frontiers | Frontiers in Nutrition

TYPE Editorial
PUBLISHED 12 September 2024
DOI 10.3389/fnut.2024.1456706

OPEN ACCESS

EDITED AND REVIEWED BY
Ellen E. Blaak,
Maastricht University, Netherlands

*CORRESPONDENCE
Kieron Rooney
✉ kieron.rooney@sydney.edu.au

RECEIVED 29 June 2024
ACCEPTED 26 August 2024
PUBLISHED 12 September 2024

CITATION
Field R, Pourkazemi F, Hashempur MH, Thiruvengadam M and Rooney K (2024) Editorial: Diet, nutrition, and functional foods for chronic pain. *Front. Nutr.* 11:1456706. doi: 10.3389/fnut.2024.1456706

Editorial: Diet, nutrition, and functional foods for chronic pain

Rowena Field[1], Fereshteh Pourkazemi[1], Mohammad Hashem Hashempur[2], Muthu Thiruvengadam[3] and Kieron Rooney[1]*

[1]Faculty of Medicine and Health, The University of Sydney, Sydney, NSW, Australia, [2]Department of Persian Medicine, School of Medicine, Research Center for Traditional Medicine and History of Medicine, Shiraz University of Medical Sciences, Shiraz, Iran, [3]Department of Applied Bioscience, College of Life and Environmental Science, Konkuk University, Seoul, Republic of Korea

KEYWORDS

diet, nutrition, chronic pain, inflammation, ultra-processed food, functional foods, integrative medicine

Editorial on the Research Topic
Diet, nutrition, and functional foods for chronic pain

Persistent or chronic pain is a global issue affecting approximately 20% of the adult population worldwide (1). Chronic pain is defined as pain persisting beyond the normal tissue healing time, typically lasting more than 3 months, and is regarded as a distinct condition rather than just a symptom of an underlying disease (2). The development of chronic pain remains poorly understood, leading to challenges in identifying definitive treatments for this highly prevalent health issue. Current standards of care emphasize a multifaceted, multidisciplinary approach that addresses the complex interplay between biological, psychological, and social factors (3, 4). However, despite efforts, the implementation of biopsychosocial approaches remains challenging (5), and the effectiveness of available treatments to significantly improve outcomes for patients with chronic pain remains limited. It is crucial that treatment strategies prioritize personalized care and management plans aimed at maintaining patient activity levels and effectively managing pain. As part of comprehensive care plans, a modified diet has recently (6, 7) been recognized as a valuable lifestyle strategy for enhancing pain management outcomes (8).

Current evidence supporting dietary intervention as a part of treatment plans for chronic pain is outlined in two recent systematic reviews with meta-analyses of human studies (7, 12) and one systematic review of preclinical studies (13). Rather than identifying a superior diet, the evidence highlights a broad range of dietary "types" (e.g., Mediterranean) with favorable outcomes for pain. These findings support that common factors among these approaches—such as weight loss, diet quality, and nutrient density—may play a role in modulating pain neurophysiology, providing a general guide applicable to any preferred dietary pattern (7). In contrast to evidence on broader dietary patterns, some research focuses on specific food elements, namely functional foods. For example, evidence suggests that incorporating functional foods such as peppermint, turmeric, ginger, and green tea into dietary modifications can enhance their effectiveness in alleviating pain and promoting overall wellbeing. These benefits may be due to the bioactive compounds in these foods, which have anti-inflammatory and analgesic properties (9–11). Additionally, the evidence underscores the need for further research on the impact of diet on pain experience. Building on the current evidence, this Research

Topic presents six papers that provide a variety of evidence demonstrating that diet can influence pain outcomes. Three of these articles were focused on dietary interventional studies for patients with chronic pain (Ciaffi et al.; Sala-Climent et al.; Ward et al.), and two were secondary analyses of genomic data investigating the association of dietary factors with chronic pain (Liu et al.; Dai et al.). One was a cross-sectional study investigating the association of a plant-based diet with migraine headaches (Karimi et al.).

A typical Western diet is characterized by the consumption of ultra-processed foods (UPF). The creation of UPF involves the degradation of the matrix of whole-food ingredients, combined with processed additives designed to restore sensory aesthetics to create hyper-palatable and nutrient-poor foods (14). Deficiency of essential vitamins and minerals may play a role in the development of chronic pain. For example, low Vitamin D levels have been associated with chronic widespread pain presentations (15) and higher levels of the inflammatory biomarker C-reactive protein (CRP) (16). Other nutrients of concern include magnesium, zinc, and vitamin B12 (17, 18). A Western diet high in UPFs is nutrient deficient and high in inflammatory mediators and has been linked to an increased risk of chronic pain (19). Improvements in diet quality and nutrient density are effective pain treatment options (6, 20). This includes both the addition of nutrients needed for optimal nervous system function and the removal of anti-nutrients, such as artificial colors, preservatives, and flavors. A case was also made for the inclusion of phytochemicals (naturally occurring molecules in plants such as curcumin and resveratrol) that have anti-inflammatory and antioxidant actions and may be of therapeutic potential in chronic pain conditions (13, 21). Sala-Climent et al. used a Mediterranean diet to add nutrient density and remove potentially inflammatory mediators, such as gluten and vegetable oils, by removing UPF and focusing on whole foods and extra virgin olive oil, demonstrating improved pain, sleep, and metabolic markers. Similarly, when investigating the effect of a plant-based diet on migraine, Karimi et al. found improvement with a whole-food approach but not with ultra-processed foods.

Most UPFs are refined carbohydrates with a high glycemic index, resulting in large blood glucose excursions and a concomitant large insulin response (22). Their removal from the diet can result in a reduction in total carbohydrate intake, blood glucose excursions, and the subsequent requirement for insulin. A recent randomized clinical trial on chronic pain that compared reducing UPF to the same diet plus reducing carbohydrate (ketogenic diet) found a significant reduction in pain in both groups. The reduction in UPF alone resulted in a reduction in total carbohydrate (209 g/day to 152 g/day) even though this was not an intended consequence (6). Chronically elevated blood glucose levels and the resultant hyperinsulinemia have been linked to pain conditions such as fibromyalgia (23, 24) and glial-mediated sensitization of nociceptors in diabetic neuropathy (25). They also generate advanced glycation end products (AGES) which sensitize nociceptors (26), stiffen tendons (27), and activate enzymes within the cartilage matrix, resulting in damage and inflammation (28). Ciaffi et al. postulated that a very low-calorie ketogenic diet (VLCKD), which has demonstrated positive outcomes in other neurological disorders that share similarities with FM, could be a plausible treatment strategy because of both weight loss and the effects of ketones on the nervous system. Their pilot study of 18 female participants demonstrated a beneficial impact of various FM measures with a personalized VLCKD. They noted that, despite weight loss, BMI did not necessarily track patient-reported outcomes over time, leading them to surmise that benefits may also come from the pleiotropic effects of a ketogenic diet, such as neuroprotection and lower neuroinflammation. The effect of weight loss is, however, important to pain outcomes. Ward et al. demonstrated improved pain and functional mobility outcomes in a secondary analysis of 110 participants on a three-month calorie-restricted diet who achieved a 7.0 kg weight loss.

Polyunsaturated fatty acids (PUFAs) are essential fatty acids that must be obtained through the diet. They are involved in immune system function, with omega-6 PUFAs being the precursor to eicosanoids, which are generally pro-inflammatory, and omega-3 PUFAs being involved in the resolution of inflammation (29, 30). Omega-6 intake per day has risen from 2.7g/day in the 1960s to up to 21g/day (31) largely due to UPF made with vegetable oils (29), resulting in the increase of the omega 6:3 ratio from around 4:1 to over 20:1 (30). Diets high in omega-6 have also been shown to contribute to chronic pain (32–34) and may be responsible for the upregulation of nociception (33). An O6:O3 ratio >5 is associated with higher pain outcomes and functional limitations, as well as higher inflammatory biomarkers of systemic inflammation (16). In contrast, diets that improve this ratio by increasing omega-3 or decreasing omega-6 intake may improve pain outcomes (33). Dai et al. analyzed PUFA concentrations and reported pain from a GWAS database to assess causal relationships. Their results also demonstrated that higher omega-3 concentrations were associated with lower abdominal, pelvic, and lower back pain. Additionally, a higher omega 6:3 ratio was positively associated with abdominal and pelvic pain and supports findings from other authors of increased orofacial and lower back pain and headache (33).

We the editors hope that, collectively, this Research Topic sustains interest in investigating the potential role diet and functional foods may play in the treatment and management of chronic pain and stimulates new areas of inquiry for the betterment of patient outcomes.

Author contributions

RF: Writing – original draft, Writing – review & editing. FP: Writing – original draft, Writing – review & editing. MH: Writing – review & editing. MT: Writing – review & editing. KR: Writing – review & editing.

Funding

The author(s) declare that no financial support was received for the research, authorship, and/or publication of this article.

Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Publisher's note

References

1. Mills SE, Nicolson KP, Smith BH. Chronic pain: a review of its epidemiology and associated factors in population-based studies. *Br J Anaesth.* (2019) 123:e273–e83. doi: 10.1016/j.bja.2019.03.023

2. Treede R-D, Rief W, Barke A, Aziz Q, Bennett MI, Benoliel R, et al. A classification of chronic pain for ICD-11. *Pain.* (2015) 156:1003–7. doi: 10.1097/j.pain.0000000000000160

3. Janke EA, Cheatle M, Keefe FJ, Dhingra L. Committee SoBMHP. Society of Behavioral Medicine (SBM) position statement: improving access to psychosocial care for individuals with persistent pain: supporting the National Pain Strategy's call for interdisciplinary pain care. *Transl Behav Med.* (2018) 8:305–8. doi: 10.1093/tbm/ibx043

4. Hruschak V, Cochran G. Psychosocial predictors in the transition from acute to chronic pain: a systematic review. *Psychol Health Med.* (2018) 23:1151–67. doi: 10.1080/13548506.2018.1446097

5. Sharma S, Breckons M, Brönnimann Lambelet B, Chung JW, List T, Lobbezoo F, et al. Challenges in the clinical implementation of a biopsychosocial model for assessment and management of orofacial pain. *J Oral Rehabil.* (2020) 47:87–100. doi: 10.1111/joor.12871

6. Field R, Pourkazemi F, Rooney K. Effects of a low-carbohydrate ketogenic diet on reported pain, blood biomarkers and quality of life in patients with chronic pain: a pilot randomised clinical trial. *Pain Med.* (2022) 23:326–38. doi: 10.1093/pm/pnab278

7. Field R, Pourkazemi F, Turton J, Rooney K. Dietary interventions are beneficial for patients with chronic pain: a systematic review with meta-analysis. *Pain Med.* (2020) 22:694–714. doi: 10.1093/pm/pnaa378

8. Elma O, Brain K, Dong H-J. The importance of nutrition as a lifestyle factor in chronic pain management: a narrative review. *J Clin Med.* (2022) 11:1–24. doi: 10.3390/jcm11195950

9. Kazemi A, Iraji A, Esmaealzadeh N, Salehi M, Hashempur MH. Peppermint and menthol: a review on their biochemistry, pharmacological activities, clinical applications, and safety considerations. *Crit Rev Food Sci Nutr.* (2023) 3:1–26. doi: 10.1080/10408398.2023.2296991

10. Darani NS, Vaghasloo MA, Kazemi A, Amri H, Rampp T, Hashempur MH. Oxymel: a review of preclinical and clinical studies. *Heliyon.* (2023). doi: 10.1016/j.heliyon.2023.e22649

11. Heydari M, Shams M, Hashempur MH, Zargaran A, Dalfardi B, Borhani-Haghighi A. The origin of the concept of neuropathic pain in early medieval Persia (9th-12th century CE). *Acta medico-historica Adriatica.* (2015) 13:9–22.

12. Brain K, Burrows T, Rollo M, Chai L, Clarke E, Hayes C, et al. A systematic review and meta-analysis of nutrition interventions for chronic noncancer pain. *J Hum Nutr Diet.* (2018) 32:198–225. doi: 10.1111/jhn.12601

13. Elma O, Lebuf E, Marnef AQ, Tumkaya Yilmaz S, Coppieters I, Clarys P, et al. Diet can exert both analgesic and pronociceptive effects in acute and chronic pain models: a systematic review of preclinical studies. *Nutr Neurosci.* (2022) 25:2195–217. doi: 10.1080/1028415X.2021.1934956

14. Fardet A, Rock E. Exclusive reductionism, chronic diseases and nutritional confusion: the degree of processing as a lever for improving public health. *Crit Rev Food Sci Nutr.* (2022) 62:2784–99. doi: 10.1080/10408398.2020.1858751

15. Yong WC, Sanguankeo A, Upala S. Effect of vitamin D supplementation in chronic widespread pain: a systematic review and meta-analysis. *Clin Rheumatol.* (2017) 36:2825–33. doi: 10.1007/s10067-017-3754-y

16. Wijayabahu AT, Mickle AM, Mai V, Garvan C, Glover TL, Cook RL, et al. Associations between vitamin D, omega 6:omega 3 ratio, and biomarkers of aging in individuals living with and without chronic pain. *Nutrients.* (2022) 14:1–9. doi: 10.3390/nu14020266

17. Glenn A, Kavanagh M, Bockus-Thorne L, McNeill L, Melina V, Jenkins D, et al. Medical nutrition therapy for chronic pain management. In: *Clinical Pain Management: A Practical Guide*. John Wiley & Sons (2022) p. 147–59. Available at: https://books.scholarsportal.info/en/read?id=/ebooks/ebooks7/wiley7/2022-03-21/1/9781119701170

18. Philpot U, Johnson MI. Diet therapy in the management of chronic pain: better diet less pain? *Pain Manag.* (2019) 9:335–8. doi: 10.2217/pmt-2019-0014

19. Strath LJ, Brooks MS, Sorge RE, Judd SE. Relationship between diet and relative risk of pain in a cross-sectional analysis of the REGARDS longitudinal study. *Pain Manag.* (2022) 12:168–79. doi: 10.2217/pmt-2021-0048

20. Cooper I, Brukner P, Devlin BL, Reddy AJ, Fulton M, Kemp JL, et al. An anti-inflammatory diet intervention for knee osteoarthritis: a feasibility study. *BMC Musculoskelet Disord.* (2022) 23:47. doi: 10.1186/s12891-022-05003-7

21. Kang L, Zhang H, Jia C, Zhang R, Shen C. Targeting oxidative stress and inflammation in intervertebral disc degeneration: therapeutic perspectives of phytochemicals. *Front Pharmacol.* (2022) 13:1–14. doi: 10.3389/fphar.2022.956355

22. Christ A, Lauterbach M, Latz E. Western diet and the immune system: an inflammatory connection. *Immunity.* (2019) 51:794–811. doi: 10.1016/j.immuni.2019.09.020

23. Pappolla MA, Manchikanti L, Candido KD, Grieg N, Seffinger M, Ahmed F, et al. Insulin resistance is associated with central pain in patients with fibromyalgia. *Pain Physician.* (2021) 24:175–84. doi: 10.36076/ppj.2021.24.175-184

24. Lattanzio SM. Fibromyalgia syndrome: a metabolic approach grounded in biochemistry for the remission of symptoms. *Front Med.* (2017) 4:198. doi: 10.3389/fmed.2017.00198

25. Rajchgot T, Thomas SC, Wang J-C, Ahmadi M, Balood M, Crosson T, et al. Neurons and microglia; a sickly-sweet duo in diabetic pain neuropathy. *Front Neurosci.* (2019) 13:25. doi: 10.3389/fnins.2019.00025

26. Bestall SM, Hulse RP, Blackley Z, Swift M, Ved N, Paton K, et al. Sensory neuronal sensitisation occurs through HMGB-1/RAGE and TRPV1 in high glucose conditions. *J Cell Sci.* (2018) 131:jcs215939. doi: 10.1242/jcs.215939

27. Snedeker JG. How high glucose levels affect tendon homeostasis. In: Ackermann PW, Hart DA, editors. *Metabolic Influences on Risk for Tendon Disorders*. Cham: Springer International Publishing (2016). p. 191–8.

28. Bradley D. The intriguing intersection of type 2 diabetes, obesity-related insulin resistance, and osteoarthritis. *J Clini Endocrinol Metabol.* (2021) 106:e2370. doi: 10.1210/clinem/dgab009

29. Saini RK, Keum Y-S. Omega-3 and omega-6 polyunsaturated fatty acids: Dietary sources, metabolism, and significance — A review. *Life Sci.* (2018) 203:255–67. doi: 10.1016/j.lfs.2018.04.049

30. Mariamenatu AH, Abdu EM. Overconsumption of omega-6 polyunsaturated fatty acids (PUFAs) versus deficiency of omega-3 PUFAs in modern-day diets: the disturbing factor for their "balanced antagonistic metabolic functions" in the human body. *J Lipids.* (2021) 2021:1–15. doi: 10.1155/2021/8848161

31. Hamilton JS, Klett EL. Linoleic acid and the regulation of glucose homeostasis: a review of the evidence. *Prostaglandins Leukot Essent Fatty Acids.* (2021) 175:102366. doi: 10.1016/j.plefa.2021.102366

32. McGinnis A, Ji R-R. Can a Western high-fat diet lead to painful neuropathy? *Nat Metab.* (2021) 3:735–6. doi: 10.1038/s42255-021-00411-w

33. Sanders AE, Weatherspoon ED, Ehrmann BM, Soma PS, Shaikh SR, Preisser JS, et al. Circulating polyunsaturated fatty acids, pressure pain thresholds, and nociplastic pain conditions. *Prostaglandins Leukot Essent Fatty Acids.* (2022) 184:1–10. doi: 10.1016/j.plefa.2022.102476

34. Boyd JT, LoCoco PM, Furr AR, Bendele MR, Tram M, Li Q, et al. Elevated dietary ω-6 polyunsaturated fatty acids induce reversible peripheral nerve dysfunction that exacerbates comorbid pain conditions. *Nat Metab.* (2021) 3:762–73. doi: 10.1038/s42255-021-00410-x