions in both tinnitus severity and anxiety, it's plausible that magnesium threonate played a role in modulating central nervous system activity, particularly in areas associated with auditory processing.[4] The improvement in sleep quality and anxiety could also be related to magnesium's known role in supporting the nervous system's balance and reducing hyperactivity.[4]

Tinnitus has been linked to neural hyperactivity in auditory pathways.[3] Augmenting inhibitory neurotransmission through GABA supplementation may counteract this hyperactivity, helping to reduce the perception of tinnitus.[3] The patient's improvement in sleep quality and overall symptom management aligns with the known calming effect of GABA on the central nervous system. This suggests that the GABA supplementation may have played a key role in reducing the neuronal hyperactivity that may have been contributing to her tinnitus.[3]

Studies have shown that acupuncture can improve cochlear blood flow, activate parasympathetic nervous system activity, and reduce neural hyperactivity in the auditory cortex.[13,16] In this case, the acupuncture treatments may have contributed to the patient's symptom relief by improving circulation to the ear and reducing the neural excitability associated with tinnitus. The patient's reports of enhanced well-being following acupuncture sessions are consistent with existing literature on acupuncture's potential to modulate both peripheral and central mechanisms implicated in tinnitus, though the specific contribution of each intervention in this multimodal approach remains unclear.

Limitations

While the improvements observed in this case are promising, several limitations must be considered. First, this article describes the outcome of a single case, which inherently limits the ability to generalize the findings to a larger population. Additionally, this case involved the collection of patient-rated symptom severity; validated outcome measures could have measured changes in symptoms with more precision. As with other case reports and trials of whole practice naturopathic care, it is not possible to identify which individual interventions were responsible for the improvement in symptoms. Lastly, as in all case reports, it is possible that other factors, beyond the care provided, were responsible for the improvement in symptoms. These could include changes in stress levels or other life circumstances.

Future Research Directions

Further research is needed to better understand the physiological mechanisms underlying tinnitus and how integrative therapies, such as those used in this case, can impact these processes. Randomized controlled trials comparing multimodal naturopathic treatments, which are holistic, patient-centric and designed to address the underlying deficiencies or dysfunction using the least force possible, with standard therapies would help clarify the therapeutic effect of a combination intervention. Future research could monitor changes in blood pressure and anxiety as possible mediators of changes in tinnitus. Additionally, research exploring the specific role of each intervention, such as Ginkgo biloba, magnesium threonate, homeopathy, and acupuncture, would provide valuable insights into which treatments may be most beneficial for specific tinnitus subtypes, such as musical tinnitus.

CONCLUSION

This case suggests that a multimodal naturopathic approach, incorporating acupuncture, nutraceuticals such as GABA, ginger, and magnesium threonate, along with individualized homeopathy, may have offered symptomatic relief and enhanced quality of life for a patient with musical subtype tinnitus. Notable

PATIENT PERSPECTIVE

"I've had ringing in both ears for years, and it never really bothered me that much. But in November, when the musical tinnitus started in my left ear, I got scared. It was constant, loud, and I couldn't sleep. I felt like I was losing control. Within weeks of starting treatment, I felt calmer, the musical tinnitus wasn't always there, and even when it was, it was not as bothersome as before. I could finally rest. I'm so grateful."

improvements were observed in tinnitus severity, sleep quality, anxiety, and nausea. While encouraging, these outcomes are based on a single case and must be interpreted with caution. Further studies involving larger sample sizes and controlled methodologies are needed to better evaluate the efficacy and mechanisms of multimodal naturopathic interventions in the treatment of tinnitus.

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Naturopathic Doctors Must Speak Out Against Human Rights Atrocities in Palestine

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At the time the first draft of this article was written, people living in the Gaza Strip were dropping dead in the street from a man-made famine,[1] and from direct attacks while trying to seek limited food, mostly by the Israeli military.[2] There was plenty of aid available, but it was being withheld by the state of Israel which has held Gaza under siege since 2007,[3] a situation that has intensified since October 2023. Even since the most recent so-called ceasefire agreement of October 10, 2025, military action against Palestinians has continued, aid is restricted, and people continue to die. These deaths are not "costs of war," or "collateral damage." These are crimes inflicted by Israel against the people of Palestine, deliberate acts of genocide as determined by nearly all the major human rights organizations in the world, including the United Nations.[4,5,6,7] Most Canadian naturopathic practitioners and institutions have been silent, despite the clear moral obligation outlined in our oath to witness and take action. We invite our colleagues to reflect on their ethical and professional obligations as naturopathic doctors, and take action in relation to this genocide, as well as other global conflicts occurring now and in the future.

Some people argue that medical spaces are or should be apolitical. However, medicine is *inherently* political. Every health outcome is embedded in a complex network of power, economics, culture, and governance. In July 2025, The Palestinian Centre for Human Rights warned that the healthcare system in the Gaza Strip had reached a stage of near total collapse in which individuals requiring care for chronic conditions faced a slow, painful death.[8] There was a dramatic spike in otherwise preventable infections, most notably meningitis in children, due to overcrowding, malnutrition, contaminated water, denial of access to antibiotics, and the collapse of the healthcare system[9]—all occurring on the background of overt state violence, starvation, and psychological torture[10,11] and inaction by major global powers.[12,13] Medicine is not practiced in a vacuum.

Many feel the horror of what they see livestreamed on their phones but feel helpless to do anything that will make a difference. Or may be afraid to speak up because others have been punished for doing so, due to the coordinated conflation of criticism of Israel with antisemitism. Unfortunately, continued silence only serves to reinforce others' fear. Speaking up encourages others to do the same.

While doctors may not be able to halt genocide, treat famine, or stop bombs directly, medical professionals have cited ethical obligations in open letters and position statements calling for ceasefire and an arms embargo.[15,16,17,18,19] As naturopathic doctors in Canada, we are guided by our oath and principles, which clearly outline an ethical obligation to advocate for an arms embargo and sanctions on Israel, and immediate and adequate humanitarian aid to Palestinians.

> *"Through precept, lecture, and example, I will assist and encourage others to strengthen their health, reduce risks for disease, and preserve the health of our planet for ourselves, our families, and future generations."*

As naturopathic doctors, we know that the primary causes of suffering in this world are due to social and ecological determinants of health.[20] Palestinians have no access to optimal determinants of health, other than pure *sumud* (صمود, steadfastness). Overt violence, destruction of land and poisoning of water; denial of free access to food, water and shelter; decimation of the healthcare and education systems: these are the epitome of dis-ease.

Militarization itself is one of the most significant contributors to ecological destruction, and subsequent poor health.[21] Armed violence destroys not only human lives, but also agricultural land, water, and air, making places unlivable. Government officials in Israel have clearly stated this intention with respect to Palestine.[22] These harms spill across borders. One study calculated that the projected emissions from 15 months of direct military activities in Palestine were greater than the combined annual emissions of 36 individual countries and territories.[23]

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"I dedicate myself to the service of humanity as a practitioner of the art and science of Naturopathic medicine."

We are in service to all of humanity. Not just those who look like us, or pray like us, or live in the same ways as us. This is the basis of anti-oppressive practice. The state of Israel and the supporters of its actions towards Palestinians have manufactured consent for genocide in part through a campaign of dehumanization.[24,25] The Arab Canadian Lawyers Association has identified several mechanisms of anti-Palestinian racism, ranging from marginalization of Palestinians and their allies from professional or personal spaces, to denial of the existence of Palestine or Palestinians, to outright justification of violence.[26] Countering these tactics is critical to de-normalize the erasure of Palestinians.

Countering anti-Jewish hate is also imperative. The horrific irony is that the coordinated weaponization of accusations of antisemitism as a tactic of silencing harms Jewish people by perpetuating the narrative that Israel's actions are done in their name, and are necessary for their safety. It is critically important that we clearly distinguish between a hatred of, or prejudice towards, people who are Jewish, and a sound criticism of state-sponsored violence, starvation, and displacement, and failure to ensure the human rights of the population residing in a territory that it occupies.[27,28]

"I will conduct my life and the practice of naturopathic health care with vigilance, integrity, and freedom from prejudice. I will abstain from voluntary acts of injustice and corruption."

As human rights activist Desmond Tutu is famously said to have stated, "If you are neutral in situations of injustice, you have chosen the side of the oppressor." Silence emboldens those with power to continue wielding it with impunity in order to uphold harmful policies. The government of Canada has yet to follow through on its calls for Israel to lift restrictions on the flow of aid and to uphold international humanitarian law,[29] enabling the ongoing murder of Palestinians. Political neutrality empowers police forces to violently arrest peaceful demonstrators who call for the end to this genocide.[30,31] Personal and institutional silence emboldens those who harass, report, doxx, and threaten those who act with integrity and freedom from prejudice.

Truth-telling and working towards the dismantling of oppression are not unique to the Palestinian struggle. We, who are settlers and descendants of settlers on Turtle Island, must draw parallels with the ongoing genocide against Indigenous Peoples here, and our collective responsibility towards reconciliation.[32] This struggle is interwoven with the struggle against Islamophobia, anti-Black racism, and anti-imperial movements around the globe.

"I will endeavour to continually improve my abilities as a healer through study, reflection, and genuine concern for humanity. I will impart knowledge of the advanced healing arts to dedicated colleagues and students."

Throughout these past 29 months, the refrain of, "it's complicated," has rung loudly. Indeed, it is. The collective and intertwined trauma and pain of the history of the land in which all Abrahamic religions are rooted is deep. However, we cannot stay silent simply because we feel we don't know enough. The claim of "complication" is a deliberate mechanism of silencing that is used to intimidate and sustain consent for genocide. The conclusion of the International Court of Justice in early 2024 that genocide was even "plausible" should have been enough to demand action to prevent further loss of life.

CONCLUSION

Our ethical code as naturopathic doctors compels us to act to promote good health for all of humanity, and the planet we call home. We must commit to lifelong learning—about history, about genocide, about mechanisms of oppression, and about the difference between existential safety and discomfort. We must draw from our principles and use our collective voice and leverage as doctors, alumni and professional members to demand justice. Naturopathic doctors are trained to think critically. Our institutions have a long history of challenging mainstream narratives and critiquing dominant rhetoric. We can and *must* do that with respect to genocide as well. We call on our naturopathic colleagues to take action (please see Appendix 1 for ways to take action towards justice).

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APPENDIX 1: WAYS TO TAKE ACTION TOWARDS JUSTICE

Taking action does not have to be big or flashy. There are many ways, both private and visible, to influence oneself, one's communities, and the systems in which we all live. Here are some considerations, with sample resources to get you started:

Commit to educating yourself on history, current sociopolitical realities, and the ways in which these inform health and the practice of health care.	▪ Zinn Education Project. Teaching about Palestine-Israel and the unfolding genocide in Gaza. *https://www.zinnedproject.org/news/teaching-palestine-israel/* ▪ Persad S, Xu C. The social determinants of health and genocide: towards a public health integrated framework of genocide and mass violence. GSP 2023;17(2):1-21. *https://doi.org/10.5038/1911-9933.17.2.1938*. ▪ The Guardian. Five of the best books to understand the Israel-Palestine conflict. *https://www.theguardian.com/books/2024/feb/29/five-of-the-best-books-to-understand-the-israel-palestine-conflict*
Engage in honest personal reflection, including on your own positionality (biases, position of privilege), and reasons for engagement or disengagement from Palestine or other forms of oppression/injustice.	▪ Amnesty International Book Club. One Day, Everyone Will Have Always Been Against This – Discussion Guide. *https://amnesty.ca/bookclub-news/one-day-omar-el-akkad-discussion-guide/*
Listen for and challenge accuracy and bias of language in conversations and reporting about Palestine and Israel.	▪ The Conversation. Bias hiding in plain sight: Decades of analyses suggest US media skews anti-Palestinian. *https://theconversation.com/bias-hiding-in-plain-sight-decades-of-analyses-suggest-us-media-skews-anti-palestinian-216967* ▪ Canadians for Justice and Peace in the Middle East. Media Accountability Project. *https://www.cjpmemap.ca/*
Advocate that governmental bodies and professional associations acknowledge and condemn the genocide in Palestine and elsewhere, and act to ensure necessary action to bring justice to those affected.	▪ Doctors Without Borders. Palestine: Ceasefire not the end of the extreme suffering in Gaza. *https://www.doctorswithoutborders.ca/palestine-ceasefire-not-the-end-of-extreme-suffering-in-gaza/* ▪ European Public Health Alliance. Joint Public Health Statement on Gaza. *https://epha.org/joint-public-health-statement-on-gaza/*
Seek community among others who are advocating for the well-being of Palestinians, and justice in the region.	▪ Doctors Against Genocide ▪ Health workers Alliance for Palestine
Leverage your public platforms to educate your followers on the links between genocide and health, and amplify the voices of those on the ground. Be aware of systemic censorship within social media related to this topic.	▪ Modern Diplomacy. Social Media's Key Role in Palestinian Activism for Gaza. *https://moderndiplomacy.eu/2024/10/25/social-medias-key-role-in-palestinian-activism-for-gaza/* ▪ Human Rights Watch. Meta's Broken Promises: Systemic Censorship of Palestine Content on Instagram and Facebook. *https://www.hrw.org/report/2023/12/21/metas-broken-promises/systemic-censorship-palestine-content-instagram-and*
Consider non-violent direct actions that you can take to withhold support for genocide.	▪ World Beyond War. Divest from Genocide: Break Up With Your Bank! *https://worldbeyondwar.org/breakupwithyourbank/* ▪ BDS Movement

For more thoughts on taking action as healthcare providers, we direct readers to the excellent article: Zafran H, Beagan BL, Shephard D, et al. Shattering silence, inviting dialogue: anti-oppressive occupational therapy during the genocide of Palestinians. *Can J Occup Ther*. 2025;0(0). https://doi.org/10.1177/00084174251356348