

Therapeutic Photobiomodulation: A Necessary Component of a Veterinary Pain Management Strategy

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The primary role of the veterinarian is to control pain and suffering, and the range of tools and methods to accomplish this is ever-increasing. Therapeutic photobiomodulation, such as provided by therapy lasers, has become an important element in a multimodal approach to pain management.

The analgesic effects achieved with the administration of therapeutic photobiomodulation are well documented in the literature. The first of the peer-reviewed papers appeared in 1991 with the bulk of the documentation detailing the mechanisms and the effectiveness of this modality being presented from 2009 to 2012.¹⁻⁵

Practicing scientific, evidence-based medicine does not allow us to merely believe: "Photons enter, a miracle happens, and then the pain is gone!" The mechanisms resulting in this physiological achievement are clearly understood.

Photons, within the infrared spectrum, act on the endogenous photoreceptors, or chromophores, of the individual cells resulting in a biochemical cascade of events. A combination of localized and systemic enzymatic, chemical and physical events effectively produce a state of analgesia.⁶⁻⁹

Increased Endorphin Release

Once the target cells receive a therapeutic dosage of photonic energy, there is a release of endorphins.¹⁰ "Endorphin" comes from the words endogenous + morphine. These endogenous peptides attach to the same cell receptors in the brain, spinal cord and other nerve endings that would accept morphine and act presynaptically to inhibit the release of the inhibitory neurotransmitter GABA (gamma-aminobutyric acid).¹¹

The almost instantaneous clinical result is a reduction in pain perception coupled with a mild euphoria.

Increased Nitric Oxide

Nitric oxide, produced in the mitochondria, can inhibit respiration by binding to cytochrome c oxidase, thus competitively displacing oxygen. This is especially true in stressed or hypoxic cells.¹¹ Following therapeutic photobiomodulation there is a photodissociation of nitric oxide from cytochrome c oxidase, thereby reversing the mitochondrial inhibition of the respiratory rate due to excessive nitric oxide binding.¹²

This increased level of nitric oxide has multiple pain relieving effects for the patient:

- * Nitric oxide serves as a neurotransmitter between nerve cells, part of its general role in redox signaling. Unlike most other neurotransmitters that only transmit information from a presynaptic to a postsynaptic neuron, the small, uncharged and fat-soluble nitric oxide molecule can diffuse widely and readily enters cells. Consequently, it can act on several nearby neurons, even on those not connected by a synapse. At the same time, the short half-life of nitric oxide means that such action will be restricted to a limited area, without the necessity for enzymatic breakdown or cellular reuptake.¹³

- * Increased levels of nitric oxide result in a decrease of conductivity in sensor nerves as a result of hyperpolarization of nerve endings and changes in potential of neuron membranes.¹⁴

- * There is an activation of metabolic processes in mito-



chondria nerve endings.¹⁵ This results in changes within the conduction of stimuli in cholinergic synapses.^{16,17}

- * A reflexory inhibition of ascending pain conduction tracts and activation of secretion of endorphins within the CNS by laser stimulation of acupuncture points.²⁶

Decreased Bradykinin Levels

Bradykinins, released from plasma protein at the site of injured tissue, elicit pain by stimulating nociceptive afferents in the skin and viscera.

Mitigation of these elevated levels through therapeutic photobiomodulation will therefore result in a reduction of pain. The photobiomodulated induced decrease in plasma kallikrein, increase in kininase II and the increases in nitric oxide are considered the contributors to the decrease in bradykinin levels.¹⁸

Blocked Depolarization of C Fiber Afferent Nerves

Another extremely important analgesic event occurs when therapeutic photobiomodulation results in the blockage of the C fiber afferent nerves.²² These non-myelinated fibers convey input signals from the periphery to the central nervous system and respond to various stimuli that can be thermal, mechanical, or chemical in nature.

Photoreceptors within the neuronal mitochondria absorb photonic energy which is then mediated and transduced into electrochemical changes.^{2,12} This results in a secondary cascade of intracellular events within the neuron that initiates a decrease in the following: mitochondrial membrane potential, available ATP required for nerve function and the maintenance of microtubules and molecular motors, dyneins and kinesins that are responsible for fast axonal flow.¹⁹

Therefore, it is a photonic induced neural blockade that slows the conduction velocity and reduces the amplitudes of the compound action potentials.²⁰ This photobiomodulatory reaction and consequential blockade is the mechanism that reduces nociceptive pain.²⁷

Increased Release of Acetylcholine

There is an increase in the reaction time in the formation of acetylcholine following therapeutic photobiomodulation. The increased availability of this neuromodulator allows for a normalization of nerve signal transmissions within the peripheral and central nervous systems.²³

Axonal Sprouting Several studies have documented the ability of laser therapy to induce axonal sprouting and some nerve regeneration in damaged nerve tis-

sues. Where pain sensation is being magnified due to nerve structure damage, cell regeneration and sprouting assist in alleviating this maladaptive neuropathic pain.^{24,25,28}

Therapeutic Dosage

Therapeutic photobiomodulation is dependent upon the delivery of a therapeutic dosage of energy to the target nerve cells. Wavelength determines the depth of penetration and the power of the laser determines the delivery of the dosage. The current literature states that a physiological and biological response within the cells is achieved at a dosage of 2 to 12 Joules/cm².^{27,28,29,30}

Incidental absorption of photons must be considered when trying to calculate the therapeutic dose. Body mass, color and thickness of the dermis and hair coat must be taken into consideration for successful and consistent results.

Prevention and treatment of superficial acute pain should be dosed at 2 to 6 Joules/cm².

A post-op incision site of 2 inches by 3 inches would require:

$$5.08 \text{ cm} \times 7.62 \text{ cm} = 38.7 \text{ cm}^2$$

$$38.7 \text{ cm}^2 \times 2 \text{ Joules/cm}^2 = 77.4 \text{ Joules/treatment}$$

Chronic pain that is deep within the tissues would require a dosage in the range of 7–12 Joules/cm²

Treatment of a 40-pound dark skinned, long haired dog with chronic hip dysplasia would require:

$$8 \text{ cm} \times 8 \text{ cm} = 64 \text{ cm}^2$$

$$64 \text{ cm}^2 \times 8 \text{ Joules/cm}^2 = 512 \text{ Joules/treatment}$$

Frequency of Treatment

Acute pain:

Aggressive phase: each day for three to four treatments

Transitional phase: (less frequent) every other day or twice per week until condition is resolved.

Chronic pain:

Aggressive phase: each day or every other day for three to four treatments.

Transitional phase: every other day or twice per week until therapeutic goal has been achieved.

Maintenance phase: as often as needed to control pain and maintain a satisfactory quality of life.

Conclusions

Therapeutic photobiomodulation is a scientifically proven modality that is an extremely effective tool for the management of pain. It is one that is easily woven into pain management protocols that already exist in the practice. For many practices, it has become part of the standard of care, and not simply reserved for those cases that fail to respond to traditional methods. ●

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For the full article and, footnotes and case examples using laser therapy as an integral part of a multimodal approach to pain management, see VeterinaryPracticeNews.com/strategy.

This Education Series article was underwritten by Companion Therapy Laser of Newark, Del.