

Efficacy and safety of non-pharmacologic interventions for management of pediatric migraine: A systematic review of randomized controlled trials

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ABSTRACT

Pediatric migraine affects approximately 7.7% to 9.1% of children and adolescents worldwide, significantly impacting their quality of life by hindering academic performance, social activities, and daily physical tasks. These effects are comparable to chronic conditions like rheumatoid arthritis or cancer. This study aimed to systematically review the effectiveness and safety of non-pharmacologic interventions for pediatric migraine. Articles were identified through systematic searches in PubMed, Cochrane, and Elsevier databases, supplemented by manual searches from inception to October 19, 2024. Data were extracted using structured forms and analysed qualitatively, with study-level results extracted and summarized narratively. Risk of bias were assessed using the Cochrane Risk of Bias Tool 2 (RoB2). Eligible studies were randomized controlled trials (RCTs) evaluating non-pharmacologic interventions as stand-alone treatments, with comparator groups receiving pharmacologic therapy alone, non-pharmacologic therapy alone or placebo/usual care with outcomes related to migraine frequency, severity, duration, or safety. Exclusion criteria included studies on non-migraine headache types, mixed populations without subgroup analysis, insufficient data, or non-RCT designs. Eighteen RCTs involving 816 pediatric participants were included. Relaxation therapy reduced migraine frequency from 2.59 to 0.89 episodes per week while biofeedback decreased episodes from 2.7 to 1.0 weekly. Acupuncture demonstrated significant benefit, reducing frequency from 9.3 to 1.4 episodes per month. Butterbur root extract achieved 36.1% reduction. Coenzyme Q10 showed promising results, whereas riboflavin did not outperform placebo. These values represent individual study finding, no meta-analysis was performed. Overall, relaxation therapy, biofeedback, and selected dietary supplements appear effective and well-tolerated, offering safer, evidence-based alternatives to pharmacologic treatments and supporting individualized strategies to improve quality of life in young patient.

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1. INTRODUCTION

Migraine is a leading cause of disability in people under the age of 50, affecting over 1 billion globally and causing significant socioeconomic impact. It is characterized by recurring, lifelong attacks that include moderate to severe, one-sided, pulsating headaches, worsened by physical activity and accompanied by light and sound sensitivity and nausea (World Health Organization [WHO], 2024). Migraine attacks are not only uncomfortable but they are also ranked as the third most significant contributor to disability-adjusted life years (DALYs) globally, following stroke and dementia (WHO, 2019, 2024). On the other hand, migraine, according to Global Burden Disease (2016) is ranked first among the disabling diseases in the 15-49 age range (Institute for Health Metrics and Evaluation [IHME], 2016; Onofri et al., 2023). Migraines often begin at puberty, primarily affecting adults (aged 21-45), and are more common in women due to hormonal factors but can also occur in children and older people. An estimated 50% to 75% worldwide suffer from migraine, while 14% report having severe headaches (WHO, 2024). Chronic migraines impact about 2% of the global population and majorly cause short-term work absences (WHO, 2024). In the Philippines, the most recent national migraine prevalence survey was conducted in 2008 by the National Health Survey. It was found that 7.9% of the population suffered from chronic migraines, with a female to male ratio of 2.29:1 (Roxas et al., 2022).

Approximately 60% of children and adolescents suffer from severe headaches, with 7.7% to 9.1% experiencing migraines. Migraines significantly hinder academic performance, social engagement, physical activity, and overall quality of life, comparable to chronic conditions such as rheumatoid arthritis or cancer. As many as 75% of children with migraines will continue to exhibit symptoms until adulthood (Onofri et al., 2023; Szperka, 2021). With adolescence being a critical phase characterized by significant migraine prevalence; approximately 10% of children aged 5 to 15 and 28% of adolescents experience migraines, leading to disability, school absenteeism, emotional or social difficulties, and the potential for headache chronification if misdiagnosed or inadequately managed (Al Khalili et al., 2023; Cleveland Clinic, 2023; Nierenburg et al., 2021).

Pharmacologic treatments for pediatric migraine, while commonly prescribed, present notable safety concerns. Preventive agents such as topiramate and valproate pose teratogenicity, amitriptyline may cause sedation, weight gain, and cardiac conduction abnormalities, while propranolol is contraindicated in asthma or cardiovascular disease. Triptans, while effective, can induce vasoconstriction and cardiovascular adverse effects, with side effects including fatigue, dizziness, paresthesia, and chest pressure (Oskoui et al., 2019; Szperka, 2021). Moreover, many preventive medications show no clear superiority over placebo, and frequent use of NSAIDs may contribute to medication-overuse headache and migraine chronification (Al Khalili et al., 2023; Haghdoost & Togha, 2022). These safety limitations underscore the need for safer, evidence-based non-pharmacologic interventions as primary or adjunctive strategies such as music therapy, neuromodulation, nutraceuticals, lifestyle modification, cognitive-behavioral therapy/techniques (CBT), biofeedback, and structured programs like Headstrong Compact Disc Read-Only Memory (CD-ROM) in pediatric migraine management (Hoover, 2020). Additionally, the focus of this research was on non-pharmacological intervention appropriate for pediatric patients. These groups are more susceptible because of their greater possibility of experiencing negative drug reactions and their somewhat lower ability to tolerate migraines (Gopalakrishnan & Ganeshkumar, 2013; Hoover, 2020). This systematic review aims to examine non-pharmacologic interventions for managing pediatric migraines, providing insights into their effectiveness, implementation, and potential role in comprehensive migraine care.

The overall objective of the study was to qualitatively synthesize the effectiveness and safety of non-pharmacologic interventions for the management of pediatric migraines. This study is significant as it provides valuable insights into alternative approaches that may reduce reliance on medications, enhance patient safety, and broaden migraine care strategies for children.

2. MATERIAL & METHODOLOGY

2.1 Data sources and search strategy

This systematic review followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidance. The selection process was summarized using a PRISMA flow diagram (Figure 1). The protocol was registered prospectively on PROSPERO (registration number CRD42024600957) on 25 October 2024. An electronic search was performed from database inception to 19 October 2024 across PubMed, ClinicalKey Elsevier, and the Cochrane Database. Searches included articles in all languages. The Cochrane Central Register of Controlled Trials CENTRAL was also explored. Reference lists of existing systematic reviews, meta-analyses, and relevant primary studies were manually searched.

The search strings were tailored according to the requirements of each database. For example, in PubMed, the terms “non-pharmacologic intervention” or “non-pharmacologic treatment” were combined with “pediatric migraine” or “children migraine,” along with related keywords such as “behavioral therapy,” “biofeedback,” “diet,” “relaxation,” “stress management,” and “dietary supplements” to capture relevant studies. In contrast, the searches in Cochrane, Elsevier, Google Scholar, and other databases were simplified to the key terms “non-pharmacologic treatment/intervention” and “pediatric migraine.” All searches were combined with Boolean operators (AND, OR) appropriate to each database. Moreover, no language and year limits were applied. This ensured that all available randomized controlled trials (RCTs), regardless of publication date, were considered for inclusion.

2.2 Study selection

Search results were exported into Microsoft Excel and screened independently by two reviewers. Titles and abstracts were checked, duplicates were removed, and uncertain records were retrieved for full-text review. Non-English articles were translated using Google Translate or other applications when needed. A screening form in Google Sheets recorded study details, eligibility decisions, reviewer names, and dates. Studies that failed inclusion criteria or met exclusion criteria were excluded, with reasons documented. Any disagreements were resolved through consensus.

Inclusion criteria:

Eligible studies were original research articles of any language, specifically RCTs in pediatric populations aged 5–19 years with migraine diagnosed by ICHD, IHS, or comparable criteria. Studies could be from any location but had to evaluate non-pharmacologic interventions against a comparator, defined as standard pharmacologic therapy, non-pharmacologic intervention, placebo, or usual care (no treatment). Intervention duration was required to be at least one month to allow sufficient time for measurable effects on migraine frequency, severity, duration and safety outcomes to emerge. Only articles published up to 19 October 2024 were considered.

Exclusion criteria:

Excluded studies included non-original works, abstracts only, duplicates, inaccessible articles, those without comparator groups, studies on other headache types or combined diseases without subgroup analysis, and non-RCT designs.

2.3 Data extraction

Two authors (J.P.G. and V.G.R.V.) independently extracted data using structured Google Sheets form. Both were pharmacy students trained in systematic review methodology with prior clinical research experience, selected for their familiarity with pediatric migraine interventions and ability to apply eligibility criteria consistently. For each included study, publication details (title, authors, year, journal, country, conflicts of interest), study design and setting (design type, time frame, sampling), and participant characteristics (age, sex distribution, race/ethnicity, sample size, inclusion/exclusion criteria, withdrawals) were recorded. Methodological features (study start/end dates, screening tools, randomization and allocation procedures, delimitations, ethical approvals), detailed descriptions of the intervention and comparator (number of participants, theoretical basis, description, duration, timing, delivery, provider), and outcome measures (measurement timepoints, instruments and validation, assessor, units, handling/imputation of missing data, reported risk estimates, power, and response rates) were also extracted. When available, numerical data for potential pooling (sample sizes, means, standard deviations, p-values) were collected. Any discrepancies in extraction were resolved through discussion until consensus was reached, and authors were contacted when additional information was needed. Moreover, risk of bias was assessed using the Cochrane Risk of Bias 2 (RoB2) tool, which evaluates randomization, deviations from intended interventions, missing data, outcome measurement, and selective reporting. Studies were judged low risk if all domains were rated low, some concerns if at least one domain raised concerns but none were high, and high risk if one or more domains were rated high.

2.4 Intervention, comparator, and outcome of interest

The review evaluated the safety and efficacy of non-pharmacologic interventions for pediatric migraine. Interventions of interest included, but were not limited to: music therapy, repetitive cathodal direct current stimulation, nutraceuticals/dietary supplements or lifestyle modifications, CBT, autogenic training, electromyographic feedback, thermal feedback, the Headstrong CD-ROM program, and butterbur root combined with music therapy. Comparators included placebo, other non-pharmacologic interventions, or standard pharmacologic treatments. Primary outcomes were reduction in frequency and intensity of migraine attacks and safety measured by adverse event occurrence and prevalence.

2.5 Data processing and qualitative analysis

The characteristics of the studies were analyzed using a narrative Synthesis Without Meta-analysis (SWiM) approach, as the data were too heterogeneous to permit meta-analysis. Study-level descriptive statistics (e.g., reported means, standard deviations, and percentages from individual RCTs) were extracted and summarized qualitatively rather than pooled quantitatively. The synthesis focused on domains of interest, including migraine type characteristics, participant characteristics, characteristic of intervention and comparator groups, characteristic of measured outcomes, and risk of bias assessment using the RoB2 tool.

3. RESULTS

3.1 Search results

Upon searching, 685 articles were retrieved, additional 48 articles were sourced through hand-searching and other sources, leading to a total of 733 articles. These articles were then filtered for eligibility based on the criteria, and duplicates were removed during this process. Each remaining article was subsequently screened by title and abstract using Microsoft Excel, resulting in 138 eligible articles. However, upon full-text review, 120 articles were excluded for various reasons detailed in Fig. 1. Eighteen studies remained and were included in the qualitative analysis, as summarized in the PRISMA flowchart (Fig. 1).

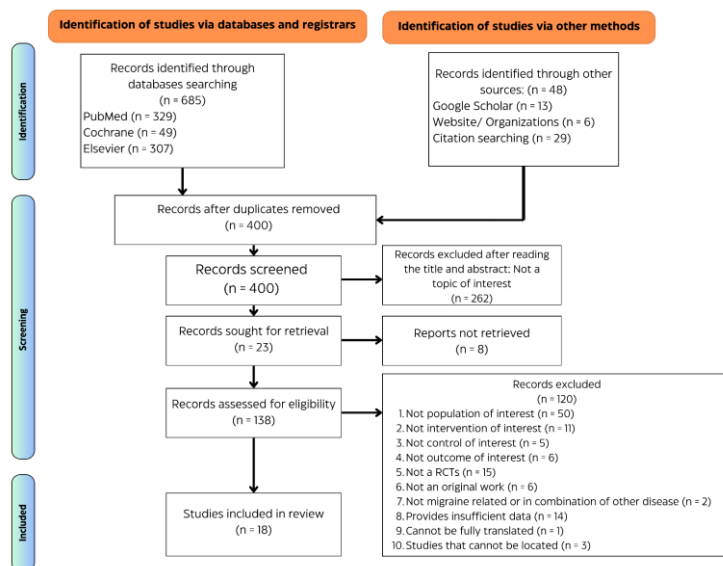


Fig. 1. Accomplished PRISMA flowchart.

3.2 Interventions and comparator

Interventions used are divided into relaxation therapy, CBT, biofeedback, acupuncture, or nutraceuticals/dietary supplements. Ten studies evaluated relaxation therapy (progressive muscle training (1), relaxation response (7) and music therapy (2)) against waiting-list control group (WCG), education or placebo. Three studies used CBT including cognitive coping strategies, clinic-based CBT, and headstrong program with CBT while one study used acupuncture comparing it to placebo acupuncture. Furthermore, five studies use biofeedback and another five studies used nutraceutical/ dietary supplements such as butterbur root extract, fish oil, magnesium oxide, riboflavin, and coenzyme Q10 (CoQ10). Details of interventions and comparators are presented in Table 1.

Comparators varied across trials. Placebo and usual care were the most common, particularly in nutraceutical studies. Some behavioral trials used wait-list controls, while one study compared interventions against active pharmacologic therapy. Although this variability complicates direct comparisons, it reflects real-world diversity in pediatric migraine management. Across studies,

comparator groups generally showed smaller or nonsignificant improvements, underscoring the relative efficacy of non-pharmacologic strategies.

3.3 Description of non-pharmacologic interventions

Relaxation therapy trains adolescents to contract and release muscle groups, eventually linking cues like “relax” with calmness to reduce migraine activity (Fichtel & Larsson, 2001). The relaxation response lowers oxygen use, heart rate, and sympathetic activity through meditation, progressive muscle relaxation, autogenic training, and biofeedback, usually taught in 8–10 sessions (Fentress et al., 1986; Labbé, 1995; McGrath et al., 1988; Richter et al., 1986). Music therapy applies relaxation, body awareness, and roleplay, using active methods like making music and receptive strategies such as music-assisted imagery (Koenig et al., 2013; Oelkers-Ax et al., 2008).

Cognitive coping strategies restructure maladaptive thoughts that trigger migraines, using pain control, problem-solving, and stress inoculation (Richter et al., 1986). CBT, whether clinic-based in structured 8-week sessions or self-guided through the Headstrong CD-ROM program for children, combines relaxation, imagery, and coping skills to reduce headache severity and disability (McGrath et al., 1992; Rapoff et al., 2014).

Acupuncture reduces migraine frequency and intensity by enhancing endogenous opioid activity (Pintov et al., 1997). Nutritional interventions such as Butterbur root extract, fish oil, magnesium oxide, riboflavin, and Coenzyme Q10 have been studied, with magnesium and CoQ10 showing promising results while riboflavin did not significantly outperform placebo (Harel et al., 2002; MacLennan et al., 2008; Oelkers-Ax et al., 2008; Slater et al., 2011; Wang et al., 2003).

3.4 Description of studies and study characteristics

A total of 18 RCTs (comprising 816 patients) conducted between 1984 and 2014 and comparing various treatments with different control groups are included. The full details of the included studies and study characteristics are presented in Table 1 to Table 3.

In total, the mean age was 11.12 years (SD 0.96) and 476 (61%) of the sample population were female. Diagnostic criteria varied: while several studies applied standardized frameworks such as ICHD-II or IHS, others relied on physician or neurologist diagnosis or older criteria (e.g., Prensky and Sommer, Barlow, Kaiser Permanente), reflecting heterogeneity in case definition. Intervention duration typically spanned 7 weeks, though protocols ranged from 4 to 16 weeks.

Geographically, half of the studies were conducted in the United States, with additional trials from Canada, Israel, Germany, Sweden, and Australia, underscoring the international but Western-dominant distribution of pediatric migraine research. Ethnicity was rarely reported; most studies omitted this information, limiting generalizability. Where reported, participants were overwhelmingly White/Caucasian, highlighting a lack of diversity in study populations.

The trials were published in leading psychology, neurology, and pain journals, reflecting the multidisciplinary interest in pediatric migraine management. Collectively, these characteristics show that while the evidence base spans multiple countries and intervention durations, it remains concentrated in Western populations with limited ethnic representation and variable diagnostic standards.

Table 1. Study-specific characteristics of included journal articles

Study ID (First Author, Year)	Intervention & Comparator	Duration	Diagnosis	Diagnostic Criteria	Mean Age per grp, SD ($\pm$)	Gender per grp (M:F)	Race/ Ethnicity per grp (% White, % Black, % Other)	Baseline Migraine Frequency (mean per month)	Baseline Migraine severity (0-10 point scale)	Baseline Duration (hour per episode)	Category of Non- pharmacologic Intervention used
Labbe 1984	Intervention: Autogenic feedback training	35 weeks	Childhood migraine	Physician's Diagnosis	10.6	7M:7F	NI	2.59 per week	0-5 scale: 3.23	6.19	Relaxation Therapy
	Comparator: Waiting list control group				10.8	7M:7F		2.64 per week	3.33	7.12	
Richter 1985	Intervention 1: Relaxation Training	6 weeks	Common Migraine	Neurologist's Diagnosis	Total: 12.87	Total: 17M:34F	NI	9.03	3.6	1.8	Relaxation Therapy & Cognitive Behavioral Therapy
	Intervention 2: Cognitive Cope							8.14	3.37	1.81	
	Comparator: Placebo							7.26	3.58	1.68	
Fentress 1986	Intervention 1: Biofeedback with Relaxation response	15 weeks	Recurrent, intermittent migraine	Neurological evaluation using Barlow 1984	10.1	2M:4F	NI	2.7		9.7	Biofeedback and Relaxation Therapy
	Intervention 2: Relaxation response				10.1	2M:4F		2.6	Not specified	14.6	
	Comparator: Waiting list control group				10.1	3M:3F		2.3		6.3	
McGrath 1988	Intervention 1: Relaxation training	18 weeks	Children & adolescent migraine	Prensky and Sommer (1979)	Total: 13.1 mean age	10:23	NI	HI: High severity: 53.5 ± 24.1 Low severity: 11.6 ± 5.1		NR	Relaxation Therapy
	Intervention 2: Own best effort							HI: High severity: 41.2 ± 16.0 Low severity: 9.7 ± 4.9			

	Comparator: Psychological placebo					10:23			HI: High severity: 42.6 ±17.0 Low severity: 10.4 ±4.7		
McGrath 1991	Intervention 1: Self- administered treatment					7M : 17F			HI: 153		
	Intervention 2: Clinic-based CBT + relaxation (therapist- administered)	1 year, 6 months	Adolescence Migraine	Pediatric Neurologist Diagnosis	Total: 14.5	7M : 16F	NI		HI: 166	NR	Cognitive Behavior Therapy and Relaxation
	Comparator: Control group (Trigger avoidance + brainstorming)					6M : 20F			HI: NR		
Labbe 1994	Intervention 1: Skin temperature biofeedback with autogenic relaxation training								2.71	HI: 0.66	5.78
	Intervention 2: Autogenic relaxation training only	6 months	Childhood migraine	Physician's Diagnosis	Total: 12	Total: 17M:13F	NI		3.67	HI: 0.74	6.71
	Comparator: Waiting list control group								3.18	HI: 0.41	3.92
Pintov 1997	True acupuncture	12 weeks	Migraine headaches	IHS Prensky and Sommer (1979)	9.8 ± 1.2 years	5M:7F	NI	4.2 ± 2.4	NR	NR	Acupuncture
	Comparator: Placebo				10.4 ± 1.6 years	4M:6F		4.1 ± 2.2			
Allen1998	Biofeedback treatment alone (BFB)	12 weeks	Migraine headaches	IHS	12.3	4M:9F	NI	4.2 ± 2.4 per week	NR	NR	Biofeedback
	Comparator: Biofeedback Treatment with Parent- Mediated Pain Behavior Management Guidelines (BFB+OP)				12.1	7M:7F		4.1 ± 2.2 per week			

Sartory 1998	Intervention 1: Progressive relaxation + stress management	1 year and 2 months	Episodic migraines with aura	IHS	11.4 ±2.4	9M : 6F	NI	2.24 ±1.89 per week	5.09 ± 1.35	19.56 ±25.98	Relaxation and Biofeedback
	Intervention 2: Cephalic vasomotor biofeedback + stress management				11.8 ±2.0	8M : 7F		1.77 ±1.17 per week	4.37 ± 2.25	9.35 ±8.07	
	Comparator: Metoprolol				10.5 ±1.9	9M : 4F		1.33 ±0.62 per week	5.34 ± 1.88	9.10 ±4.67	
Fichtel 2000	Progressive muscle relaxation + stress management	1 year and 3 months	Migraine headache	IHS	Total: 15.4±1.55	5M : 15F	NI	1.85 ±2.1	3.35 ± 0.7 0-5 point scale	4.05 ±6.8	Relaxation therapy
	Comparator: Waiting list control group					6M : 10F		1.10 ±0.8	3.25 ± 0.8 0-5 point scale	2.13 ±5.3	
Harel 2001	Fish Oil (1g n- 3 ethyl ester)	5 months	Chronic migraine	IHS	15 ±1	1M:2.29F	Whites: 77.78% Hispanics: 14.81% Afri-Ameri: 3.70% Cape- Verdean: 3.70%	15.5 ± 2	5.0 ± 0.3 (0-7 point)	NR	Nutraceuticals/ Dietary Supplements
	Comparator: Placebo (olive oil)										
Scharff 2002	Intervention 1: Handwarming Biofeedback	6 months	Pediatric migraine	IHS	13.3 (2.5)	4M:9F	NI	11.3 ± 3.0	3.6 ± 0.8 0-4 point scale	31.8 ± 25.6 months	Biofeedback
	Intervention 2: Handcooling Biofeedback				13.2 (2.0)	6M:5F		9.5 ± 4.7	3.4 ± 1.0	29.8 ± 26.9 months	
	Comparator: Waiting list control group				12.0 (2.7)	2M:10F		12.0 ± 3.2	3.4 ± 1.0	28.3 ± 25.1 months	

Wang 2003	Oral Magnesium Oxide (9mg/kg)	16 weeks	Chronic migraine	Kaiser Permanente	12.2	1M:2.63F	White: 51.7% Hispanic: 19% Other: 8.6% Multiethnic: 19%	9.3	NR	NR	Nutraceuticals/ Dietary Supplements
	Comparator: Placebo				11.8	1M:1.86F	White: 66.7% Hispanic: 13.3% Other: 5% Multiethnic: 15%	11.5			
Oelkers-Ax 2007	Intervention 1: Butterbur Root Extract (Petadolex) capsule + headache education	28 weeks	Migraine with or without aura	IHS	10.6 ±1.2	10M:9F		9.8 ±7.6	4.9 ±1.7 0-6 point scale	NR	Nutraceuticals/ Dietary Supplements and Relaxation Therapy
	Intervention 2: Music therapy + headache education				9.9 ±1.4	17M:3F	NI	5.0 ±2.5	4.6 ±1.5		
	Comparator: Placebo + headache education				10.6 ±1.5	13M:6F		5.5 ±4.4	5.0 ±1.4		
MacLennan 2008	Riboflavin (200 mg daily)	12 weeks (3 months)	Migraine (with or without aura)	IHS	11.1 ± 2.1	15M:12F	White: 93% Others: 7%	4.4 ± 1.9	NR	8.0 ± 7.8	Nutraceuticals/ Dietary Supplements
	Comparator: Placebo				11.5 ± 2.5	9M:12F	White: 86% Others: 15%	4.2 ± 2.0		6.5 ± 6.6	
Slater 2011	100mg CoEnzyme Q10	32 weeks	Migraine with or without aura; Episodic or Chronic	ICHD-II	13.3 ±3.0	1M:2F (ratio)	White: 91.7% Black: 6.7% Other: 1.7%	14.3 ±6	6.4 ±1.8	10.1 ±16.9	Nutraceuticals/ Dietary Supplements
	Comparator: Placebo				14.0 ±2.6	1M:1.7F	White: 78.3% Black: 16.7% Other: 5%	15.4 ±7.5	6.2 ±1.7	8.3 ±8.8	
Koenig 2013	Music therapy	24 weeks (6 months)	Migraine with or without aura; Chronic	ICHD-II	15.19 ± 1.76	9M:31F		18.5 ± 8.4	5.0 ± 1.6	4.3 ±2.8	Relaxation Therapy
	Comparator: Rhythm pedagogic program				14.96 ± 1.67	11M:27F	NI	17.1 ± 8.1	4.7 ± 1.3	4.6 ±2.9	

Rapoff 2014	Headstrong Program (Self-guided CD-ROM) with cognitive-behavioral self-management strategies	16 weeks	Migraine with or without aura	ICHD-II	10.2 ±2.0	8M:10F	Caucasian = 94%	41.09 ±22.67 mean percentage per day	5.06 ±1.84	5.47 ±4.2	Cognitive Behavior Therapy
	Comparator: Educational CD-ROM Program				10.2 ±1.5	2M:15F	Caucasian = 94%	40.67 ±28.79 mean percentage per day	6.0 ±1.52	4.15 ±3.88	

Abbreviations:
Headache index (HI) = combines both the frequency and the severity of headaches over a given time frame; International Headache Society = IHS; NI = No information; NR = Not reported

Table 2. Characteristics about sample or target population of included journal article

Study ID	Final Number of Patients	Number of px used in treatment grp		Number of px grp in comparator	Drop-out patient	Age group
		Intervention 1	Intervention 2			
Labbe 1984	28	14	-	14	0	7 to 16 years of age
Richter 1985	42	15	15	12	9	9 to 18 years
Fentress 1986	18	6	6	6	17	8 to 12 years old
McGrath 1988	99	33	-	33	37	9 to 17 years old
McGrath 1991	74	24	23	26	18	11 to 18 years old
Labbe 1994	46	10	10	10	16	7 to 18 years old
Pintov 1997	22	12	-	10	0	7 to 15 years of age
Allen 1998	27	13	14	13	6	7 to 18 years old
Sartory 1998	27	15	15	13	16	8 to 16 years old
Fichtel 2000	31	20	-	16	5	13 to 18 years old
Harel 2001	23	14	-	13	4	12 to 21 years old
Scharff 2002	36	13	11	12	0	7 to 17 years old
Wang 2003	86	42	-	44	32	3 to 17 years old
Oelkers-Ax 2007	50	19	20	19	26	8 to 12 years old
MacLennan 2008	48	27	-	21	0	5 to 15 years old
Slater 2011	50	34	-	28	70	6 to 17 years old
Koenig 2013	74	40	-	38	7	12 to 17 years old
Rapoff 2014	35	40	-	30	13	7 to 12 years old

Table 3. Methods characteristics of included journal articles

Study ID	Study Design	Participant inclusion criteria	Random allocation	Blinding	Sample size determination	Duration of intervention 1	Duration of Intervention 2	Duration of Control/ Placebo	Post-treatment Follow-up	Wash-out period	Statistical Methodology	Time Frame	Limitation of the study
Labbe 1984	PGT	Stated	SR	NB	NR	7 weeks	-	7 weeks	1 mos & 6 mos	NA	ANOVA, Post-hoc comparisons (Newman-Keuls Statistic), t-tests	NSD	Six-month follow-up data were not included in the analysis of data because there were no subjects from the control group for comparison. This results in lack of comprehensive understanding on the long-term effects of the intervention on both groups; Small sample size; No blinding
Richter 1985	PGT	Stated	SR	DB	NR	6 weeks	6 weeks	6 weeks	4 mos	NA	Repeated Measures ANOVA	NSD	Small sample size. No blinding, leading to possible expectancy bias. Self-reported headache diaries may introduce recall bias. No long-term follow-up beyond 16 weeks.
Fentress 1986	PGT	Stated	SR	NB	18	15 weeks	15 weeks	15 weeks	1 year	NA	Kruskal-Wallis tests, Wilcoxon tests	NSD	Small sample size. No blinding. There was an attempt to quantify the amount of time patients spent practicing the relaxation response in their natural environment, but it failed to produce sufficient data for analysis.
McGrath 1988	PGT	Stated	StR	SB	99	6 weeks	6 weeks	6 weeks	4 weeks, 3 mos, & 12 mos	NA	ANOVA, post-hoc analysis, treatment credibility analysis	May 1986 to December 1987	No significant difference between relaxation training and placebo/self-management. Some children dropped out, but reasons and numbers were not detailed. Short treatment duration (6 weeks) might not be sufficient for long-term behavioral changes. Expectancy effects may have influenced results, as all groups showed improvement. The placebo intervention involved psychological discussions, which may have had a therapeutic effect. Natural variability in childhood migraines may have influenced results.
McGrath 1991	PGT	Stated	StR	NB	NR	4 weeks	4 weeks	4 weeks	1 mo, 3 mos, & 1 year	NA	Repeated measures ANOVA, chi-square, planned contrasts	NSD	Possible selection bias (referral-based recruitment) No physiological measures High female predominance
Labbe, 1994	PGT	Stated	SR	NB	NR	7 weeks	7 weeks	7 weeks	1 mo & 6 mos	NA	ANOVA, T-tests	NSD	Small sample size No blinding Short follow-up period

Pintov 1997	PGT	Stated	SR	DB	22	10 weeks	-	10 weeks	NA	NA	Student's t-test, radioimmunoassay techniques.	December 1996- May 1997	Small sample size (only 22 children) limits the generalizability of the findings. Lack of long-term follow-up beyond 10 weeks after treatment. Possible placebo effects despite blinding efforts. Limited to children mature enough to cooperate with acupuncture treatment, excluding younger age groups. Did not assess other potential confounding factors such as diet, sleep patterns, and stress levels.
Allen 1998	PGT	Stated	SR	SB	27	6 weeks	-	6 weeks	1 mo, 3 mos, & 1 year	NA	ANOVA, Student's t-test, Fisher's exact test, correlational analysis	September 1997 to January 1998	No control group without treatment (only comparison between BFB Alone and BFB+OP). Lack of blinding for therapists and parents, which could introduce bias. No direct measure of parent compliance with behavior management guidelines. Long-term impact beyond 1 year was not assessed. Did not analyze different migraine subtypes separately. Parental behavior might have changed naturally as children learned self-regulation, which wasn't fully controlled
Sartory 1998	PGT	Stated	SR	NB	NR	6 weeks	-	10 weeks	2 weeks	NA	MANOVA, Wilcoxon signed-ranks test, chi-square	NSD	Small sample size Short psychological treatment duration Vasomotor feedback effects unclear
Fichtel 2000	PGT	Stated	SR	NB	NR	5 weeks	-	5 weeks	8 to 12 mos	NA	One-tailed t-tests, dependent t-tests, chi-square	NSD	High attrition (44% lost at follow-up) Comorbidity (86% migraine + TTH) No long-term efficacy data
Harel 2001	COT	Stated	SR	DB	27	8 weeks	-	8 weeks	NA	1 month	Two tailed t-test Chi-square analysis Multiple Regression Analysis	NSD	Placebo choice - olive oil have anti-inflammatory properties that might have influence the effect Small sample size study did not control for participants' dietary intake of other sources of omega-3 and omega-6 fatty acids
Scharff 2002	PGT	Stated	StR	NB	NR	6 weeks	6 weeks	8 weeks	3 mos, 6 mos, & 12 mos	NA	Multivariate analysis, Nonparametric analyses, chi-square analysis, repeated MANOVA, Univariate ANOVAs	NSD	Small sample size No blinding Only one single investigator conducted all evaluation, treatment, and follow-up sessions. Thus, no treatment integrity checks, and drift may have occurred.
Wang 2003	PGT	Stated	BR	DB	120	16 weeks	-	16 weeks	NA	NA	Chi-square test T-test and Wilcoxon Rank Sum Tests Poisson Regression Generalized Estimating Equations Intention to treat	June 1997 - January 2000	Small sample size The high placebo response rate in pediatric headache studies made it challenging to detect a strong treatment effect. The study duration was 16 weeks, which may not be long enough to observe the full effects of magnesium oxide on migraine prevention The study did not monitor blood levels of magnesium during the trial, which could have provided

											analysis	additional insights into the treatment's effectiveness.
Oelkers-Ax 2007	PGT	Stated	BR	DB	60	12 weeks	12 weeks	12 weeks	6 mos	NA	ANOVA Fischer's Exact Test Repeated ANOVA Robust Regression Analysis Wald Test Power Analysis	August 2001 - November 2003 Small sample size High drop-out rate Short duration While the study included a placebo control group, the music therapy was not blinded, which could introduce bias. A substantial reduction in headache frequency was observed during the baseline period, which could be due to non-specific effects such as the medical consultations and the use of headache diaries. The study did not fully account for the potential influence of psychological factors and comorbidities on the outcomes. The study allowed for dose adjustments of butterbur root extract, which could complicate the interpretation of the results.
MacLennan 2008	PGT	Stated	StR	DB	NR	12 weeks	-	12 weeks	NA	NA	Analysis of Variance (ANOVA) Chi-square tests	NSD Small sample size. Short study duration (only 12 weeks of treatment). No long-term data on efficacy or safety. Possible placebo effect, given the high response rate in both groups.
Slater 2011	COT	Stated	SR	DB	120	16 weeks	-	16 weeks	NA	No wash-out period	ANOVA Logistic Regression Analysis of Covariance	NSD Short study duration (only 12 weeks of treatment). The study had a relatively small sample size, with only 50 patients analyzed at the endpoint There was a high dropout rate, with 48 participants lost to follow-up at the crossover point and an additional 22 participants lost at the endpoint No wash out period. All participants received multidisciplinary treatment, including acute therapy, prophylactic therapy, and biobehavioral therapy. There were differences in baseline headache frequency between the CoQ10 and placebo groups, particularly in the chronic migraine group. The study used a fixed dose of 100 mg of CoQ10, which may not be optimal for all participants.
Koenig 2013	PGT	Stated	PBR	SB	NR	8 weeks	-	8 weeks	NA	NA	Chi-square tests, Random-effects regression models, intention-to-treat analysis	January 11, 2010-September 4, 2011 Music therapy was not superior to placebo. Small sample size. No formal power analysis to determine an appropriate sample size. Possible nonspecific psychological effects influencing results. Lack of comparison with pharmacological treatments.

Rapoff 2014	PGT	Stated	StR	NB	80	4 weeks	-	4 weeks	3 mos	NA	ANOVA Effect size (Cohen's d) Univariate Test One-tailed test Descriptive statistics	December 2004 - March 2010	The study was underpowered according to predetermined sample size requirements. The final group sizes fell short of those required by the sample-size determination, which limited the statistical power of the study. The generalizability of the findings is limited due to the exclusion of children with headaches less than once per week and those with mental health conditions. This may lead to sample biases.
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Abbreviations:
 PGT = Parallel Group Trial; COT = CrossOver Trial; SR = Single Randomization; StR = Stratified Randomization; BR = Blocked Randomization; PBR = Permuted Blocked Randomization;
 NB = No Blinding; SB = Single Blinding; DB = Double Blinding; NR = Not Reported; NA = Not Applicable; NSD = No Specified Date

3.5 Risk of bias

Across the 18 studies, five were overall low risk, 12 were high risk, and one had some concerns (Figures 2.1–2.3). Randomization was generally adequate, but blinding was often incomplete due to the nature of behavioral interventions. Missing outcome data was a frequent concern, particularly in long-term follow-ups. Selective reporting was noted in several studies, with incomplete disclosure of secondary outcomes.

Parallel RCTs (Intention-to-Treat), four studies were low risk due to proper randomization, allocation concealment, blinding, appropriate outcome measures, and adherence to pre-specified analyses. Five were high risk because of unclear allocation, lack of blinding, inappropriate measurement, missing data, and multiple eligible analyses, while one had some concerns with allocation concealment. In contrast, all six parallel RCTs analyzed per-protocol had at least one high-risk domain, reflecting problems with allocation concealment, adherence, missing data, and outcome measurement.

For the Crossover RCTs, one study was low risk with proper randomization, adequate wash-out, complete data, appropriate measurement, and pre-specified analyses, while the other was high risk due to multiple domain concerns. This underscores the need for more rigorous methodology in future pediatric migraine research.

Study ID	INTENTION-TO-TREAT					Overall		
	D1	D2	D3	D4	D5			
Wang 2003	+	+	+	+	+	+	+	Low risk Some concerns High risk
Oelkers-Ax 2007	+	+	+	+	+	+	+	
Rapoff 2014	+	+	+	+	!	+	+	
Fentress 1986	!	!	+	+	!	+	+	D1 Randomisation process D2 Deviations from the intended interventions D3 Missing outcome data D4 Measurement of the outcome D5 Selection of the reported result
Pintov 1997	!	+	+	+	+	+	!	
Allen 1998	!	!	+	+	+	+	+	
McGrath 1988	+	+	+	+	+	+	+	D1 Randomisation process D2 Deviations from the intended interventions D3 Missing outcome data D4 Measurement of the outcome D5 Selection of the reported result
Koenig 2013	+	+	+	+	+	+	+	
Richter 1986	+	+	+	+	!	+	+	
MacLennan 2008	+	+	+	+	+	+	+	
	+	+	+	+	+	+	+	

Fig. 2.1. Risk of bias summary for Parallel RCT (Intention-to-Treat).

Study ID	PER PROTOCOL					Overall		
	D1	D2	D3	D4	D5			
McGrath 1991	!	!	-	-	!	-	+	Low risk
Fichtel 2000	!	!	-	-	!	-	!	Some concerns
Sartory 1998	!	!	-	-	!	-	-	High risk
Labbe 1984	+	+	+	-	+	-		
Labbe 1994	+	+	+	-	+	-		
Scharff 2002	!	!	+	-	!	-		
							D1	Randomisation process
							D2	Deviations from the intended interventions
							D3	Missing outcome data
							D4	Measurement of the outcome
							D5	Selection of the reported result

Fig. 2.2. Risk of bias summary for Parallel RCT (Per-Protocol).

Study ID	D1	D5	D2	D3	D4	D5	Overall		
PP Harel 2001	+	+	+	+	+	+	+	+	Low risk
ITT Slater 2011	+	-	+	-	+	+	-	!	Some concerns
								-	High risk
								D1	Randomisation process
								D5	Bias arising from period and carryover effects
								D2	Deviations from the intended interventions
								D3	Missing outcome data
								D4	Measurement of the outcome
								D5	Selection of the reported result

Fig. 2.3. Risk of bias summary for Crossover RCT.

3.6 Outcome measures

The included studies primarily utilized headache diaries as tools for outcome assessment. These diaries enabled participants to record key migraine parameters, including frequency, intensity, and duration of episodes. In most cases, these parameters were evaluated using standardized scales, such as Likert or numeric severity scales, ensuring consistency across studies. Among the studies, 14 out of 18 defined clinical improvement as a reduction in headache frequency by $\geq 50\%$, which was considered the primary outcome measure. When this primary measure was unavailable, alternative metrics parameters as changes in frequency, intensity and duration were adopted for analysis. Additional measures, like headache indices combining both frequency and severity, were also used in five studies to provide treatment impact to the participant.

The reliance on self-reported data, such as headache diaries, underscores the subjective nature of these outcomes. Although effective for capturing individual experiences, these methods were sometimes affected by recall bias and variability in participant compliance. Future research may benefit from integrating more objective measures, such as physiological assessments, to enhance data reliability. Moreover, all the findings highlight the promise of non-pharmacologic strategies in alleviating migraine frequency, severity, and duration, while underscoring the need for cautious interpretation and further research.

3.7 Efficacy analysis

3.7.1 Migraine frequency

Migraine frequency (Table 4) was the most consistently reported outcome, assessed in 16 of the 18 trials presented as the mean number of migraine days per month unless stated before and after interventions. Across short term analyses, acupuncture demonstrated marked efficacy, reducing monthly frequency from 9.3 to 1.4 episodes. Behavioral interventions such as relaxation therapy and biofeedback demonstrated significant reductions, typically cutting episode frequency by one to two attacks per week – these improvements were often statistically significant and sustained in long term follow ups, with several trials reporting continued benefit up to 6–12 months. Nutraceuticals showed mixed results: butterbur root extract achieved about a one-third reduction in attacks with some durability at six months, Coenzyme Q10 yielded promising but less consistent effects, while riboflavin and magnesium oxide did not outperform placebo.

Overall, the evidence indicates that relaxation therapy and biofeedback provide the most reliable short and long-term efficacy in reducing migraine frequency among pediatric patients, while nutraceuticals offer potential but heterogeneous benefits.

3.7.2 Migraine severity

Migraine severity was assessed in 12 of the 18 trials (Table 5), using point scales (e.g., 0-4, 0-5, 0-6, 0-7, 0–10). Behavioral interventions, particularly relaxation therapy, consistently produced significant short-term reductions in severity, with benefits sustained in several long-term follow-ups. Biofeedback and CBT showed moderate improvements, though results were less consistent across studies. Acupuncture also demonstrated significant reductions, reinforcing its potential as a non-pharmacologic option.

Nutraceuticals produced mixed outcomes, fish oil and Coenzyme Q10 showed notable short-term improvements, though durability was inconsistent, while riboflavin and butterbur root extract did not demonstrate significant benefit compared to controls. Music therapy likewise failed to show long-term differences. Overall, severity outcomes suggest that relaxation therapy and acupuncture provide the most consistent efficacy, selected nutraceuticals offer potential but variable effects, and other interventions show limited durability.

3.7.3 Migraine duration

Migraine duration was reported in nine trials, measured as average hours per episode (Table 6). Behavioral interventions, particularly cognitive coping, relaxation therapy and biofeedback, consistently shortened attack length in the short term, often by several hours, with benefits sustained in some long-term follow-ups up to 6–12 months. Nutraceuticals did not demonstrate significant benefit. Duration outcomes highlight relaxation therapy, and biofeedback as the most reliable strategies for reducing attack length, whereas only CoQ10 shows only a significant difference within group. These findings emphasize the potential of non-pharmacologic interventions to not only reduce migraine frequency but also shorten episode duration, though further rigorous trials are needed to confirm long-term efficacy.

Table 4. Migraine frequency (mean days per month unless stated) outcome of the included journal article.

Study ID	I/C	Baseline (Mean, $\pm$ SD, n)	Post- treatment/ Post- intervention (PI)	Follow-up	Simplified Outcome (Decrease, Increase, or No effect; significant or not)		P value		Confidence Interval
					Within group (WG)	Between group/s (BW)	WG	BW	
*Labbe 1984	I1	2.59 n = 14	0.89 n = 14	1 mos (n = 14) = 0.91 6 mos (n = 8): 1.79	Decrease, significant		NR		
	C	2.64 n = 14	2.68 n = 14	1 mo (n = 14): 2.57 6 mos (n = 5): NR	Increase at PI but decrease at 1 mo follow up, NS	Significant difference		P < 0.05	95%
Richter 1985	I1	9.03 $\pm$ 8.05 n = 15	5.17 $\pm$ 5.16 n = 15	4 mos (n = 15): 2.91 $\pm$ 3.40	Decrease, significant		P < 0.05		
	I2	8.14 $\pm$ 7.82 n = 15	4.50 $\pm$ 5.29 n = 15	4 mos (n = 15): 2.52 $\pm$ 2.94	Decrease, significant	Significant difference (I1 & I2 x C)	P < 0.025	P < 0.05	95%
	C	7.26 $\pm$ 6.12 n = 12	6.45 $\pm$ 6.09 n = 12	4 mos (n = 12): 4.68 $\pm$ 5.83	Decrease, NS		NR		
*Fentress 1986	I1	2.7 n = 6	1.0 n = 6	1 yr (n = 5): 1.1	Decrease, significant	Significant difference (I2 x C)	P < 0.01	I1 x I2: NS	
	I2	2.6 n = 6	0.4 n = 6	1 yr (n = 5): 0.4	Decrease, significant	No significant difference	P < 0.01	I1 x C: P > 0.07 I2 x C: P < 0.01	95%
	C	2.3 n = 6	2.2 n = 6	1 yr: NR	Decrease, NS	(I1 x I2 & I1 x C)	NS		
*Labbe 1994	I1	2.87 n = 10	0.60 n = 10	1 mos (n = 10): 0.05 6 mos (n = 10): 0.12	Decrease, significant	Significant difference (I1 & I2 x C)	P < 0.001	P < 0.01	95%

	I2	3.67 n = 10	1.00 n = 10	1 mos (n = 10): 0.38 6 mos (n = 10): 0.42	Decrease, significant		P < 0.05		
	C	3.18 n = 10	2.35 n = 10	1mos (n = 10): 2.17 6 mos (n = 10): 2.52	Decrease, not significant		NS		
Pintov 1997	I1	9.3 ±1.6 n = 12	1.4 ±0.6 n = 12	NR	Decrease, significant	Significant difference	NR	NR	95%
	C	9.4 ±1.5 n = 10	9.3 ±1.4 n = 10	NR	No effect, not significant		NR		
Allen 1998	I1	4.2 ±2.4 n = 13	3.3 ±2.9 n = 13	1 mo (n = 13): 3.4 ±2.9 3 mos (n = 13): 2.2 ±1.4 1 yr (n = 10): 1.6 ±1.6	Decrease significant	No significant difference	P ≤ 0.05	NS	95%
	C	4.1 ±2.2 n = 14	1.5 ±2.3 n = 14	1 mo (n = 14): 0.9 ±1.9 3 mos (n = 14): 0.8 ±0.9 1 yr (n = 11): 0.9 ±0.9	Decrease, significant		P ≤ 0.05		
	I2	1.75 ±1.44 n = 15	0.82 ±1.15 n = 15	2 wks (n = 11): 1.14 ±1.19	Decrease, significant		P < 0.01		
	I2	1.73 ±1.25 n = 15	1.73 ±1.25 n = 15	2 wks (n = 10): 1.05 ±0.72	Decrease, significant		P < 0.02		
*Sartory 1998	C	1.46 ±0.9 n = 13	1.03 ±0.78 n = 13	2 wks (n = 6): 1.25 ±0.82	Decrease, not significant	No significant difference (I1 x I2 & I2 x C)	NS	NS	95%
Fichtel 2000	I1	1.85 ±2.1 n = 21	1.07 ±1.8 n = 20	NA	Decrease, significant	Significant difference	P < 0.05	P < 0.05	95%
	C	1.10 ±0.8 n = 16	0.77 ±0.7 n = 16	NA	Decrease, not significant		NS		

	Fish oil x Placebo	15.5 ±2 n = 14	2 ±0.5 n =14	NA	Decrease, significant	P < 0.001		
Harel 2001						No significant difference	NS	95%
	Placebo x Fish oil	15.5 ±2, n = 13	2 ±0.5, n = 13	NA	Decrease, significant	P < 0.001		
	I1	11.3 ±3 n = 13	53.8% achieved improvement (≥ 50% reduction)	3 mos: 72.2% had ≥50% reduction 6 mos: 100% maintained clinical improvement	Decrease, significant	P < 0.002		
Scharff 2002	I2	9.5 ±4.7 n =11	10% achieved improvement (≥ 50% reduction)	3 mos: 33% had ≥50% reduction 6 mos: 62.5% maintained clinical improvement	Decrease, not significant	Significant difference (I1 x I2&C) P < 0.002	P < 0.05	95%
	C	12.0 ±3.2 n = 12	None achieved improvement	NR	No effect, not significant	NS		
	I1	9.3 n = 60	Starts at around 0.38 (38%) and decreases steadily over time to approximately 0.15 (15%) by the end of txn	NA	Decrease, significant	P = 0.0037		
Wang 2003						No significant difference	P = 0.88	95%
	C	11.5 n = 58	Starts at about 0.36 (36%) and decreases to roughly 0.25 (25%) by week 6 but then plateaus	NA	Decrease, not significant	P = 0.086		

Oelkers-Ax 2007	I1	9.8 ±7.6 n = 19	36.1% ±57.3 reduction from baseline, n = 19	6 mos (n = 15): 58.7% ±34.6 reduction	Decrease, significant	Significant difference (I2 x C)	P < 0.05	I2 x C: P = 0.005	95%
	I2	5.0 ±2.5 n = 20	65.7% ±31.0 reduction from baseline, n = 17	6 mos (n = 17): 63.2% ±33.9 reduction	Decrease, significant	No significant difference (I1 x C; I1 x I2)	P < 0.05	I1 x C (PI): P = 0.159	
	C	5.5 ±4.4 n = 19	28.8% ±39.5 reduction from baseline, n = 19	6 mos (n = 18): 31.4% ±41.8 reduction	Decrease, significant	Significant difference at follow up (I1 x C)	P < 0.05	I1 x I2: P = 0.124 I1 x C (FU): P = 0.044	
MacLennan 2008	I1	4.4 ±1.9 n = 27	response rate (50% or greater reduction) was 44.4% (12/27)	NA	Decrease, NR	No significant difference	NR	P = 0.125	95%
	C	4.2 ±2.0 n = 21	Response rate was 66.6% (14/21)	NA	Decrease, NR		NR		
Slater 2011	Q10 x Placebo	14.3 ±6.0 n = 44	Wks 1-4: 8.2 ±5.6	NA	Decrease, significant	Wks 1-4; 17- 20: Significant difference	P < 0.001	Wk 1-4: P = 0.03	95%
			Wks 13-16: 5.8 ±5.2, n = 33					Wk 13- 16: P = 0.15	
			Wks 17-20: 6.6 ±5.7, n = 26					Wk 17- 20: P = 0.01	
	Placebo x Q10	15.4 ± 7.5 n = 42	Wk 29-32: 4.0 ±3.3, n =18	NA	Decrease, significant	Wks 13-16; 29-32: No significant difference	P < 0.001	Wk 29- 32: P = 0.19	
			Wks 1-4: 10.8 ±7.4					Wk 1-4 vs 29-32: P > 0.05	
			Wks 13-16: 7.3 ±6.2, n = 28						
			Wks 17-20: 6.3 ±6.7, n = 27						
			Wk 29-32: 4.6 ±6.1, n = 24						

Koenig 2013	I1	18.5 ±8.4 n = 40	15.4 ±9.8 n = 40	6 mos (n = 40): 14.7 ±9.9	Decrease, not significant	No significant difference	P = 0.37	P = 0.37	95%
	C	17.1 ±8.1 n = 38	12.6 ±7.9 n = 38	6 mos (n = 38): 12.7 ±8.6	Decrease, not significant		P = 0.37		
Rapoff 2014	I1	41.09% ±22.67% n = 18	31.28% ±28.24% n = 18	3 mos (n = 11): 21.43% ±23.47%	Decrease, not significant	No significant difference	NS	P=0.46	95%
	C	40.67% ±28.79% n = 17	32.14% ±22.23% n = 17	3 mos (n = 11): 18.18% ±17.16%	Decrease, not significant		NS		

Abbreviations:

I1 = Intervention 1; I2 = Intervention 2; C = Comparator; n = sample size; NR = not reported; BG = between groups; WG = within groups; NS = not significant; NA = not applicable; txn = treatment

*study id = frequency/week

Wang = Proportion (graph) of days with headaches over time; numerical value not reported

Table 5. Migraine severity (0-10 point scale unless stated) outcome of the included journal articles.

Study ID	I/C	Base-line (Mean, ± SD, n)	Post-treatment/ Post-intervention (PI)	Follow-up	Simplified Outcome (Decrease, Increase, or No effect; significant or not)		P value		Confidence Interval
					Within group	Between group/s	WG	BW	
Labbe 1984	I1	3.23 n = 14	1.14 n = 14	1 mo (n = 14): 1.04 6 mos (n = 8): 2.13	Decrease, significant	Significant difference	P < 0.001	P < 0.05	NR
	C	3.33 n = 14	3.43 n = 14	1 mo (n = 14): 3.23 6 mos (n = 5): NR	No effect, not significant		NS		
Richter 1985	I1	3.60 ±1.08 n = 15	2.52 ±1.19 n = 15	4 mos (n = 15): 2.08 ±1.73	Decrease, not significant	No significant difference	NS	NR	NR
	I2	3.37 ±0.77 n = 15	2.52 ±1.14 n = 15	4 mos (n = 15): 1.96 ±1.23	Decrease, not significant		NS		

	C	3.58 ±0.76 n = 12	2.39 ±1.33 n = 12	4 mos (n = 12): 2.02 ±1.39	Decrease, not significant		NS		
Pintov 1997	I1	8.7 ±0.4 n = 12	3.3 ±1.0 n = 12	NA	Decrease, significant	Significant difference	P < 0.01	P < 0.001	NR
	C	7.8 ±0.6 n = 10	6.2 ±0.4 n = 10	NA	Decrease, not significant		P > 0.05		
Sartory 1998	I1	5.24 ±1.18 n = 11	3.37 ±2.68 n = 11	2 wks: 2.98 ±2.65	Decrease, significant		P < 0.02		
	I2	4.65 ±2.57 n = 10	3.37 ±2.68 n = 10	2 wks: 2.98 ±2.65	Decrease, not significant	No significant difference	NS	NS	NR
	C	4.65 ±2.57 n = 6	5.02 ±2.22 n = 6	2 wks: 4.19 ±2.42	Increase, not significant		NS		
*Fichtel 2000	I1	3.35 ±0.7 n = 21	2.43 ±1.2 n = 20	NA	Decrease, significant	Significant difference	P < 0.05	P < 0.05	NR
	C	2.45 ±1.1 n = 16	1.80 ±1.2 n = 16	NA	Decrease, not significant		NS		
**Harel 2001	Fish oil x Placebo	5.0 ±0.3 n = 14	2.9 ±0.5	NA	Decrease, significant	No significant difference	P < 0.01	NS	NR
	Placebo x Fish oil	5.0 ±0.3 n = 13	3.5 ±0.4 n = 13	NA	Decrease, significant		P < 0.03		
***Scharff 2002	I1	3.6 ±0.8 n = 13	2.5 ±0.8 n = 13	3 & 6 mos: sustained improvement	Decrease, significant	Significant difference	P < 0.01	P < 0.05	NR

	I2	3.4 ±1.0 n = 11	2.8 ±1.0 n = 11	3 & 6 mos: moderate improvement	Decrease, significant	(I1 x I2&C)	P < 0.01		
	C	3.4 ±1.0 n = 12	3.4 ±1.0 n = 12	3 & 6 mos: No improvement	No effect, not significant		NS		
Wang 2003	I1	NR	The magnesium oxide group had significantly lower headache severity relative to the placebo group.	NA	Decrease, significant	NR	P = 0.0029	NR	95%
	C	NR		NA	Decrease, not significant		NS		
****Oelkers- Ax 2007	I1	4.9 ±1.7 n = 19	21.1% reduction n = 19	33.3% reduction, n = 15	Decrease, not significant	No significant difference	NS	P = 0.505	NR
	I2	4.6 ±1.5 n = 20	5.9% reduction n = 17	29.4% reduction, n = 17	Decrease, not significant		NS		
	C	5.0 ±1.4 n = 19	15.8% reduction n = 19	22.2% reduction, n = 18	Decrease, not significant		NS		
Slater 2011	Q10 x Placebo	6.4 ±1.8 n = 43	Wk 1-4: 4.187, n = 30 Wk 13-16: 3.387, n = 30	NA	Decrease, significant	No significant difference	P = 0.006	P > 0.05	95%
	Placebo x Q10	6.2 ±1.7 n = 39	Wk 1-4: 4.706, n = 26 Wk 13-16: 4.035, n = 26	NA	Decrease, significant		P = 0.006		
Koenig 2013	I1	5.0 ±1.6 n = 40	4.7 ±1.7 n = 40	6 mos (n = 40): 4.8 ± 1.7	Decrease, not significant	No significant difference	NS	P = 0.42	
	C	4.7 ±1.3 n = 38	4.7 ±1.1 n = 38	6 mos (n = 38): 4.7 ± 1.3	No effect, not significant		NS		

Rapoff 2014	I1	5.06 ±1.84 n = 18	5.06 ±1.50 n = 18	3 mos (n = 11): 4.46 ±1.88	No effect, not significant	Significant difference	NS	P = 0.03	95%
	C	6.00 ±1.52 n = 17	6.25 ±1.92 n = 17	3 mos (n = 11): 3.68 ±2.04	Increase, not significant		NS		

*study id = 0 - 5 point scale; **0 - 7 point scale; ***0 - 4 point scale; ****0 -6 point scale

Table 6. Migraine duration (average hour per episode of migraine attack unless stated) outcome of the included journal articles.

Study ID	I/C	Base- line (Mean, ± SD, n)	Post- treatment/ Post- intervention (PI)	Follow- up (FU)	Simplified Outcome (Decrease, Increase, or No effect; significant or not)		P value		Confidence Interval
					Within group	Between group/s	WG	BW	
Labbe 1984	I1	6.19 n = 14	2.21 n = 14	1 mo (n = 14): 2.19 6 mos (n = 8): 0.82	Decrease, significant	Significant difference	P < 0.01	P < 0.001	95%
	C	7.12 n = 14	7.92 n = 14	1 mo (n = 14): 7.38 6 mos (n = 5): NR	Increase, not significant		NS		
Richter 1985	I1	1.80 ±0.92 n = 15	1.36 ±0.65 n = 15	4 mos (n = 15): 0.88 ±0.80	Decrease, significant	Significant difference (I1 & I2 x C)	P < 0.05	P < 0.05	95%
	I2	1.81 ±0.83 n = 15	1.34 ±0.87 n = 15	4 mos (n = 15): 0.89 ±0.68	Decrease, significant		P < 0.05		
	C	1.68 ±0.61 n = 12	1.45 ±0.89 n = 12	4 mos (n = 12): 1.08 ±0.81	Decrease, not significant		P > 0.05		
*Fentress 1986	I1	9.7 n = 6	4.1 n = 6	1 yr (n = 5): 2.4	Decrease, significant		P < 0.01	P < 0.05	95%

	I2	14.6 n = 6	1.0 n = 6	1 yr (n = 5): 1.4	Decrease, significant	Significant difference (I1 & I2 x C)	P < 0.01		
	C	6.3 n = 6	7.0 n = 6	1 yr (n = 5): NR	Increase, not significant		NS		
Labbe, 1994	I1	5.87 n = 10	2.0 n = 10	1 mo (n = 10): 0.30 6 mos (n = 10): 0.30	Decrease, significant	Significant difference (I1 & I2 x C)	P < 0.001		
	I2	6.71 n = 10	2.36 n = 10	1 mo (n = 10): 1.25 6 mos (n = 10): 1.30	Decrease, significant		P < 0.001	P < 0.02	95%
	C	3.92 n = 10	3.71 n = 10	1 mo (n = 10): 5.10 6 mos (n = 10): 4.80	Decrease, not significant		P > 0.05		
Sartory 1998	I1	11.74 ±12.47 n = 11	7.05 ±12.81 n = 11	6.68 ±6.38	Decrease, significant	No significant difference	P < 0.01		
	I2	8.32 ±5.42 n = 10	3.01 ±3.60 n = 10	6.27 ±5.82	Decrease, significant		P < 0.05	P > 0.05	95%
	C	10.9 ±5.54 n = 6	6.61 ±6.7 n = 6	7.82 ±3.52	Decrease, not significant		NS		
*Fichtel 2000	I1	4.05 ±6.8 n = 21	2.54 ±6.6 n = 20	NA	Decrease, significant	No significant difference	P < 0.05		
	C	2.13 ±5.3 n = 16	1.59 ±3.4 n = 16	NA	Decrease, not significant		NS		
MacLennan 2008	I1	4.58 n = 27	5.25 n = 27	NA	Increase, not significant	No significant difference	NR	P > 0.05	95%

	C	4.32 n = 21	5.27 n = 21	NA	Increase, not significant		NR		
Slater 2011	Q10 x Placebo	10.1 ±16.9 n = 43	Wk 1-4: 3.698, n = 27 Wk 13-16: 2.666, n = 27	NA	Decrease, significant		P = 0.016		
						No significant difference at any time point	P > 0.05	95%	
	Placebo x Q10	8.3 ±8.8 n = 38	Wk 1-4: 3.763, n = 20 Wk 13-16: 3.001, n = 20	NA	Decrease, significant		P = 0.016		
Rapoff 2014	II	5.47 ±4.20 n = 18	4.47 ±4.26 n = 18	1.53 ± 0.91, n = 11	Decrease, not significant		NS	PI: P = 0.24	
	C	4.15 ±3.88 n = 17	5.56 ±4.01 n = 17	4.25 ± 5.19, n = 11	Increase, not significant	No significant difference	NS	FU: P=0.07	95%
*study id = hrs/week									

4. DISCUSSION

A migraine is not just a simple headache; instead, it is one of the most common debilitating neurological disorders that negatively contributes to the lives of millions of children across the world. An estimated 7-10% of the pediatric population endure acute and chronic migraines, with the adolescent population capturing the majority of cases due to multiple factors including hormonal variations, educational pressures, and psychosocial obstacles, which are among the key factors contributing to the onset, progression, and exacerbation of migraines (Labbé & Williamson, 1984; McGrath et al., 1988). The advent of the modern era has not only introduced but also exacerbated the risk factors for migraines, including extended screen exposure, disrupted sleep patterns, and dietary practices—such as heightened caffeine consumption and the omission of meals—which all play a role in amplifying migraine incidence in the youth of today (Scharff, 2002; Wang et al., 2003).

The consequences of these practices leading to migraine development extend beyond pain, since migraines also impair school performance, social interactions, and emotional well-being, as well as drastically maneuvering the trajectory of a child's path into their transition to adulthood, especially if unmanaged (McGrath et al., 1992; Pintov et al., 1997). Although pharmacologic interventions remain as

the frontline options (Triptans and NSAIDs), their limitations pose a significant problem for the younger population due to side effects, variable efficacy per person, and the risk of medication abuse and overuse (MacLennan et al., 2008; Oelkers-Ax et al., 2008). Hence, these problems call for the need for urgent alternative strategies that would make the battle against migraine winnable. In this systematic review, the researchers synthesized evidence from 18 RCTs to evaluate the efficacy and safety of non-pharmacologic interventions for pediatric migraine, which shall offer a new and comprehensive perspective on their possible roles as standalone interventions or adjunct therapies (Allen & Shriver, 1998; Pintov et al., 1997).

Each key finding presented in this review supports the efficacy of specific non-pharmacological interventions in the significant reduction of pediatric migraines. Methods like relaxation therapy, CBT, biofeedback, and dietary supplementation support significant reduction in pediatric migraine frequency, severity, and duration, which is consistent with literature from the past (Fichtel & Larsson, 2001; Oelkers-Ax et al., 2008; Wang et al., 2003). However, this paper fills a critical gap in previous studies regarding its effect on the pediatric migraineur population (Slater et al., 2011). Additionally, the evidence given in these studies further affirms not only the short-term efficacy of the interventions but also highlights their mid- to long-term efficacy (Labbé, 1995; Rapoff et al., 2014). Hence, this paper compels further research in the future regarding trials that have better and more standardized reporting, with limited self-reporting and extended follow-up durations (MacLennan et al., 2008; Scharff, 2002). Clinically, given the low probability of adverse events based on the safety profiles of each intervention, the validity of using these interventions in the pediatric population is more heavily reinforced, especially since their efficacy has already supported previous adult-focused studies (Harel et al., 2002; Oelkers-Ax et al., 2008).

Although we aggregated our crucial insights and information from a total of 18 studies, a meta-analysis was not conducted. The conduction of a systematic review instead of a complete meta-analysis was a methodological decision the researchers made in order to avoid result misinterpretation. A few notable reasons for this decision stemmed from obvious heterogeneity across studies in terms of their outcome measures, treatment protocols, sample sizes, and follow-up durations (Koenig et al., 2013; Richter et al., 1986). Furthermore, the high presence of significant risk of bias in two-thirds of studies abated the possibility of pooling results to have an accurate and reliable statistical analysis (Rapoff et al., 2014). Thus, the use of a qualitative analysis was preferred to prevent result misinterpretation and uphold contextual integrity.

Considering these risks, it is advisable that upcoming trials implement stricter methodological standards, such as utilizing larger and more varied sample sizes, applying uniform diagnostic criteria, and incorporating both subjective and objective measures of outcomes (Allen & Shriver, 1998; Wang et al., 2003). Researchers in the future should also emphasize clear reporting of adverse events and employ stratified analyses to evaluate differing responses based on age, gender, or migraine subtype (Page et al., 2021; Slater et al., 2011).

4.1 The promising trajectory of non-pharmacologic interventions

Non-pharmacologic interventions propose a better path towards treating migraines. Some of which may serve as complete replacements for the traditional medications we all use today while some represent a complementary adjunct towards making migraine treatment more feasible especially for pediatrics (Labbé & Williamson, 1984; Richter et al., 1986). These interventions, including relaxation therapy, CBT, biofeedback, acupuncture, and dietary supplements, all address the intricate pathophysiology of migraine.

Most of these methods target stress, neurovascular dysregulation, and nutritional deficiencies without suffering from the systemic side effects of pharmacologic alternatives (Fichtel & Larsson, 2001; Sartory et al., 1998). Dietary supplements are hypothesized to influence nutritional and metabolic processes, yet the included RCTs focused only on clinical outcomes (frequency, severity, duration) and did not measure nutritional deficiencies or metabolic biomarkers (Harel et al., 2002; Slater et al., 2011). Future studies should incorporate these assessments to clarify mechanisms and identify subgroups most likely to benefit.

As an instance, a study (Labbé & Williamson, 1984) suggested that relaxation techniques like progressive muscle relaxation and autogenic training produced significant reductions in migraine frequency from 2.59 to 0.89 episodes per week. This was induced by the modulation of stress response, which is one of the most well-established migraine triggers (Fichtel & Larsson, 2001; Koenig et al., 2013). Additionally, another non-pharmacologic intervention in the form of biofeedback gave way for children to gain voluntary control over physiological processes which yielded improvements in migraine attack duration through the use of thermal and electromyographic (EMG) biofeedback mechanisms. This reduced migraine duration from 9.7 hours to 4.1 hours (Fentress et al., 1986).

The utilization of dietary supplements also showed promise in several controlled pediatric trials by Wang et al. (2003). Magnesium oxide was administered to children and resulted in neuronal stabilization as well as vasoregulation, thus reducing migraine severity and frequency. Butterbur extract achieved a 36.1% reduction in attacks (Oelkers-Ax et al., 2008). These findings suggest possible roles in neuronal excitability and vascular regulation. However, the included trials did not assess whether these effects were linked to correcting nutritional deficiencies or metabolic imbalances (Harel et al., 2002; Slater et al., 2011).

Emerging evidence from other studies points to gut-brain axis and metabolic imbalances as potential contributors to migraine risk, underscoring the need for future trials to incorporate these measures (Harel et al., 2002; Slater et al., 2011). Apart from dietary supplements, migraines were also managed via coping strategies used to mitigate pain and stress, such as the CBT structured as Headstrong CD-ROM program, which further demonstrated long-term efficacy in reducing reliance on pharmacological treatments for pediatric migraine (Rapoff et al., 2014).

4.2 Comparative advantages over pharmacologic therapies

Out of the 18 reviewed studies, only three studies reported mild side effects which primarily involved gastrointestinal discomfort from supplements (MacLennan et al., 2008; Oelkers-Ax et al., 2008; Wang et al., 2003). Hence, when compared to traditional drug treatments for pediatric migraines which often cause drowsiness, GI distress, and tolerance issues, these non-drug therapies offer a superior safety profile. This makes these alternatives suitable for pediatric populations, especially when used long-term, due to concerns regarding the developmental and cognitive impacts of medication on the growth and development of children (Allen & Shriver, 1998; Pintov et al., 1997).

On top of previously mentioned advantages, non-pharmacological alternatives encourage active patient engagement and self-management since interventions like CBT and biofeedback encourage lifelong learning and coping strategies which reduce medication dependency (McGrath et al., 1992; Rapoff et al., 2014). This is in contrast to pharmacological therapies which, in most instances, require consistent uninterrupted supervision from medical practitioners in order to be effective (Fichtel & Larsson, 2001;

Sartory et al., 1998). Additionally, accessibility is also a key advantage of non-pharmacologic treatments like the Headstrong program, which is digital and thus has better scalability (Rapoff et al., 2014). This not only enhances accessibility for patients but also provides future-proofing toward migraine treatments being accessible anywhere at any time. This is particularly crucial in low-income settings where specialized care is limited (Oelkers-Ax et al., 2008; Wang et al., 2003). These methodologies are more congruent with patient preferences, as articulated by Xu et al. (2021), who identified that patients favor autonomy and minimal invasiveness, resulting in reduced side effects and enhanced convenience.

4.3 Clinical implications: Adjunct or alternative?

All evidence points that the aforementioned non-pharmacologic intervention may be intended to serve its purpose as both complete alternatives and complementary adjuncts to pharmacologic therapy (Labbé & Williamson, 1984; Richter et al., 1986). Milder and moderate types of migraines may be treated non-pharmacologically through relaxation techniques or supplements which would often suffice as first-line non-pharmacologic treatments therefore avoiding the various drug-related side effects (Fentress et al., 1986; Harel et al., 2002). More severe and refractory cases may involve the use of medications with non-pharmacologic therapies combinedly called low-dose preventatives such as amitriptyline with CBT (Powers et al., 2013). These combinations have the advantage of enhancing treatment efficacy while minimizing medication mishaps (Scharff, 2002). This combined approach offer a pragmatic middle-ground for clinicians hesitant to move entirely away from pharmacotherapy. This integrated approach balances efficacy with safety, reducing long-term medication burden (Powers et al., 2013) while reinforcing the role of non-pharmacologic therapies as essential components of individualized, stepwise care in pediatric migraine management (Labbé & Williamson, 1984; Richter et al., 1986).

In the future, we suggest researchers prioritize large and more rigorous RCTs that have standardized protocols as well as long term follow-ups (Koenig et al., 2013; Rapoff et al., 2014). An improvement to be made also would be the incorporation of objective biomarkers like cortisol assays and serum magnesium levels along with patient-reported outcomes like quality-of-life metrics as these would further substantiate the interventions we have reviewed (MacLennan et al., 2008; Wang et al., 2003). Future research should include interaction studies on combined pharmacologic and non-pharmacologic treatments to better inform integrated care models. At present, non-pharmacologic therapies remain safe and effective, and are expected to play an increasingly central role in pediatric migraine management.

In closing the divide of evidence and application, this review urges the need for a major shift in pediatric migraine care: pediatric migraines should not be managed with drugs alone but instead with safe, better non-invasive alternatives (Labbé, 1995; Sartory et al., 1998). From CBT to supplementation, the future of pediatric migraine treatment lies in the human body's capacity for healing without carrying the weight of side effects and dependency (Fichtel & Larsson, 2001; Oelkers-Ax et al., 2008).

4.4 Comparison with current literature

The results of this systematic review showcased consistency with review and meta-analyses in the past which also showed the efficacy of various non-pharmacological methods and interventions responsible for reducing the frequency of migraine, their severity, and their duration (McGrath et al., 1988; Wang et al.,

2003). However, there is still a high risk of bias in many of the included studies which is very well limiting to the generalizability of these findings (Labbé & Williamson, 1984; Richter et al., 1986). Additionally, this review calls for the need to have more rigorous Randomized Controlled Trials with bigger sample sizes as well as longer follow-up periods to confirm if the efficacy and safety results of previous studies are indeed accurate and a good basis for future studies (Fentress et al., 1986; Scharff, 2002).

The outcomes reported here in this review are consistent with the parameters used by other reviews and articles used in migraine research which mainly include frequency, severity, and duration (Harel et al., 2002; McGrath et al., 1992). While the results here simplify reporting of the results, it is somewhat obscure that the individual contributions of frequency and severity outcomes do not provide detailed results on how they interfere with each other (Koenig et al., 2013; Sartory et al., 1998). An example (McGrath et al., 1988), which reported a significant decrease in the headache index which ranges from 53.5 ± 24.1 to 32.3 ± 22.1 , $p < 0.05$ without providing any detailed information on how the two parameters were independently affected (McGrath et al., 1988). Future studies must from now on, consider reporting not only combine frequency and severity metrics but also both of these combines to provide a better understanding of the efficacy of the given intervention (Rapoff et al., 2014; Slater et al., 2011).

This review also provides significant comparative insights alongside the systematic review and meta-analysis conducted by Koechlin et al. (2021). Although both studies concentrated on pediatric populations, a key difference lies in Koechlin et al. focusing exclusively on episodic migraine, while this review did not impose such limitations, thus offering wider clinical relevance. In terms of methodology, Koechlin et al. adopted a meta-analytic approach utilizing both splitting and lumping methods, whereas our study implemented Synthesis Without Meta-analysis (SWiM), opting for a narrative synthesis approach that is more appropriate for the qualitative diversity and heterogeneity observed across the studies included. It is important to note that while Koechlin et al. expressed a desire to assess safety and tolerability, none of the studies they reviewed reported safety-related outcomes. In contrast, this review encompasses research—particularly those related to nutraceuticals—that presents pertinent safety information, facilitating a more thorough evaluation of both effectiveness and adverse effects. Additionally, our search approach went beyond the four conventional databases (Medline, Cochrane, Embase, PsycINFO) utilized by Koechlin et al. by including citation chaining, Google Scholar, and data from relevant organizations.

In terms of study overlap, nine of the articles included in our review are also featured in the Koechlin review. However, five articles from their review are not present in ours; upon further examination, it was determined that four of these articles are appropriate for inclusion, while one was excluded due to insufficient subgroup data on migraine outcomes. Conversely, our review includes five studies, particularly those investigating nutraceuticals, that are absent from their analysis, highlighting a significant area of contribution in our synthesis. Therefore, while both reviews affirm the effectiveness of non-pharmacologic interventions, our study offers a broader scope and a greater emphasis on safety.

4.5 Strengths and limitations

All outcomes used in the studies are clinically relevant and align with patient-reported experiences, making them meaningful for practice. Metrics such as frequency, severity, and duration are simple, widely accepted, and facilitate comparison across studies. The value of this review to the existing knowledge is multifaceted: it not only brings together scattered information regarding non-drug treatments for pediatric

migraine but also offers a clear framework for clinicians to explore integrative and safer therapies as primary or complementary options.

Limitations include reliance on self-reported data, which may introduce recall bias or compliance variability. While the migraine index is a useful composite measure, it may mask the distinct contributions of attack frequency and severity. In addition, the limited availability of long-term data constrains conclusions regarding sustained efficacy. Future studies should continue using standardized parameters for frequency, severity, and duration, while also incorporating quality of life, functional disability, and physiological assessments. The migraine index should complement, but not replace, specific outcome measures.

5. CONCLUSION

The results of this review suggest that notable non-pharmacological interventions – particularly relaxation therapy, biofeedback, CBT, acupuncture, and selected dietary supplements – used in the treatment of pediatric migraine can be an effective form of migraine management in children and adolescents. These have shown generally favorable safety profiles with only mild, self-limiting adverse events reported and to be good adjuncts and even alternatives to present pharmacological treatments especially in those cases where medications are not well tolerated or even contraindicated to the patients. Relaxation therapy, biofeedback, and certain dietary supplements are notably better than some in managing migraine but the choice of the intervention to be used for a particular population or person should always be individualized based on the patient's needs, the severity of their condition, preferences, and the resources that they have access. Healthcare providers should consider non-pharmacologic interventions as first-line or adjunct therapy for pediatric migraine for a drug-free approach. Incorporate relaxation techniques as they demonstrated efficacy in reducing migraine frequency and minimal risk. Moreover, clinicians should individualize treatment plans based on patient characteristics and preferences. CBT for children with frequent or disabling migraines is recommended because of its long-term effects observed.

Current findings provide valuable insights into reducing reliance on medications and enhancing patient safety, the limited number and heterogeneity of trials mean conclusions remain preliminary. Future high-quality RCTs with standardized protocols, long-term follow-up, and incorporation of objective biomarkers are needed to strengthen evidence and guide integrated migraine care strategies for pediatric populations.

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CONFLICT OF INTEREST STATEMENT

The authors declared that they have no conflicts of interest to disclose.

AUTHORS' CONTRIBUTIONS

JPG, VGRV, MNYSV, PEYL, LCT, MSL: Conceptualisation, Literature search, Data collection, Evidence synthesis, Data analysis, Writing—original draft, Draft corrections. **JPG, VGRV:** Revision prior publication – review & editing. **JPMDS, MAJG, RJAB:** Technical review and editing, Draft corrections.

ETHICS STATEMENT

The research proposal was submitted for ethical review and is exempted from review (FEU-NRMF IERC 2024-0120).

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