INTRODUCING

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THE FIRST & ONLY GREEN & VIOLET LASER

FDA CLEARED SEPTEMBER 2022:
CHRONIC NECK AND SHOULDER PAIN

Special Thanks to: Dr. Kirk Gair, Dr. Robert Silverman, Dr. Albert Comey

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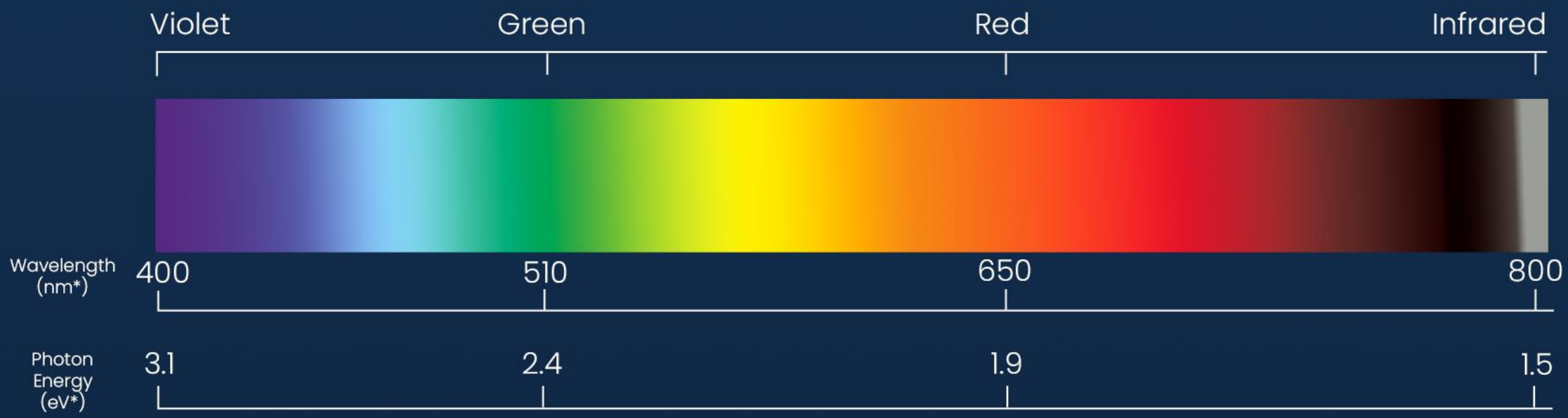

Photochemistry is dependent on the Photon Energy **(Electron Volts).. NOT POWER**

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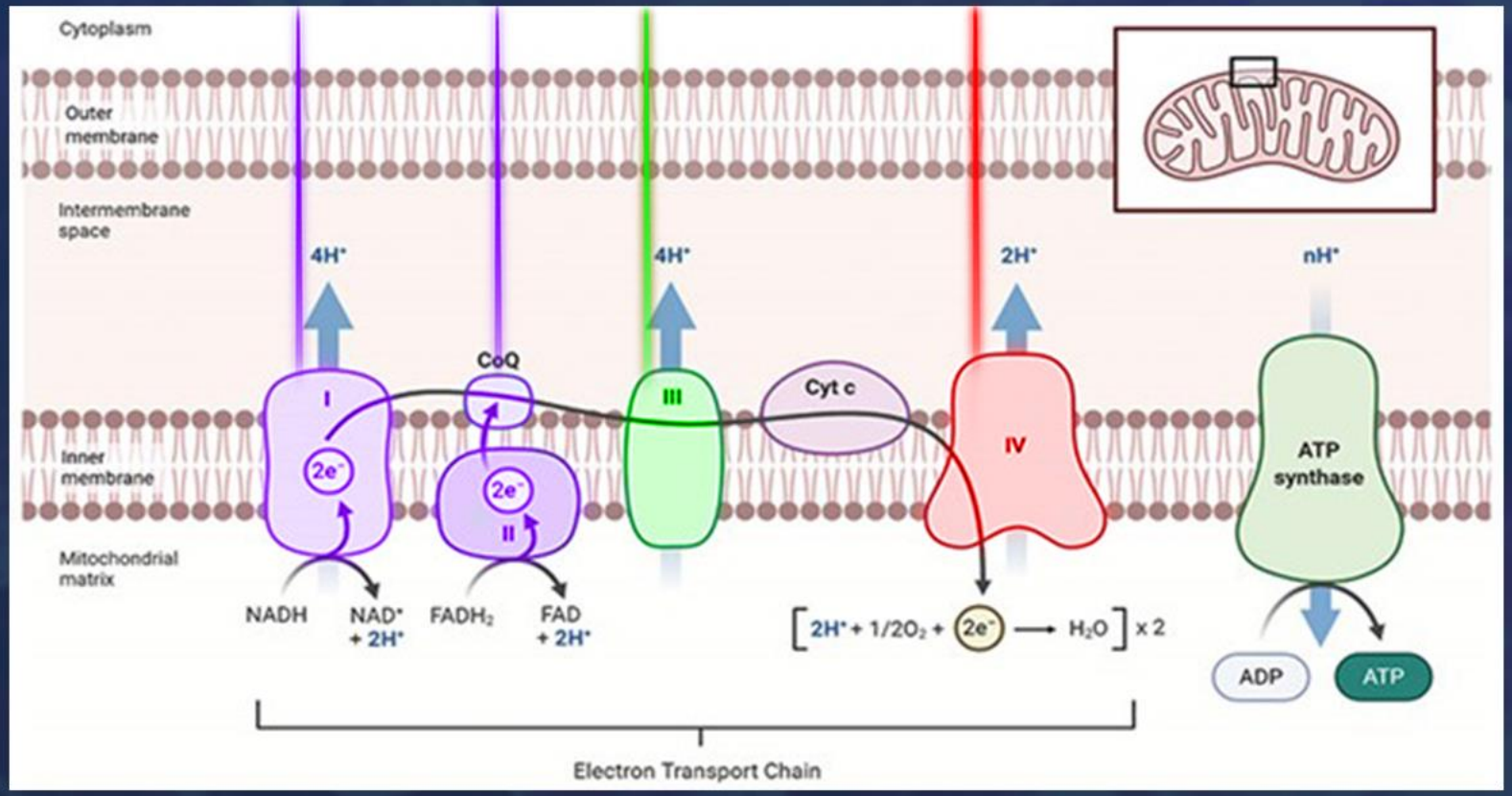
PHOTON ENERGY IS ABSORBED BY THE MITOCHONDRIA

Mitochondria are responsible for 90% of the Adenosine triphosphate (ATP) our body needs to function and has a pivotal role in cell life and cell death.

Mitochondria Electron Transport Chain



Photon Energy (Wavelength) required to excite each complex.

Complex 1&2	Complex 3	Complex 4
405nm	520nm	635nm

OSTEOARTHRITIS

Recent ex vivo studies have reported mitochondrial dysfunction in human OA chondrocytes, and analyses of mitochondrial electron transport chain activity in these cells show decreased activity of Complexes I, II and III compared to normal chondrocytes.

Review > Nat Rev Rheumatol. 2011 Mar;7(3):161-9. doi: 10.1038/nrrheum.2010.213. Epub 2011 Jan 4.

The role of mitochondria in osteoarthritis

Francisco J Blanco [1], Ignacio Rego, Cristina Ruiz-Romero

Affiliations + expand

PMID: 21200395 DOI: 10.1038/nrrheum.2010.213

Abstract

Mitochondria are important regulators of cellular function and survival that may have a key role in aging-related diseases. Mitochondrial DNA (mtDNA) mutations and oxidative stresses are known to contribute to aging-related changes. Osteoarthritis (OA) is an aging-associated rheumatic disease characterized by articular cartilage degradation and elevated chondrocyte mortality. Articular cartilage chondrocytes survive and maintain tissue integrity in an avascular, low-oxygen environment. Recent ex vivo studies have reported mitochondrial dysfunction in human OA chondrocytes, and analyses of mitochondrial electron transport chain activity in these cells show decreased activity of Complexes I, II and III compared to normal chondrocytes. This mitochondrial dysfunction may affect several pathways that have been implicated in cartilage degradation, including oxidative stress, defective chondrocyte biosynthesis and growth responses, increased cytokine-induced chondrocyte inflammation and matrix catabolism, cartilage matrix calcification, and increased chondrocyte apoptosis. Mitochondrial dysfunction in OA chondrocytes may derive from somatic mutations in the mtDNA or from the direct effects of proinflammatory mediators such as cytokines, prostaglandins, reactive oxygen species and nitric oxide. Polymorphisms in mtDNA may become useful as biomarkers for the diagnosis and prognosis of OA, and modulation of serum biomarkers by mtDNA haplogroups supports the concept that mtDNA haplogroups may define specific OA phenotypes in the complex OA process.

Similar articles

Mitochondrial dysfunction in osteoarthritis.
Blanco FJ, López-Armada MJ, Maneiro E.
Mitochondrion. 2004 Sep;4(5-6):715-28. doi: 10.1016/j.mito.2004.07.022. Epub 2004 Oct 1.
PMID: 16120427

Mitochondrial DNA damage is involved in apoptosis caused by pro-inflammatory cytokines in human OA chondrocytes.
Kim J, Xu M, Xo R, Mates A, Wilson GL, Pearsall AW 4th, Grishko V.
Osteoarthritis Cartilage. 2010 Mar;18(3):424-32. doi: 10.1016/j.joca.2009.09.008. Epub 2009 Oct 1.
PMID: 19822235

NEURODEGENERATIVE DISEASES

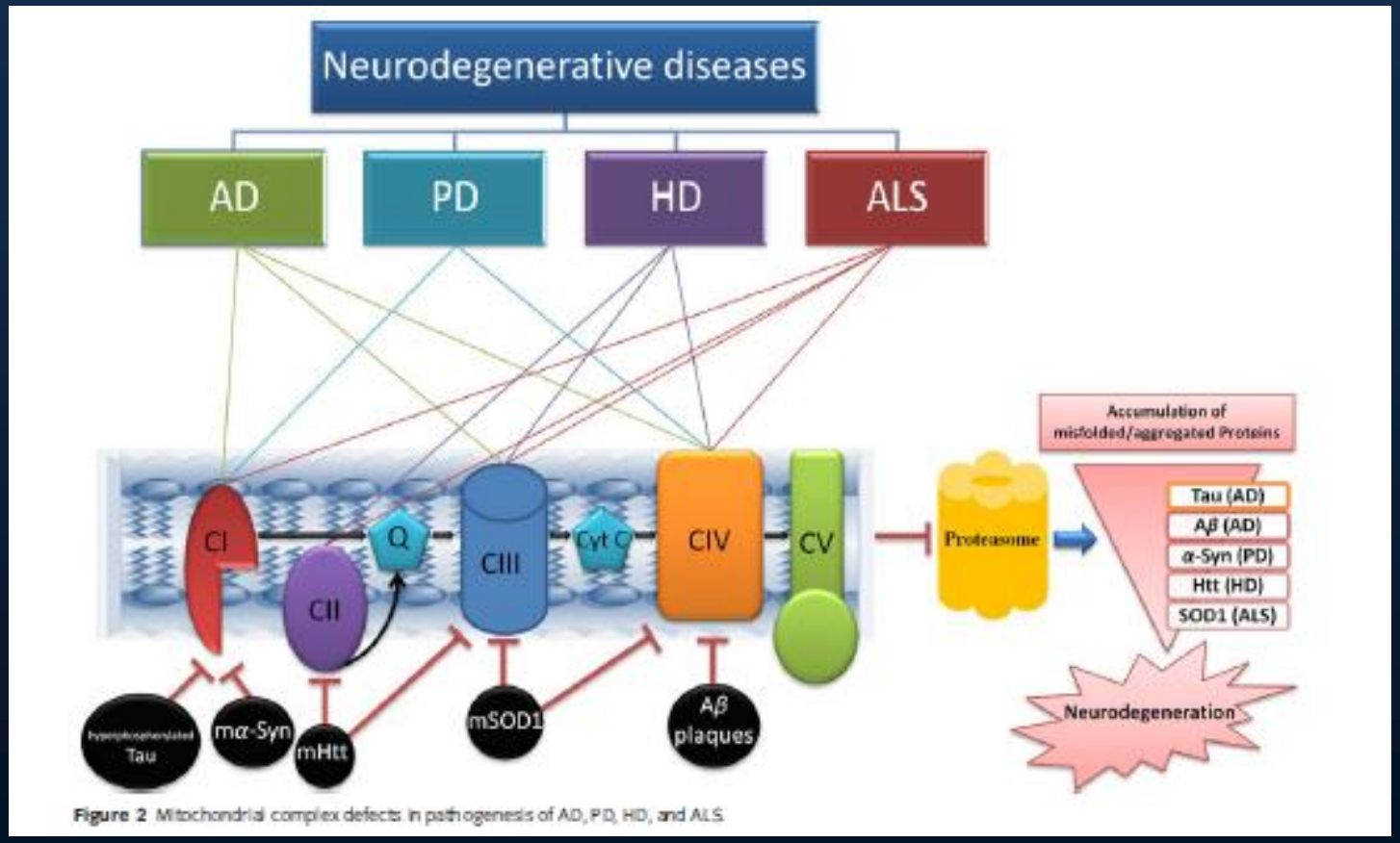


Figure 2 Mitochondrial complex defects in pathogenesis of AD, PD, HD, and ALS.

CNS Neuroscience & Therapeutics

REVIEW

CNS Neuroscience & Therapeutics

Mitochondrial Dysfunction and Biogenesis in Neurodegenerative diseases: Pathogenesis and Treatment

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Keywords
Mitochondrial Biogenesis; Mitochondrial complexes; Mitochondrial Dysfunction; Neurodegenerative Diseases; Pathogenesis; Treatment.

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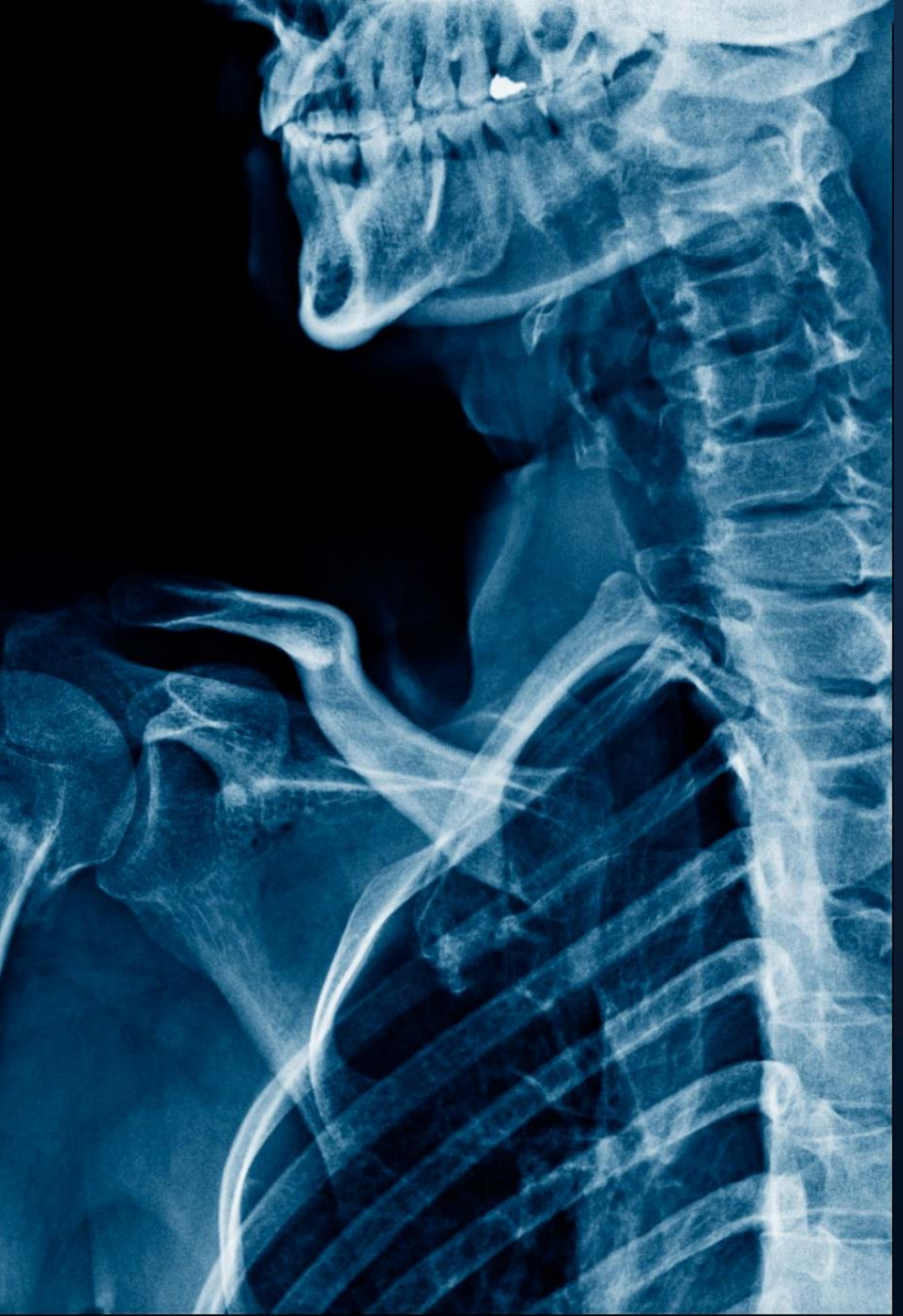
The first two authors contributed equally to this work.

SUMMARY

Neurodegenerative diseases are a heterogeneous group of disorders that are incurable and characterized by the progressive degeneration of the function and structure of the central nervous system (CNS) for reasons that are not yet understood. Neurodegeneration is the umbrella term for the progressive death of nerve cells and loss of brain tissue. Because of their high energy requirements, neurons are especially vulnerable to injury and death from dysfunctional mitochondria. Widespread damage to mitochondria causes cells to die because they can no longer produce enough energy. Several lines of pathological and physiological evidence reveal that impaired mitochondrial function and dynamics play crucial roles in aging and pathogenesis of neurodegenerative diseases. As mitochondria are the major intracellular organelles that regulate both cell survival and death, they are highly considered as a potential target for pharmacological-based therapies. The purpose of this review was to present the current status of our knowledge and understanding of the involvement of mitochondrial dysfunction in pathogenesis of neurodegenerative diseases including Alzheimer's disease (AD), Parkinson's disease (PD), Huntington's disease (HD), and amyotrophic lateral sclerosis (ALS) and the importance of mitochondrial biogenesis as a potential novel therapeutic target for their treatment. Likewise, we highlight a concise overview of the key roles of mitochondrial electron transport chain (ETC.) complexes as well as mitochondrial biogenesis regulators regarding those diseases.

Introduction

Mitochondria play important roles in cell respiratory processes, metabolism, energy production, intracellular signaling, free radical production, and apoptosis [1,2]. These super dynamic organelles can change their morphology, number and function in reaction to physiological situations, and stressors like hormones, diet, temperature, and exercise [2]. Many lines of evidence suggest that mitochondria can critically regulate cell death and survival, play an essential role in aging, and are one of the key features of neurodegeneration [3]. In the central nervous system (CNS), sufficient energy supply which required for neuronal survival and excitability is mostly dependent on mitochondrial sources; therefore, brain is much more vulnerable to mitochondrial dysfunction [4]. Appropriate function of mitochondria is fundamental for activation of proper stress reactions and maintenance of metabolic homeostasis that have been implicated in life span extension and aging [5]. Cellular programs do the task of maintenance of mitochondrial quality and integrity by monitoring and substituting dysfunctional mitochondria with new



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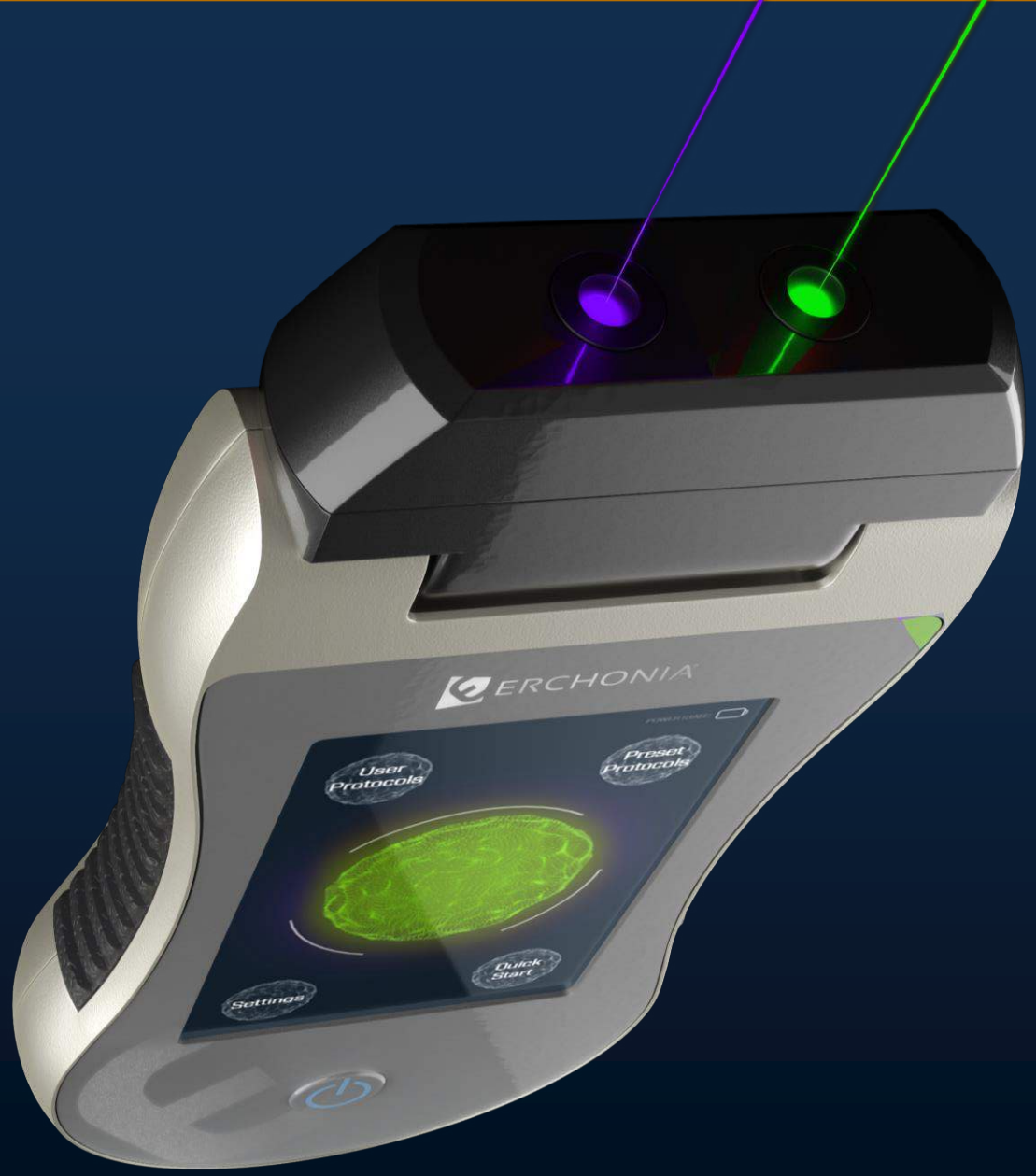
FDA CLINICAL TRIAL

NECK & SHOULDER PAIN

	Red Only	Red & Violet	Green & Violet
Subjects (n)	N=43	N=44	N=43
Duration of pain (months)	61.7	76.58	89.19
Subjects meeting study success criteria, ≥ 30% pain reduction	65%	75%	81%
(%) Improvement in Pain from baseline to immediately after treatment	48%	45%	52%
(%) Improvement in Pain from study endpoint to 48 hrs. post-treatment	43%	50%	65%
(°) Improvement in Range of Motion	14°	29°	32°



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98% OF SUBJECTS RECORDED THEY WERE SATISFIED WITH TREATMENT OUTCOMES

100% OF SUBJECTS RECORDED PAIN REDUCTION AT 24 HOURS POST TREATMENT