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Photobiomodulation Before and After Chemotherapy for Oral Mucositis: A Mini Review

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ABSTRACT

Background: Oral mucositis (OM) is a painful, dose-limiting toxicity of cytotoxic chemotherapy. It impairs nutrition and quality of life, raises infection risk and can force dose reductions or treatment delays. Photobiomodulation (PBM), the application of low-power red or near-infrared light to tissue, has emerged as the most extensively studied non-pharmacological intervention for OM.

Objective: To summarize current evidence on PBM delivered before and during chemotherapy (preventive use) and after OM onset (therapeutic use), with emphasis on clinical protocols relevant to dental practitioners.

Findings: The 2019/2020 Multinational Association of Supportive Care in Cancer/International Society of Oral Oncology (MASCC/ISOO) guidelines recommend intraoral PBM for OM prevention in adults receiving hematopoietic stem cell transplantation (HSCT) conditioned with high-dose chemotherapy (level of evidence I), but found the evidence insufficient for a guideline in conventional-dose chemotherapy or for treating established OM. Subsequent systematic reviews and randomized trials, including pediatric trials with methotrexate-based regimens, report lower OM incidence and severity with preventive PBM, and faster healing with less pain when PBM is applied after onset. Adverse events are rare and mild. Heterogeneous dosimetry, small samples and incomplete blinding remain the main limitations.

Conclusion: PBM is a safe, well-tolerated adjunct to basic oral care. It is best started at or before the first day of chemotherapy and continued, or resumed, once lesions appear. Standardized, adequately powered trials in conventional-dose chemotherapy are needed to extend formal guideline support.

Keywords: Oral mucositis; Photobiomodulation; Low-level laser therapy; Chemotherapy; Supportive care; Stomatitis.

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1. Introduction

Oral mucositis (OM) is the inflammation and ulceration of the oral mucosa caused by cytotoxic cancer therapy. It is among the most frequent and distressing acute toxicities of chemotherapy [1–3]. Reported incidence depends on the regimen, the patient population and the scoring system used. In adults receiving conventional-dose chemotherapy for lymphoma or solid tumors, 20% to 40% develop OM of any grade, although severe (World Health Organization [WHO] grade 3–4) disease is much less common and incidence may rise in later cycles [3]. Among patients undergoing myeloablative conditioning before hematopoietic

stem cell transplantation (HSCT), 75% to 100% develop OM of any grade [3]. In children, estimates range from about 52% to 80%, and OM is more frequent in hematologic malignancies than in solid tumors [4]. Commonly implicated agents include 5-fluorouracil, methotrexate, melphalan, busulfan, doxorubicin, etoposide and taxanes [1,3].

Clinically, OM begins with erythema and burning, then progresses to ulcers covered by pseudomembranes. With cyclical chemotherapy and HSCT conditioning, the first signs usually appear about 3–4 days after drug infusion, ulcers form shortly afterward, severity peaks roughly 7–14 days later, and lesions resolve over a further 5–10 days [3]. Severe OM causes pain that often requires opioids, impairs eating and speech, and may require parenteral nutrition [3,5]. In neutropenic patients, the broken epithelial barrier is a portal for bacteremia and sepsis [3,5]. In HSCT recipients, severe OM has been associated with more infections, greater opioid and parenteral nutrition use, higher costs and higher 100-day mortality [6]. OM can also force dose reductions or treatment delays, with potential effects on cancer control and survival [1,3].

Options for prevention and treatment remain limited. Basic oral care is universally advised, and palifermin, cryotherapy and benzydamine have guideline support only in specific settings [1]. Photobiomodulation (PBM), formerly called low-level laser therapy (LLLT), uses red or near-infrared light to induce non-thermal biological effects in tissue. It is now one of the best-studied interventions for OM [7,8]. Dental professionals are often the clinicians who deliver it.

This mini review summarizes the evidence for PBM in chemotherapy-induced OM, distinguishing preventive use (started before or at the start of chemotherapy) from therapeutic use (after OM has developed). It also outlines practical protocols, safety considerations and remaining gaps.

2. Pathobiology of Oral Mucositis and Mechanisms of PBM

OM is not simply direct epithelial death. Sonis described it as a five-phase process involving the epithelium, submucosa and immune cells [2,3]. Chemotherapy first generates reactive oxygen species (ROS) and DNA damage (initiation). This activates transcription factors, chiefly nuclear factor-κB (NF-κB), which upregulate pro-inflammatory cytokines such as tumor necrosis factor-α, interleukin-1β and interleukin-6 (primary damage response). These cytokines amplify tissue injury through positive feedback (signal amplification). The epithelium then breaks down and ulcers are colonized by oral bacteria (ulceration), before the mucosa regenerates once cytotoxic exposure ends (healing) [2].

PBM acts on several of these steps. Red (approximately 630–690 nm) and near-infrared (approximately 780–980 nm) photons are absorbed mainly by cytochrome c oxidase in the mitochondrial respiratory chain [7]. Absorption displaces inhibitory nitric oxide, increases ATP synthesis and produces a brief, low-level ROS signal. This in turn modulates transcription factors and downstream gene expression [7]. The net clinical effects are reduced inflammation, enhanced epithelial and fibroblast proliferation, and analgesia [7,8]. The response is biphasic: too little energy produces no effect, while excessive energy can inhibit cell proliferation. This is why reported dosimetry matters [9,10].

Table 1 summarizes the five phases and the proposed PBM effect at each. The phase descriptions follow Sonis's model [2], as restated in a later epidemiology and pathobiology review [3]. The proposed PBM effects come from mechanistic reviews of PBM [7,10]; the effects on pain and healing are additionally supported by the clinical evidence synthesized in the Multinational Association of Supportive Care in Cancer/International Society of Oral Oncology (MASCC/ISOO) systematic review [8]. Much of the mechanistic evidence is preclinical, so these effects remain proposed rather than proven for human oral mucosa. This mechanistic profile explains why timing matters. PBM delivered during phases 1–3, before ulcers form, aims to blunt the inflammatory cascade (prevention). PBM delivered in phases 4–5 aims to relieve pain and speed repair (treatment).

Table 1. Phases of chemotherapy-induced oral mucositis and corresponding photobiomodulation effects. Mucositis phases and key events follow Sonis [2,3]; proposed PBM effects are drawn from mechanistic reviews [7,10], with pain and healing effects also supported by the clinical evidence in [8].

Mucositis phase [2,3]	Key events [2,3]	Proposed PBM effect [7,8,10]
1. Initiation	ROS generation, DNA strand breaks in basal epithelium	Enhanced mitochondrial function and antioxidant response [7]
2. Primary damage response	NF-κB activation, cytokine gene upregulation	Down-regulation of NF-κB and pro-inflammatory cytokines [7,10]
3. Signal amplification	TNF-α, IL-1β, IL-6 feedback loops, submucosal injury	Reduced cytokine release and edema [7,10]
4. Ulceration	Epithelial loss, pseudomembrane, bacterial colonization, pain	Analgesia; faster re-epithelialization [8,10]
5. Healing	Epithelial proliferation and migration	Increased ATP, fibroblast and keratinocyte proliferation, growth factor release [7,10]

3. Preventive PBM: Before and During Chemotherapy

3.1 Guideline position

Preventive PBM has the strongest evidence base. In the 2019 systematic review by the Mucositis Study Group of MASCC/ISOO, intraoral PBM received a *recommendation* (level of evidence I) for preventing OM in adults undergoing HSCT conditioned with high-dose chemotherapy, with or without total body irradiation [8]. Recommendations were also issued for head and neck radiotherapy with or without concomitant chemotherapy [8]. These were carried into the 2020 MASCC/ISOO guideline update [1]. For conventional-dose chemotherapy outside HSCT, however, the panel concluded that no guideline was possible. Trial populations were mixed and treatment parameters were too variable to pool [1,8].

The HSCT recommendation rests on protocols applied from the first day of conditioning, or immediately after it, through the early post-transplant period [8]. Randomized trials in this setting, such as that of Antunes and colleagues using a 660 nm diode laser, showed less severe OM in irradiated patients than in controls [11]. HSCT is also the setting with the highest baseline risk: 75% to 100% of patients receiving myeloablative conditioning develop OM of any grade, compared with roughly 20% to 40% of adults receiving conventional-dose chemotherapy for lymphoma or solid tumors [3].

3.2 Evidence since the guidelines

Newer data now extend to conventional chemotherapy. A 2025 systematic review restricted to randomized trials of preventive PBM in chemotherapy patients without prior radiotherapy identified 13 studies with 828 participants [12]. Across these trials, 41% of patients developed OM over the chemotherapy cycles studied; this pooled figure reflects the mixed trial populations and regimens and should not be read as a population incidence (see Introduction for incidence by setting). Among those who did, 211 were in control arms and 128 in PBM arms. The authors concluded that prophylactic PBM reduces both the occurrence and the severity of lesions [12].

Pediatric data are particularly relevant because children receiving high-dose methotrexate are at high risk. In a 2025 trial in children with leukemia, each patient served as their own control across two comparable methotrexate cycles [9]. PBM with an 808 nm diode laser on days 1 and 3 of the cycle (a study-specific protocol; see Table 2) lowered OM incidence from 66.7% (10/15) to 6.7% (1/15), with no adverse effects [9]. These findings come from a single small trial and should not be generalized. A Brazilian referral service reported similar benefit over five years of routine prophylactic 660 nm PBM in pediatric chemotherapy patients [13]. A 2023 review of 15 pediatric studies noted inconsistent reporting of laser parameters and the absence of an established pediatric protocol [4].

Results are not uniformly positive. An earlier pediatric randomized trial found no significant difference in OM prevalence or grade when optimal dental and oral care was provided to both groups [14]. This

underlines that PBM is an adjunct to, not a substitute for, meticulous oral hygiene and dental assessment before chemotherapy.

3.3 Practical timing

Across studies, preventive PBM is most often started on the first day of chemotherapy or conditioning, or in the days before it. It is then repeated daily or on alternate days through the period of peak mucosal injury, usually until about day +2 to +8 of the cycle or until neutrophil recovery after HSCT [8,9,12]. A pre-chemotherapy dental visit is the natural point to examine the mouth, remove infection sources, give hygiene instructions and schedule the first PBM session.

4. Therapeutic PBM: After Chemotherapy-Induced OM Develops

Therapeutic PBM shortens healing and reduces pain, but its evidence is weaker than for prevention. MASCC/ISOO found it insufficient to issue a guideline for established OM in 2019 [8]. Since then, several trials and pooled analyses have strengthened the case.

The multicenter, double-blind LaMPO trial randomized children with severe chemotherapy-induced OM to PBM or sham laser on four consecutive days [15]. Blinded assessment on days +4, +7 and +11 showed faster mucosal recovery and lower pain scores with PBM, without added side effects [15]. A meta-analysis with trial sequential analysis of randomized trials in chemotherapy-induced OM found that PBM reduced OM severity compared with sham (risk ratio 0.43, 95% CI 0.20–0.93) [16]. The trial sequential analysis judged this evidence conclusive, and GRADE certainty was moderate [16].

Other syntheses urge caution. A 2024 umbrella review identified 40 meta-analytic estimates on PBM for chemotherapy- and radiotherapy-induced OM [17]. Most (87.5%) were statistically significant, but only one reached “highly suggestive” credibility. The authors rated the evidence for treating chemotherapy-induced OM as coming from low- to critically low-quality reviews [17].

Two refinements are emerging. First, *preemptive* PBM, delivered at the earliest signs of OM and continued through therapy, may give immediate pain relief rather than the delayed relief seen in most trials [18]. Second, antimicrobial photodynamic therapy (PDT), in which a photosensitizer such as methylene blue or indocyanine green is activated by laser light, may be combined with PBM for ulcerated, colonized lesions. A meta-analysis found that adding PDT to low-power laser therapy shortened healing time compared with laser therapy alone [19]. In the pediatric methotrexate trial, indocyanine green-mediated PDT also lowered WHO, National Cancer Institute (NCI) and Woolworths Centre for Childhood Nutrition Research (WCCNR) scores by days 3 and 5 compared with sham [9].

In practice, therapeutic PBM is usually given daily or every other day from diagnosis until lesions resolve [9,15]. For patients who already received preventive PBM, the same device and points can simply be continued, with attention to areas of active ulceration.

5. Clinical Protocols and Dosimetry

Reproducible PBM depends on reporting and controlling a full set of parameters, not just wavelength. Expert consensus for OM suggests a red (633–685 nm) or near-infrared (780–830 nm) source, output of 10–150 mW, and 2–3 J/cm^2 per point, not exceeding 6 J/cm^2 [20]. Earlier recommendations distinguished prophylactic doses (at least 2 J/cm^2 for red and 3 J/cm^2 for infrared light) from therapeutic doses (at least 4 J/cm^2) [21]. Irradiation time per point follows from the target dose:

$$\text{time (s)} = \text{fluence (J/cm}^2\text{)} \times \text{spot area (cm}^2\text{)} / \text{power (W)}$$

For example, a 100 mW device delivering 2 J/cm^2 to a 1 cm^2 spot needs 20 s; 4 J/cm^2 needs 40 s [21]. Light is applied in stationary mode, point by point, in contact or near-contact, over the labial and buccal mucosa, lateral and ventral tongue, floor of mouth and soft palate. Red light is used intraorally; near-infrared light can also reach these sites transcutaneously through the cheeks and lips [20,21].

Table 2 shows how widely reported protocols differ in fluence, number of points and schedule. Rows 1–2 are the guideline-supported protocols for adult HSCT [8], row 3 is the protocol of a single pediatric trial, and row 4 is an expert consensus range. The 20 J/cm^2 used in the pediatric trial is a study-specific parameter set. It lies well above the 2–3 J/cm^2 consensus range (maximum 6 J/cm^2) [20] and must not be read as a general clinical recommendation or target dose. Clinicians should follow one published protocol exactly rather than mixing parameters, and should record wavelength, power, spot size, time per point, number of points and session dates in the patient record [8].

Table 2. Examples of preventive PBM protocols for chemotherapy-related oral mucositis. Rows 1–2 are MASCC/ISOO guideline-supported protocols for adult HSCT [8]; row 3 is a study-specific protocol from a single pediatric trial and is not a clinical recommendation [9]; row 4 is an expert consensus range [20].

Setting	Source	Wavelength	Dose per point	Sites	Schedule	Reference
Adult HSCT (MASCC/ISOO protocol A)	He-Ne laser, 31.25 mW/cm², 40 s/spot, 0.8 cm² spot	632.8 nm	1.25 J/cm² (1.0 J per spot)*	18	Daily for 5 days from the day after conditioning ends	[8]
Adult HSCT (MASCC/ISOO protocol B)	Diode laser, 1000 mW/cm², 2 s/spot, 0.04 cm² spot	650 nm	2.0 J/cm² (0.08 J per spot)	54–70	Daily from day 1 of conditioning to day +2 (7–13 days)	[8]
Pediatric leukemia, high-dose methotrexate (single-trial protocol; not a recommendation) †	Diode laser, 250 mW, 40 s/point, 0.5 cm² spot, 220 J/session	808 nm	20 J/cm² (10 J per point)†	22	Days 1 and 3 of each cycle	[9]
Consensus range for OM	Laser or LED, 10–150 mW	633–685 or 780–830 nm	2–3 J/cm² (max 6 J/cm²)	Whole mucosa at risk	2–3 times weekly to daily	[20]

**Value verified against the published MASCC/ISOO protocol table, which lists 31.25 mW/cm² for 40 s, 0.8 cm² spot, and gives the dose as 1.0 J/cm² [8,10]. Irradiance multiplied by time gives 1.25 J/cm² (31.25 mW/cm² × 40 s). Over the 0.8 cm² spot this equals 1.0 J of total energy per spot (25 mW × 40 s). The published value of 1.0 therefore appears to represent energy per spot (J) rather than energy density (J/cm²); both are given here.*

†Study-specific protocol from a single small pediatric trial [9]: 250 mW for 40 s delivers 10 J per point over a 0.5 cm² spot (20 J/cm²), or 220 J per session across 22 points. This fluence is not guideline-supported and exceeds the 6 J/cm² upper value of the consensus range [20]. It is shown only to illustrate protocol variability, and pediatric PBM protocols remain unstandardized [4].

6. Safety, Limitations and Future Directions

6.1 Safety

PBM is well tolerated. No short- or long-term adverse events were reported in any randomized trial reviewed by MASCC/ISOO, despite wide variation in parameters [8]. The only notable effect was a transient, non-painful burning sensation in 15% of patients in one cohort treated with a 635 nm diode laser [8]. Recent pediatric trials likewise reported no protocol-related adverse effects [9,15].

A theoretical concern is that light that stimulates normal cells could also stimulate residual tumor cells. A systematic review by the World Association for Laser Therapy found conflicting in vitro results. However, in vivo studies and clinical trials with follow-up showed no adverse effect on tumor growth, recurrence or survival with currently recommended parameters [22]. Prudent practice still avoids directly irradiating known tumor sites in the oral cavity, uses protective eyewear for patient and operator, and reviews photosensitizing medications.

6.2 Limitations of the evidence

The main weakness is heterogeneity. Trials vary in wavelength (from 630 to 980 nm), fluence (from about 1 to 20 J/cm^2 or more, the upper end representing study-specific protocols such as the pediatric trial in Table 2), number of points, timing and OM scales (WHO, National Cancer Institute–Common Terminology Criteria for Adverse Events (NCI-CTCAE), WCCNR, Oral Mucositis Assessment Scale (OMAS)) [8,9,12]. Many

trials are small, single-center and not fully blinded, and some combine chemotherapy-only and chemoradiotherapy populations [1,17]. These issues explain why guideline bodies have not yet extended a formal recommendation to conventional-dose chemotherapy despite largely positive individual results.

6.3 Future directions

Priorities include:

- Adequately powered, sham-controlled, blinded trials in conventional-dose chemotherapy, stratified by drug regimen and age.
- Head-to-head comparison of intraoral and extraoral delivery; such trials are underway in pediatric oncology [23].
- Home-based or self-applied LED devices that could make preventive PBM more accessible [24].
- Defined protocols for preemptive pain relief and for PBM combined with photodynamic therapy [18,19].
- Cost-effectiveness analyses and consistent reporting of full dosimetry in every study.

7. Conclusion

PBM is a safe, non-invasive adjunct for chemotherapy-induced OM with benefit at both ends of the treatment course. Started before or on the first day of chemotherapy, it reduces the incidence and severity of OM; this use carries a formal MASCC/ISOO recommendation in adults undergoing HSCT. Applied after lesions appear, it accelerates healing and relieves pain, although the supporting evidence is of lower certainty. Dental professionals are well placed to integrate PBM into pre-chemotherapy oral care, provided they follow a published protocol precisely and document full dosimetry. Standardized, adequately powered trials in conventional-dose chemotherapy are the key step toward broader guideline endorsement.

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10. Conflict of Interest

The authors declare no conflict of interest.

11. Author Contributions

Natalia Elson: conceptualization, literature search, writing of the original draft. Elena Damiano: critical revision. Irene Brandes: supervision, review and editing. All authors approved the final manuscript and are accountable for its content.

12. Use of Artificial Intelligence

An AI writing assistant (Claude) was used to help structure and draft the manuscript. The authors verified all content and references against the original sources and take full responsibility for the final text.

13. Data Availability

Not applicable; no new data were generated.

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