Low Level Laser Therapy

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INTRODUCTION

Low level laser therapy (LLLT) is widely used to accelerate tissue repair including wound healing. It is also used to alleviate skin conditions such as acne (Hirsch and Shalita, 2003) and scarring (Patel and Clement 2002). These conditions involve tissue injury, sometimes acquired many years ago. Their improvement is achieved by tissue repair, which can be initiated and stimulated by exposure to low intensities of red light and to some other forms of electromagnetic radiation such as infrared (IR) electromagnetic radiation. Exposure to red light increases blood flow to the skin thus improving its metabolism and stimulates the manufacture of collagen, the protein that gives strength to the skin (Bjerring et al 2002). Other uses of red light and infrared irradiation include accelerating the resolution of inflammation (Dyson 2004) and the reduction of pain (Moore et al 1988; Chow and Barnsley 2005).

The laser technique used to deliver this light is usually termed *low level laser therapy* (LLLT), also referred to as *low intensity laser therapy* (LILT), *low energy photon therapy* (LEPT) and *phototherapy*. Unlike the high intensity medical lasers used to cut and coagulate tissues, LLLT involves the use of medical lasers such as the Beurer SoftLaser™ and Laser Therapeutics SL50 Cluster Laser that operate at intensities too low to damage living tissues. Unlike most LLLT devices that are relatively large and designed for clinical use, the Beurer SoftLaser™ is a small, single-diode, hand held device emitting red light and designed for home use. The Laser Therapeutics SL50 Cluster Laser consists of twelve laser diodes combined in a treatment head but smaller than the devices designed for clinical use. These devices are now available for home use; the Laser Therapeutics Inc. SL50 Cluster Laser is an example of a cluster probe suitable for home use. It contains 8 laser diodes that emit red light at 640-660 nm and 4 laser diodes that emit infrared (IR) electromagnetic radiation at 775-795 nm.

The Beurer SL30 Softlaser

The Laser Therapeutics SL50 Cluster Laser

Wound healing can be stimulated by photons from the visible and infrared parts of the electromagnetic spectrum when applied to the skin and mucous membranes by low level lasers and light-emitting diodes (LEDs) at appropriate wavelengths, powers and durations. When absorbed these photons induce cellular changes which accelerate tissue repair (Fulop et al 2009) and relieve pain (Chow, Barnsley 2005). Changes induced by photons in immune cells and stem cells assist in the acceleration of wound healing (Dyson 2008); changes induced in nerve conduction assist in the short term relief of pain (Baxter 1994)

LIGHT

Light consists of photons transmitted at wavelengths of the electromagnetic spectrum that are visible to the human eye. This part of the spectrum extends from violet to red. Infrared (IR) is just beyond the visible range. The perceived colour depends on the wavelength. White light is a mixture of all the visible wavelengths. For photons to reach the skin, all that is required is that it be either exposed to air or to be covered by a transparent dressing. Exposure to red light and/or infrared radiation can stimulate the healing of both chronic injuries of the skin (Mester et al 1985) and acute injuries (Dyson & Young 1986).

Photons are quanta of electromagnetic radiation that originate in the burning gases of the sun. They have zero mass, are electrically neutral, behave both as particles and as waves and are pure energy. When they are absorbed this energy is transferred to the chemicals that absorb them, for example cytochromes (coloured materials present in all cells). Absorption of photons by cytochrome C oxidase in mitochondria increases the amount of energy-rich ATP the mitochondria produce, (Karu 1988) and also temporarily increases cell membrane permeability to calcium ions, the latter acting as a stimulus for cell activity (Young et al 1990). Depending on their type and metabolic status, the cells are induced to proliferate, manufacture proteins, secrete mediators, contract, conduct, phagocytose pathogens or kill cancer cells. Following absorption, photons trigger metabolic activities that stimulate wound healing and relieve pain. They can be delivered in effective wavelengths and doses by low level laser therapy (LLLT) devices (Tuner, Hode 2002).

LASER

This is an acronym for **L**ight **A**mplification by the **S**timulated **E**mission of **R**adiation. The stimulated emission of radiation occurs when a photon interacts with an energized atom. When an atom is energized, for example by electricity, one of its electrons is excited, i.e. raised to a higher energy orbit than its orbit when in the resting state. If the energy of the incident photon is equal to the energy difference between the electron's excited and resting states, then stimulated emission of a photon occurs and the excited electron returns to its resting state. This photon has the same properties as the incident photon, which is also emitted. This process is repeated in the adjacent energized atoms, producing a laser beam. Unlike light from non-laser sources, this light is:

- Monochromatic, i.e. of a single wavelength
- Collimated, i.e. its light rays are non-divergent
- Coherent, i.e. in phase, the troughs and peaks of the waves coincide in time and space.

With regard to the biomedical effects of LLLT, wavelength is particularly important. To produce an effect, the light must be absorbed, and absorption is wavelength-specific. Different substances absorb light of different wavelengths. Mitochondria, present in all mammalian cells except erythrocytes, contain cytochromes that absorb red light**.** Light emitting diodes emitting effective

wavelengths are now often used instead of the more expensive lasers, making phototherapy more economical. **L**ight could now be substituted for **L**aser in the LLLT acronym.

Only low powers (5-500 milliwatts) are required for effectiveness. The duration for which the photons are applied is clinically important because there is a temporal window of effectiveness. Within this window longer treatments are more effective than shorter treatments in accelerating healing, probably because they allow more of the circulating immune cells and stem cells of the body to be exposed to photons. Energy (power x duration) doses of 4-20 Joules/cm^2 are usually effective in stimulating wound healing and relieving pain (Tuner, Hode 2002).

Generally red or infrared electromagnetic radiation is employed using either single-diode probes to irradiate small areas such as acupuncture points and trigger points or cluster probes to irradiate larger areas such as wounds or joints.

LLLT EQUIPMENT

This has three essential components:

1. *Lasing medium*, which is capable of being energized sufficiently for light amplification by the stimulated emission of radiation to occur
2. *Resonating cavity* containing the lasing medium
3. *Power source* that transmits energy into the lasing medium.

The type of lasing medium used determines the wavelength, and therefore the colour, of the laser beam. For example, a HeNe laser, in which the lasing medium is a mixture of helium and neon gases, produces red light with a wavelength of 632.8 nm. Gallium, aluminium and arsenide, the lasing medium of GaAlAs semiconductor diodes, also produces monochromatic radiation, the wavelength of which depends on the ratio of these three materials and is in the red-infrared range of the electromagnetic spectrum, typically 630-950 nm.

The resonating cavity containing the lasing medium has two parallel surfaces, one being totally reflecting, the other being partially reflecting. Photons emitted from the lasing medium are reflected between these surfaces, some of them leaving through the partially reflecting surface as the laser beam. The cavity of a HeNe laser is many cms long, whereas that of a GaAlAs semiconductor diode is tiny, the diode being the lasing medium and its polished ends the reflecting surfaces.

Modern low intensity laser therapy devices are generally of the GaAlAs type. Their treatment heads may contain either one or many diodes. Those with one diode resemble laser pointers and are designed to treat acupuncture and trigger points; they can also be used to treat points in and around skin injuries. Those with many diodes are generally called cluster probes and allow large areas to be treated rapidly. The diodes may be housed in a rigid head or in a flexible material. The latter can be applied around curved surfaces such as the shoulder. Each diode emits either red or IR radiation. Red light is absorbed by all cells, whereas different wavelengths in the infrared range appear to target specific cell types.

The power source for a LLLT device may be either a battery or mains electricity. Many LLLT devices are portable. The main function of the power source is to energize the lasing medium.

HOW LLLT PRODUCES ITS EFFECTS

For LLLT to be effective, the tissue targeted must absorb photons. Absorption is wavelength dependent. Red light is absorbed by cytochromes in the mitochondria; all human cells, other than

mature red blood cells (erythrocytes) contain mitochondria. Provided that appropriate wavelengths and energy densities are used, cell activity can be stimulated if it is suboptimal. Cells in which this has been investigated include mammalian keratinocytes, lymphocytes, macrophages, mast cells, fibroblasts and endothelial cells, all cells of significance in tissue repair. Much of the research on this has been reviewed by Baxter (1994) and by Tuner and Hode (2002). Cells affected by LLLT show a temporary increase in permeability of their cell membranes to calcium ions (Young et al 1990). This may be an important component of the mechanism by which LLLT modulates cell activity; other electrotherapeutic modalities, such as ultrasound, may act in a similar fashion (Dyson 2004).

The triggering of cell activity by reversible changes in membrane permeability when photons are absorbed could be responsible for the stimulation of tissue repair (Young & Dyson 1993). Increase in calcium uptake by macrophages exposed to red light and IR *in vitro* has been shown to be wavelength and energy density dependent. Of the wavelengths tested, 660, 820 and 870 nm were effective; 880 nm was ineffective. These same wavelengths also affected growth factor production by the macrophages, 660, 820 and 870 nm being stimulatory, whereas 880 nm was not. Energy densities of 4 and 8 J/cm^2 were found to be effective; 2 and 19 J/cm^2 were not (Young et al 1990). Red light of 660 nm wavelength is absorbed by the cytochromes of mitochondria, where it stimulates ATP production and increases cytoplasmic H^+ concentration, which can affect cell membrane permeability (Karu 1988). IR radiation of 820 and 870 nm may be absorbed by components of the cell membrane. Some of these components vary in different cell types, which may be why the IR wavelengths absorbed by cells differ according to the cell type. For example, 870 nm affects macrophages (Young et al 1990) but not mast cells (El Sayed & Dyson 1990). It may be possible to selectively stimulate macrophages but not mast cells *in vivo* by exposure to an 870 nm probe.

Following a reversible change in membrane permeability to calcium ions, the cells respond by doing what they are programmed to do. In the case of macrophages, this is to produce soluble protein mediators such as growth factors and to phagocytose debris, whereas fibroblasts manufacture collagen and other extracellular components of the dermis.

The molecular mechanisms by which LLLT affects cell activity begin with photoreception, when the photons are absorbed. This is followed by signal transduction, amplification and a photoresponse, e.g. cell proliferation, protein synthesis and growth factor production, all of which assist in tissue repair. Membrane structure differs according to the cell type, which, if IR is absorbed by parts of the membrane, may explain why different cell types absorb different wavelengths of IR. Theoretically, it should be possible, by the judicious selection of IR wavelengths, to affect some cell types while leaving others unaffected. In contrast red light, since it is absorbed by the mitochondrial cytochromes present in all mammalian cells other than erythrocytes, and also by the haemoglobin contained in erythrocytes, affects all mammalian cells.

WOUND HEALING

Wound healing consists of a closely regulated cascade of events that follow injury and in skin normally result in the regeneration of the epidermis and the replacement of the damaged dermis with scar tissue. The events can be grouped into the sequential and overlapping phases of *inflammation, proliferation,* and *remodeling.* If the dermis is damaged, haemostasis is the initial major component of inflammation, following which debris and damaged tissue are removed from the wound site by neutrophils and macrophages. Antigens are also detected and presented to T-lymphocytes by macrophages such as Langerhans cells. All these cells are components of the immune system. During proliferation, angiogenesis and the formation of matrix rich in type III collagen results in the production of granulation tissue over which the epidermis migrates and

regenerates. Myofibroblasts which develop in the granulation tissue produce wound contraction, reducing the size of the wound. During remodeling, the granulation tissue is gradually transformed into less vascular, less cellular and more collagenous scar tissue which replaces the injured dermis. Much of the type III collagen is replaced by stronger type I collagen arranged in wider fibre bundles, increasing the tensile strength of the scar tissue although this remains weaker than uninjured dermis (Ovington, Schultz 2004).

Regulation of wound healing

For wound healing to be successful, the multitude of events comprising it must be spatially and temporally regulated. This regulation is dependent on intercellular communication. Soluble protein mediators (SPMs), produced initially by immune cells and consisting of chemokines, cytokines and growth factors, together with hormones, neurotransmitters and their receptors are involved in this communication; protease and protease inhibitors modify the wound bed and affect the ease with which cells can migrate within it. (Ovington and Schultz 2004). SPMs are produced mainly by immune cells, eg neutrophils, macrophages and lymphocytes, but also by peripheral nerve fibres, fibroblasts, endothelial cells and other non-immune cells. Following SPM synthesis and secretion, the SPMs diffuse to target cells involved in the healing process or are transported to them in blood and lymph vessels. They bind to specific receptor sites on the target cell surface. Binding triggers cell activation, the activity depending on the target cell type. For example, myofibroblasts will contract, fibroblasts will (depending on their stage of differentiation) either proliferate or secrete matrix materials, endothelial cells will produce new blood capillaries.

SPM actions during wound healing include the following:

1. Initiation of inflammation, by Il-1, TNF, etc.

2. Cell recruitment to wound bed, by PAF, Il-1, Il-3, Il-6, TNF, etc.

3. Debris removal, by Il-1, Il-2, Il-4, Il-5, Il-6, TNF, etc.

4. Promotion of proliferative phase of healing, by FGF, PDGF, TGF-b, Il-1, Il-6, TNF etc

Key: Il = Interleukin; TNF = Tumor necrosis factor, PAF = Platelet activating factor, FGF = Fibroblast growth factor, PDGF = Platelet derived growth factor, TGF-b = Transforming growth factor-beta.

Acute inflammation is a vital stage in healing, setting the stage for the proliferative phase by the removal of debris and pathogens, and by the secretion of regulatory SPMs. In contrast, chronic inflammation inhibits healing. For chronic wounds to heal, acute inflammation must be induced in them by, for example, debridement.

LLLT ACCELERATES WOUND HEALING

Many publications during the last 30 years report the acceleration of delayed healing by LLLT and other forms of phototherapy when used appropriately. To quote from a recent meta-analysis '…our findings leave no doubt whatsoever that phototherapy promotes tissue repair' (Fulop et al 2009).

In addition to treating the wound bed, it is recommended that the intact tissue around the wound also be treated (Baxter 1994). This will induce the peripheral nerve fibres and immune cells present in epidermis and dermis to secrete SPMs. Acute inflammation is a vital part of wound healing. Its resolution should be accelerated so that the proliferative phase of repair begins earlier, thus accelerating the healing process. Cells that have absorbed sufficient quantities of photons of effective wavelengths will secrete these SPMs earlier and thus accelerate healing. In contrast, chronic inflammation inhibits repair; it has to be converted to acute inflammation for healing to progress. This may require debridement and should be followed as soon as possible by phototherapy so that the immune cells are stimulated to secrete SPMs. It is recommended that this be continued, ideally on a daily basis or at every dressing change, throughout the acute inflammatory phase of repair. Continuing the treatment into the proliferative phase may also be of value since phototherapy can stimulate the proliferation of endothelial cells (Ghali, Dyson 1992) and fibroblasts (Hawkins, Abrahamse 2006), accelerating the development of the granulation tissue over which epidermal cells migrate.

The Beurer SoftLaser™

This hand-held LLLT device is a low power Class 2M laser manufactured by Beurer GmbH. It contains a single 5 mW GaAlAs diode producing red light of 635-670 nm wavelength. It is powered by 2 AAA batteries.

Application of SoftLaser™ to Skin

The probe is placed in contact with clean skin or over a transparent dressing at right angles to the skin's surface and moved slowly over the region to be treated for a few minutes, typically 3-6 minutes for a region of about 1 cm diameter. A convenient way to use it is twice daily, shortly after cleansing the skin in the morning and evening, and before the application of a moisturizing cream and/or cosmetics.

Laser Therapeutics Inc. SL50

The Laser Therapeutics Inc. SL50 is an example of a cluster probe suitable for home use. It has 8 laser diodes that emit red light at 640-660 nm and 4 laser diodes that emit infrared electromagnetic radiation at 775-795 nm.

Application of Laser Therapeutics Inc. SL50 to Skin

The cluster probe is placed in contact with clean skin, or, if the skin has an open wound, over a transparent wound dressing. The cluster probe does not operate if contact is broken and there is no need to move the cluster during the treatment period, typically 5 minutes per point.

LLLT EFFECTS ON DAMAGED SKIN

Effects of the Laser Therapeutics SL50 Cluster Laser and Beurer SoftLaser™ on Skin

The Laser Therapeutics SL50 Cluster Laser and Beurer SoftLaser™ have been reported by its users to:

- Reduce wrinkles
- Make scars less visible
- Tighten large pores
- Elevate pock marks
- Improve skin tone
- Give a temporary radiance to the skin
- Soften chapped lips
- Accelerate wound healing

Treatment of damaged skin with red light accelerates the resolution of acute inflammation, leading to faster repair (Dyson 2004). The stimulated secretion of collagen by fibroblasts at the site of a wrinkle or of a pock mark will increase the thickness of the dermis locally, helping to fill in the tissue deficit. The gradual removal of excessive scar tissue may be due to the activation of fibroblasts, fibrocytes and other cells in and around the scar.

As with any other technique, tissue repair can only be stimulated by LLLT if it is absent or delayed. In these circumstances, epithelialisation and granulation tissue production can be stimulated by LLLT as can wound contraction (Dyson & Young 1986) which reduces the area in which scar tissue is produced resulting in less obvious scarring.

THE IMMUNE SYSTEM

The immune system plays a vital role in the response of the body to pathogens, cancer and injury. The main cellular components of the immune system are lymphocytes and macrophages, including the Langerhans cells of the epidermis. These are located either in peripheral tissues such as the epidermis and dermis of the skin, the epithelium and lamina propria of mucous membranes and superficial lymph nodes or in deeper organs such as the deep lymph nodes. The key molecular components of the immune system are antibodies and SPMs such as cytokines and growth factors.

All the components of the immune system are linked by blood vessels and lymphatic vessels, via which immune cells and the molecules they secrete are carried around the body. SPMs released from peripheral immune cells such as Langerhans cells in response to the *direct* action of absorbed photons can be transported to and affect cells that have not been exposed to photons. Injuries other than those directly exposed to photons can therefore be affected by them *indirectly*.

Peripheral immune cells are located mainly located in the skin associated lymphoid tissue (SALT) and mucous membrane associated lymphoid tissue (MALT). Their superficial location renders them accessible to photons during phototherapy. Other immune cells, the natural killer (NK) cells, patrol the body in the blood and lymph, lysing cancer cells and virus-infected cells. The initial response of the immune system is non-specific and immediate. It is enhanced by cytotoxins secreted by the NK cells. During it neutrophils, macrophages, NK cells, T lymphocytes and antimicrobial proteins inhibit the spread of the invading substances. SPMs released locally recruit immune cells to the infected region and promote tissue repair. SPMs consist of 3 groups:

1. CHEMOKINES, for example fractalkine, are chemotactic molecules that attract and activate inflammatory cells

2. CYTOKINES, for example interleukins, are molecules that regulate division and differentiation of immune (inflammatory) cells

3. GROWTH FACTORS, for example platelet derived growth factor (PDGF), are molecules that stimulate division of both immune and non-immune (non-inflammatory) cells.

Immune or inflammatory cells include Langerhans cells, neutrophils, natural killer cells, monocytes, macrophages, T & B lymphocytes, plasma cells and mast cells. All play significant roles during the inflammatory and proliferative phases of wound healing (Martin, Leibovich 2005). Non-immune or non-inflammatory cells that are of importance during wound healing include epidermal cells, endothelial cells, fibroblasts and myofibroblasts.

Photons can be absorbed not only by the superficially-located immune cells of the SALT and MALT and but also by immune cells and stem cells in transit through the superficially-located blood and lymph capillaries of the skin and mucous membranes. Phototherapy can have a direct effect on the secretion of SPMs by these cells. By doing so it can accelerate the resolution of inflammation and thereby accelerate repair if this is delayed. The deeper cells of the immune system and also non-immune cells of injured tissues can be affected indirectly by SPMs released from peripherally-located cells that have absorbed photons. Phototherapy thus has both local and systemic effects. Cells of injured tissues are more sensitive to phototherapy that cells of intact tissues, so lower power and energy levels can affect them while leaving less susceptible cells unaffected.

The secretion of different SPMs may assist chronic wounds to heal by allowing them to progress from inflammation to the proliferative phase of wound healing when granulation tissue is formed and re-epithelialization occurs. Because of the indirect, systemic, effects of photons, the treatment of one wound of a patient may lead to improvements not only in this wound but in the patient's other wounds.

Link between cutaneous nerves and SALT

Cutaneous contact hypersensitivity (CH) reactions are closely correlated with Langerhans cells (LC), macrophages that arise from stem cells in the bone marrow and migrate into the epidermis (Streilen et al 1999). Also known as epidermal dendritic cells they help to activate the immune system by presenting antigens to lymphocytes. LCs may be linked synaptically to cutaneous nerve termini containing calcitonin gene-related peptide (CGRP), suggesting that there is a link between innervation and immune responses in the skin. It has been proposed that 'cutaneous nerves dictate whether antigen applied to the skin will lead to sensitivity or tolerance'(Streilen et al 1999), linking the nervous system to the immune system. There is evidence that phototherapy can affect mast cell degranulation (El Sayed and Dyson 1990) resulting in activation of pain fibres. Nerve conduction (Vinck et al 2005) is also affected by phototherapy, supporting the hypothesis that it may affect the immune system via the nervous system.

CLINICAL RELEVANCE OF EFFECTS OF LLLT ON IMMUNE SYSTEM TO WOUND HEALING

Phototherapy has been used for many decades to treat the chronic wounds of patients (Mester et al 1985). It is suggested that treatment of the intact skin around chronic wounds may, provided that the correct parameters are used, activate immune cells of the SALT. This will increase the efficiency with which pathogens and debris are removed and stimulate the release of cytokines of value in the inflammatory and proliferative phases of repair. Furthermore latent SPMs such as transforming growth factor–beta 1 (TGF-ß1), of crucial importance in wound healing, can be activated by phototherapy. In addition to exposing SALT to phototherapy, irradiation of peripheral lymph nodes could also be of value in that more immune cells will be exposed to the beneficial effects of phototherapy. Immune cells from these nodes will enter the lymphatics and be transported to the wounds where they and the cytokines they secrete can assist in the healing process (Dyson 2008).

It is possible that variation in the treatment parameters used may determine which SPMs are secreted. Different mediators are necessary for different activities during wound healing, including the initiation of inflammation, the recruitment of inflammatory and non-inflammatory cells to the

wound bed, debris removal by neutrophils and macrophages, and the induction of granulation tissue formation. Chronic wounds may be trapped in the inflammatory phase of healing; compared with healing wounds, they have more inflammatory cytokines, higher protease activity, lower mitogenic activity and contain fewer mitotically competent cells (Dyson 2008). Selection of appropriate treatment parameters may move them on to the proliferative phase of healing. What these parameters are remains to be determined. Antibody array screening allows the rapid monitoring of the induction of different SPMs (Chang et al 2009). Selection of the best parameters could optimize the treatment of chronic wounds with phototherapy, helping improve the quality of life of millions of people world wide.

Cellular effects relevant to skin repair

The cellular effects of LLLT relevant to skin repair include the stimulation of

- adenosine triphosphate (ATP) production
- growth factor release by macrophages
- keratinocyte proliferation
- collagen synthesis
- angiogenesis.

All of the above are required for skin to renew itself and repair the damage done to it by, for example, environmental factors such as excessive exposure to the elements, damage that accumulates with age.

Temporary vasodilatation following the exposure to red light improves the transport of essential nutrients and oxygen to the skin and the removal of toxic waste materials from it. It also gives sallow skin a radiant glow.

PAIN RELIEF BY LLLT

Although many of the reports of pain relief following exposure to LLLT are anecdotal, there have been a number of reports based on trials aimed at assessing LLLT as an antinociceptive or analgesic modality, one of the earliest being that of Walker 1983 who implicated alteration in serotonin metabolism as one mechanism of LLLT-mediated analgesia.

Rheumatoid pain

Walker et al (1987) reported a highly significant reduction ($p<0.001$) in the levels of pain and analgesic medication intake reported by rheumatic patients either treated with low intensity red laser or sham-irradiated, pain relief being greater in those given laser treatment. Palmgren et al (1989) found that treatment of the small joints of the hand in rheumatic patients with low intensity infrared laser was followed by reduced pain and swelling, reduced early morning stiffness and increased grip strength and range of movement. In contrast Basford et al (1987) found that red laser irradiation of the osteoarthritic thumbs of patients was not followed by significant reduction in pain; however, the power and energy levels used (0.9 mW and 0.081J) are well below those recommended for clinical application (Baxter 1994) and may have been sub threshold.

Chronic neurogenic pain

Moore et al (1988a) have investigated the effect of red laser in the treatment of patients with chronic neurogenic pain including that of post-herpetic neuralgia. It was found that there was a significant reduction in reported pain following treatment in comparison to that in sham-irradiated patients. Similar effects have been reported by Hong et al (1990) using the same equipment.

Mechanisms
It has been suggested by Obata et al (1990) that laser-mediated relief of rheumatic pain may be linked to autonomic changes that produce vasodilatation and slight increases in local temperature. It is also possible that laser treatment affects the synthesis, release and metabolism of a range of neurochemicals involved in nerve transmission and pain relief (Walker 1983). Relief following the stimulation of acupuncture points with LILT has been ascribed to the production of endogenous opiate-like peptides and serotonin (Zhong et al 1989).

CONCLUSIONS

Cells of the immune system initiate acute inflammation, an essential part of the healing process. The peripheral components of the immune system such as the Langerhans cells of the epidermis are readily accessible to photons and can be affected by them directly, triggering the release of a variety of SPMs which orchestrate the sequential events of the inflammatory, proliferative and remodeling phases of wound healing. These SPMs can either diffuse or be transported by blood and lymph vessels to the other parts of the immune system and to distant injured tissue where they can initiate reparative changes, thus amplifying the direct effects of the superficially absorbed photons. Cells can therefore be affected indirectly by photons without the need to absorb them. Photon-induced changes in peripherally located nerve fibers and in the endocrine system can also modulate wound healing and relieve pain either directly or indirectly. There is some evidence that exposure of immune cells to different parameters of phototherapy can alter the types of SPMs produced. Further research on the effects of different parameters on SPM production by immune cells is indicated. It may therefore be possible to select the most effective parameters to use to accelerate healing where it is either delayed or chronic.

Scarring associated with acne and skin deterioration due to ageing and sun damage can be alleviated by LLLT. These skin conditions involve tissue injury, the repair of which is improved by exposure to LLLT in the form of red and IR radiation. LLLT can reduce the duration of inflammation, improving tissue repair where this is delayed or defective. It can also reduce both acute and chronic pain. By assisting in the resolution of inflammation, the proliferative phase of tissue repair begins earlier and the reparative process is completed earlier. Cell activity is jump-started by changes in membrane permeability. This occurs when the cells absorb red and/or infrared radiation. The cells are also energized when red light is absorbed by their mitochondria, stimulating the synthesis of ATP and thus providing readily available energy for cell activity. The improvement in the skin produced by LLLT has been described as skin rejuvenation (Lee 2002). The portable Beurer SoftLaser™ and the Laser Therapeutics Inc. SL50 **t**ake LLLT from the clinic into the home where it can be used regularly for skin care and pain relief.

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CURRICULUM VITAE

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DATE OF BIRTH 4th November 1939

MARITAL STATUS Widowed

DEGREES

1961 BSc Class I Special Honours in Zoology
Specialist Subject: Embryology
Bedford College, University of London

1965 PhD
Topic: Wound Healing
Bedford College, University of London

ADDITIONAL ACADEMIC AWARDS

1958 State Scholarship
Pfeiffer Scholarship (University of London)
West Riding of Yorkshire County Major Scholarship (Honorary)

1961 Busk-Howell Research Scholarship (University of London)
Department of Scientific & Industrial Research Postgraduate Scholarship

1988 History of Medical Ultrasound Pioneer Award (presented by the American Institute of Ultrasound in Medicine and the World Federation for Ultrasound in Medicine and Biology, in recognition of contributions to the development of medical ultrasound)

1989 Elected Honorary Fellow of the American Institute of Ultrasound in Medicine for "outstanding contributions to the field of medical ultrasound"

1990 Elected Honorary Fellow of the Chartered Society of Physiotherapy for research into the biological effects of electrotherapy

1992 Elected President of the International Laser Therapy Association

1996 Awarded the degree of Doctor of Humane Letters (honoris causa) by the Pennsylvania College of Podiatric Medicine

1998 Elected Honorary Member of the World Association of Laser Therapy

1998 Conferred with the title of Emeritus Reader in Biology of Tissue Repair by King's College London, University of London, in recognition of services to the University and the subject of Tissue Repair.

PRESENT APPOINTMENTS

Since 1991: Biomedical Consultant and Director, Dyderm Ltd.
Since 1996: Research Director, Quality Medical Instruments Ltd.
Since 1998: Emeritus Reader in Biology of Tissue Repair, University of London
Since 1998: Director of Research & Development, Longport Inc.
Since 1998: Member of Board of Directors of World Walk Foundation.
Since 2000: Executive Vice-President, Longport Inc.

PAST EMPLOYMENT

1964-70 Research Associate
Department of Anatomy
Guy's Hospital Medical School
University of London

1970-75 Lecturer
Department of Anatomy
Guy's Hospital Medical School
University of London

1975-87 Senior Lecturer
Department of Anatomy
Guy's Hospital Medical School
University of London

1987-98 Reader in Biology of Tissue Repair
Head, Tissue Repair Research Unit
Department of Anatomy
Guy's Hospital Medical School (later United Medical and Dental Schools of Guy's and St Thomas's Hospitals)
University of London

1998-2000 Director of Research and Development
Longport Inc
740 South Chester Road, Suite A
Swarthmore, PA 19081, USA

PROFESSIONAL APPOINTMENTS

Teaching

1974 Recognised as a Teacher of the University of London
1974-98 Member of the Board of Studies in Human Anatomy and Morphology, University of London
1976-98 Examiner in Cytology and Histology, Guy's Hospital Medical School
1976-98 Examiner in Anatomy, Guy's Hospital Medical School

1981-98 Member of the Preclinical Subjects Sub-Committee of the Board of Studies in Dentistry, University of London

1985-98 Member of the Panel of Visiting Examiners in Anatomy, University of London

1987-88 Member of Working Party considering introduction of projects into the clinical curriculum (UMDS Education Committee)

1987 Appointed as a Teacher of the University of London and as Reader in Biology of Tissue Repair

1988 Elected by the Board of Studies in Human Anatomy and Morphology as Chairman of the Course-Unit Approval Sub-Committee and Chairman of the Panel of Course Unit Examiners, University of London

1989-95 Admissions Tutor (Medical), United Medical and Dental Schools of Guy's and St Thomas' Hospitals (UMDS)

1992-95 Organiser of Access Course for prospective medical and dental students at UMDS in conjunction with Lambeth College

EDITORIAL

Since 1977 Member of Advisory Editorial Board of "Ultrasound in Medicine and Biology"

1979-87 Associate Editor of "Gray's Anatomy" 36th edition

Since 1985 Member of the Advisory Editorial Board on "Physiotherapy Practice"

1989 Appointed as an Editor of " Gray's Anatomy" 37th edition

1991 Appointed to the Editorial Board of "Gray's Anatomy" 38th edition

1990 Appointed Section Co-Editor of "Gray's Anatomy" 38th edition.

ADVISORY

1973-74 Adviser to the Bureau of Radiological Health of the Food and Drugs Administration on the performance requirements of ultrasonic therapy equipment

1980 Member of the Scientific Committee of the 10th LH Gray Conference, University of Oxford

Since 1983 Member of the International Anatomical Nomenclature Committee

1984-93 Member of the European Committee for Ultrasonic Radiation Safety

1986 Appointed as a Reviewer by the American Medical Association

1986-87 Adviser to the Electrotherapy Working Party of the Chartered Society of Physiotherapy

1988-90 Honorary Treasurer of the International Laser Therapy Association

1989 Member of Chief Scientist's Assessment Group, Department of Health, for the Nursing Practice Research Unit, University of Surrey

Since 1989 Academic Board of UMDS Representative to Lewisham and North Southwark Ethical Committee (now the Guy's Hospital Local Research Ethics Committee)

1990-95 Member of the Special Advisory Committee in Health Studies, University of London

1990-92 President Elect of the International Laser Therapy Association

1992-94 President of the International Laser Therapy Association

Since 1991 Member of the Electrotherapy Advisory Group of the Chartered Society of Physiotherapy

1992-96 Vice-Chairman, Division of Biological Services, UMDS

CURRENT MEMBERSHIP OF EDITORIAL BOARDS

Laser Therapy, Physical Therapy Reviews, Physiotherapy Research International, Physiotherapy Practice, Ultrasound in Medicine and Biology.

REVIEWING

1. Manuscripts submitted to the Journals listed above and also to the British Journal of Surgery, Clincal Materials, Journal of Anatomy, Lasers in Medical Science, Lasers in Surgery and Medicine, Physiotherapy and Ultrasonics.
2.Grant Applications submitted to the Medical Research Council (MRC), Science and Engineering Research Council (SERC) and Wellcome Trust.

MEMBERSHIP OF SOCIETIES

Anatomical Society of Great Britain and Ireland, British Connective Tissue Society, British Medical Ultrasound Society, British Orthopaedic Research Society, British Society for Cell Biology, British Society for Developmental Biology, Institute of Biology, World Association of Laser Therapy, North American Association of Laser Therapy, Zoological Society of London.

EXAMINING

2nd BDS and BMS (UMDS); Anatomy Course Units (UMDS); 2nd BDS (University of Birmingham); BSc (Universities of Brighton, London and Salford); MSc (University of London); PhD (Universities of Aberdeen, Brighton London UK, London Ontario, Manchester, Queens University Belfast, Ulster).

DEPARTMENTAL RESPONSIBILITIES WHILE EMPLOYED AS READER

1.**Teaching**

(a) gross anatomy to preclinical medical and dental students
(b) histology to preclinical medical and dental students
(c) supervision of BSc, MPhil, PhD and MBPhD students

2. **Research**

(a) analysis of effects of MHz and kHz ultrasound on tissue repair
(b) bioeffects of low level laser therapy
(c) control of angiogenesis during wound healing
(d) development of the Longport Digital Scanner, a noninvasive high resolution instrument currently utilising 20MHz ultrasound associated with fractal analysis, as a device for assessing structural changes associated with the development and repair of damage in skin and subcutaneous soft tissue.
(e) diabetes associated changes in skin of relevance to wound healing
(f) role of the immune system in wound healing

This work was carried out in the Tissue Repair Research Unit supported by grants obtained from the MRC, SERC, Department of Health, Department of Trade and Industry, National Fund for Research into Crippling Diseases, University of Kuwait, and a number of industrial companies including Johnson and Johnson, Omega Universal Technologies, Smith and Nephew, and Topox.

RESEARCH

1. Dyson, M. (1965) An experimental study of wound healing in *Arion*. PhD Thesis, University of London.
2. Joseph, J. and Dyson, M. (1965) Sex differences in the rate of tissue regeneration in the rabbit's ear. Nature, 209, 599-600.
3. Joseph, J. and Dyson, M. (1966) Tissue replacement in the rabbit's ear. Br.J. Surg., 53, 372-380.
4. Joseph, J. and Dyson, M. (1966) The effect of anabolic androgens on tissue replacement in the rabbit's ear. Nature, 211, 193-194.
5. Pond, J.B. and Dyson, M. (1967) A device for the study of the effect of ultrasound on tissue growth in rabbits' ears. J. Sci. Instrum., 44, 165-166.
6. Dyson, M. and Joseph, J. (1968) The effect of androgens on tissue regeneration. J. Anat.103, 491-505.
7. Dyson, M., Joseph. J, Pond, J. and Warwick, R. (1968) The stimulation of tissue regeneration by means of ultrasound. Clin. Sci., 35,273-285.
8. Joseph, J. and Dyson, M. (1970) The effect of abdominal wounding on the rate of regeneration in the rabbit's ear. Experientia, 26, 66-67.
9. Dyson, M. and Pond, J. (1970) The effect of pulsed ultrasound on tissue regeneration. Physiotherapy, 56, 136-142.
10. Dyson, M., Pond, J., Joseph, J., and Warwick, R. (1970) Stimulation of tissue regeneration by pulsed, plane wave ultrasound. IEEE Trans. Sonics and Ultrasonics, SU-17, 133-139.
11. Joseph, J. and Dyson, M. (1970) The effect of mechanical obstruction on tissue regeneration in the rabbit's ear. Br. J. Surg., 58, 277-285.
12. Pond, J., Woodward, B. and Dyson, M. (1971) A microscope viewing ultrasonic irradiation chamber. Phys. Med. Biol., 16, 521-524.
13. Joseph, J. and Dyson, M. (1971) The effects of oxymethalone on tissue replacement in the rabbit's ear. Experientia, 27, 1309-1310.
14. Dyson, M., Pond, J. and Woodward, B. (1971) Flow of red blood cells stopped by ultrasound. Nature, 232, 572-573.
15. Dyson, M. and Joseph, J. (1971) The effects of female sex hormones on tissue regeneration. J. Endocrinol., 51, 685-697.
16. Taylor, K. and Dyson, M. (1972) Possible hazards in diagnostic ultrasound. Br. J. Hosp. Med., 8, 571-577.
17. Dyson, M., Pond, J. and Woodward, B. (1972) The induction of red cell stasis in embryos by ultrasound. In: "Interaction of ultrasound and biological tissues: workshop proceedings", edit. J.M. Reid and M.R. Sikov, DHEW Publication(FDA) 73-8008,139-141.
18. Dyson, M. and Pond, J. (1973) The effects of ultrasound on circulation. Physiotherapy, 59, 284-287.
19. Dyson, M. and Pond, J. (1973) Biological effects of therapeutic ultrasound. Rheum. Rehab., 12, 209-212.
20. Dyson, M., Pond, J., Woodward, B. and Broadbent, J. (1974). The production of blood cell stasis and endothelial damage in the blood vessels of chick embryos treated with ultrasound in a stationary wave field. Ultrasound Med. Biol., 1, 133-148.
    *This paper was selected for publication as a Benchmark paper in "Acoustics in Ultrasonic Biophysics", edit. F. Dunn and W.D. O'Brien Jr., Hutchinson and Ross,Inc., Stroudsburg, Pennsylvania, 1976.
21. Taylor, K., and Dyson, M. (1974) Toxicity studies on the interaction of ultrasound on embryonic and adult tissues. In: "Proceedings of the Second World Conference on Ultrasound in Medicine", edit. M. de Vleiger, D.M. White and V.R. McReady, Excerpta Medica, Amsterdam, pp.353-359.

22. Harvey, W., Dyson, M., Pond, J. and Grahame, R. (1975) The *in vitro* stimulation of protein synthesis in human fibroblasts by therapeutic levels of ultrasound. In: "Proceedings of the Second European Congress on Ultrasonics in Medicine", Excerpta Medica International Congress Series, No.363, pp.10-21.
23. Dyson, M., Franks, C. and Suckling, J. (1976) Stimulation of healing of varicose ulcers by ultrasound. Ultrasonics, 14, 232-236.
24. Dyson, M. and Suckling, J. (1978) Stimulation of tissue repair by ultrasound: a survey of the mechanisms involved. Physiotherapy, 64, 105-108.
25. ter Haar, G., Dyson, M. and Talbert, D. (1978) Ultrasonically induced contraction of mouse uterine smooth muscle *in vivo*. Ultrasonics, 16, 275-276.
26. Webster, D., Harvey, W., Dyson, M. and Pond, J. (1978) The role of ultrasound-induced cavitation in the *in vitro* stimulation of protein synthesis in human fibroblasts by ultrasound. Ultrasound Med. Biol. 4, 343-351.
27. Webster, D., Harvey, W. and Dyson, M. (1979) Ultrasonically-induced stimulation of collagen synthesis *in vivo*. In: "Proceedings of the Fourth European Symposium on Ultrasound in Biology and Medicine", edit. P.Greguss, Vol.1, pp.135-140.
28. Dyson, M., Webster, D.F., Pell, R. and Crowder, M. (1979) Improvement in the mechanical properties of scar tissue following treatment with therapeutic levels of ultrasound *in vivo*. In: Proceedings of the Fourth European Symposium on Ultrasound in Biology and Medicine, edit. P. Greguss, Vol.1,pp.129-134.
29. ter Haar, G., Dyson, M. and Smith, S. (1979) Ultrastructural changes in mouse uterine blood vessels brought about by ultrasonic irradiation at therapeutic intensities in standing wave fields. Ultrasound Med. Biol., 5, 167-179.
30. Webster, D.F., Harvey, W., Dyson, M. and Pond, J. (1980) The role of ultrasound-induced cavitation in the *in vitro* stimulation of collagen synthesis in human fibroblasts. Ultrasonics, 16, 33-37.
31. Gibson, T., Laurent, R., Highton, J., Wilton, M., Dyson, M. and Millis, R. (1981) Synovial histopathology of Behcet's syndrome. Ann. Rheum. Dis., 40, 376-381.
32. Dyson, M. (1980) The effect of ultrasound on the rate of wound healing and the quality of scar tissue. In: "Proceedings of the International Symposium on Therapeutic Ultrasound. Manitoba", edit. A. Mortimer and N. Lee, Canadian Physiotherapy Association, Winnipeg, pp.110-123.
33. Dyson, M. (1982) Non-thermal cellular effects of ultrasound. Br. J. Cancer,35,suppl.V, 165-171.
34. Dyson, M. (1982) Stimulation of tissue repair by therapeutic ultrasound. Infections Surg., 1, 37-44.
35. Dyson, M. and Brookes, M. (1983) Stimulation of bone repair by ultrasound. In: "Ultrasound '82", edit. R.A. Lerski and P. Morley, Pergamon Press,Ltd., Oxford, pp.61-66.
36. Grahame, R., Armstrong, R., Simons, N., Wilton, M., Dyson, M., Laurent, R., Millis, R. and Mims, C.A. (1983) Chronic arthritis associated with the presence of intra-synovial *Rubella* virus.
37. Dyson, M. and Smalley, D. (1983) Effects of ultrasound on wound contraction. In: "Ultrasound Interactions in Biology and Medicine", edit. R. Millner and U. Corbet, Plenum Publishing Corporation, New York, pp.151-158.
38. Dyson, M. (1984) Biological and therapeutical effects of ultrasound. In: "Proceedings of the Centennial Congress of the Italian Surgical Society. Postgraduate Course on Emerging Technologies in Surgery", edit. L. Angelini, F. Fegiz and P.N.T. Wells, Masson Italia Editori, Milan, pp.61-71.
39. Dyson, M. (1984) The effect of ultrasound on tissue repair. Scripta Medica (Praha), 57, 212-213.

40. Dyson, M. (1985) Therapeutic applications of ultrasound. Clinics Diagnost. Ultrasound, 16, 121-134.
41. Gibson, T., Fagg, N., Highton, J., Wilton, M. and Dyson, M. (1985) The diagnostic value of synovial biopsy in patients with arthritis of unknown cause. Br. J. Rheum. 24, 232-241.
42. Dyson, M. and Luke, D.A. (1986) Induction of mast cell degranulation in skin by ultrasound. IEEE Trans. Ultrasonics, Ferroelectics and Frequency Control, URRC-33 (2), 194-201.
43. Dyson, M. and Young, S. (1986) The effects of laser therapy on wound contraction and cellularity. Lasers. Med. Sci., 1, 125-130.
44. Dyson, M (1986) A review of recent experimental evidence on the effects of diagnostic ultrasound on tissue. Inst. Phys. Sci. Med., Conference Report Series, 47, 1-11.
45. Crum, L.A., Daniels, S., Dyson, M., ter Haar, G.R. and Walton, A.J. (1986) Acoustic cavitation and medical ultrasound. Proc. Inst. Acoust.,8(2), 137-146.
46. Dyson, M. (1986) The effect of laser therapy on wound contraction. Med. Laser Reports, 4, 2-8.
47. Hodgson, S., Child, A. and Dyson, M. (1987) Endocardial fibro-elastosis: possible X-linked inheritance. J. Med. Genet., 24, 210-214.
48. Coleman, A.J., Saunders, J.E., Crum, L.A and Dyson, M. (1987) Acoustic cavitation generated by an extra-corporeal shockwave lithotriper. Ultrasound Med. Biol., 13(2), 69-76.
49. Dyson, M. (1987) Review of recent experimental evidence on the effects of diagnostic ultrasound on tissue. In: "Obstetric and Neonatal Blood Flow", edit. C.D. Sheldon, D.H. Evans and J.R. Savage. Conference Proceedings 2, Biological Engineering Society, pp.1-7.
50. Dyson, M. (1987) Mechanisms of therapeutic ultrasound. Physiotherapy, 73, 116-120.
51. Daniels, S., Blondel, D., Crum, L.A., ter Haar, G.R. and Dyson, M. (1987) Ultrasonically induced gas bubble production in agar based gels: Part I, Experimental Investigation. Ultrasound Med. Biol., 13(9), 557-539.
52. Crum, L.A., ter Haar, G.R., Daniels, S. and Dyson, M. (1987) Ultrasonically induced gas bubble production in agar based gels: Part II, Theoretical analysis. Ultrasound Med. Biol., 13(9), 541-554.
53. Crum, L.A., Walton, A.J., Mortimer,A., Dyson, M., Crawford,D. and Gaitan, D.F. (1987) Free radical production in amniotic fluid and blood plasma by medical ultrasound. J. Ultrasound. Med., 6, 643-647.
54. Lovell, C.R., Smolenski, K.A., Duance,V.C., Light, N.D., Young, S. and Dyson, M. (1987) A study of Type I and III collagen content and fibre distribution in normal human skin during ageing. Br. J. Dermatol., 117, 419-428.
55. ter Haar, G., Dyson, M. and Oakley, E.M. (1987) The use of therapeutic ultrasound by physiotherapists in Britain:1985. Ultrasound Med. Biol., 13, 659-663.
56. Dyson, M. (1987) Why membrane changes matter. Euroson '87, edit. S.Bondesram, A. Alanen and P.Jouppila. Finnish Society for Ultrasound in Medicine and Biology, p.397.
57. ter Haar, G., Dyson, M. and Oakley, E.M. (1988) Ultrasound in Physiotherapy in Britain: the results of a questionnaire. Physiotherapy Practice, 4, 69-72.
58. Mortimer, A.J. and Dyson, M. (1988) The effect of therapeutic ultrasound on calcium uptake in fibroblasts. Ultrasound Med. Biol., 14, 499-506.
59. Dyson, M., Young, S., Pendle, C.L., Webster, D.F. and Lang, S.M. (1988) Comparison of the effects of moist and dry conditions on tissue repair. J. Invest. Dermatol., 91, 434-439.
60. Dyson, M. (1989) The use of ultrasound in sports physiotherapy. In: "International Perspectives on Physical Therapy". Series editors: I. Bromley and N.T. Watts; Vol. 4 "Sports Injuries", edit. V.Grisogono, Churchill-Livingstone:Edinburgh, pp.213-232.
61. Dinno, M.A., Dyson, M., Young, S.R., Mortimer, A.J., Hart, J. and Crum, L.A. (1989) The significance of membrane changes in the safe and effective use of therapeutic and diagnostic ultrasound. Phys. Med. Biol., 43, 1543-1552.

62. Young, S., Bolton, P., Dyson, M., Harvey, W. and Diamantolpoulos, C. (1989) Macrophage responsiveness to light therapy. Lasers Surg. Med., 9, 497-505.
63. Young, S.R. and Dyson, M. (1989) The effect of therapeutic ultrasound on angiogenesis. Ultrasound Med. Biol. 16, 261-269.
64. Young, S.R. and Dyson, M. (1990) Effect of therapeutic ultrasound on the healing of full-thickness excised lesions. Ultrasonics, 28,175-180.
65. Dyson, M., (1990) Role of ultrasound in wound healing. In: Contemporary Perspectives in Rehabilitation. Editor-in-Chief: S. Wolf. "Wound Healing: Alternatives in Management", 1st edition. Volume editors: L.C. Kloth, J. Feedar and J. McCullough, F.A.Davis Company, Philadelphia, pp.229-285.
66. Young, S.R. and Dyson, M. (1990) Macrophage responsiveness to therapeutic ultrasound. Ultrasound Med. Biol., 16, 809-816.
67. El-Sayed, S. and Dyson, M. (1990) A comparison of the effect of multiwave-length light produced by a cluster of semiconductor diodes and each individual diode on mast cell number and degranulation in intact and injured skin. Lasers Surg. Med., 10, 559-568.
68. Young, S.R., Dyson, M. and Bolton, P.A. (1990) The effect of light on calcium uptake in macrophages. Laser Therapy, 2, 53-57.
69. Bolton, P.A., Young, S.R. and Dyson, M. (1990) Macrophage responsiveness to light therapy- a dose reponse study. Laser Therapy, 2, 101-106.
70. Dyson, M. (1990) Electrotherapy: the need for critical evaluation and continuing education. Physiotherapy Theory and Practice, 6, 105.
71. Young, S.R. and Dyson, M. (1991) Het effect van ultrageluid en licht op het genezen van weefsels. Ned. T. Fysiotherapie, 101, 20-23.
72. Al Hassan, J., Dyson, M., Young, S.R., Thompson, M. and Criddle, R.S. (1991) Acceleration of wound healing responses induced by preparations from the epidermal secretions of the Arabian Gulf Catfish (*Arius lineatus*, Valenciennes). J. Wilderness Med.,2, 153-163.
73. Young, S.R., Dyson, M., Hickman, R., Lang, S.M., and Osborne, C. (1991) Comparison of the effects of semi-occlusive polyurethane dressings and hydrocolloid dressings on dermal repair - 1: Cellular changes. J. Invest. Dermatol., 97, 586-592.
74. Young, S.R., Dyson, M. and Bolton, P. (1991) Can the treatment of intact skin, prior to surgery, with light therapy have an effect on the rate of repair? Proceedings of the 8th Congress of the International Society for Laser Surgery and Medicine, pp.103-108.
75. Bolton, P.A., Young, S.R. and Dyson, M. (1991) Macrophage responsiveness to light therapy with varying power and energy densities. Laser Therapy, 3, 105-112.
76. Bolton, P.A., Young, S.R. and Dyson, M. (1991) The effect of broad band wavelength light on mediator release from macrophages. Laser Therapy, 4, 25-30.
77. Dyson, M. (1992) Cellular and subcellular aspects of low level laser therapy. In: "Progress in Laser Therapy: Selected papers from the 1990 ITLA Congress", edit. T. Ohshiro and R.G. Calderhead. John Wiley, Chichester.
78. Ghali, L and Dyson, M. (1992) The direct effect of light therapy on endothelial cell proliferation *in vitro*. In: "Angiogenesis: Key Principles - Science - Technology - Medicine", edit. R. Steiner, P.B. Weisz and R. Langer. Birkhauser Verlag, Basel, pp.411-414.
79. Bolton, P.A., Dyson, M. and Young, S.R. (1992) The effect of polarised light on the release of growth factors from the U937 macrophage-like cell line. Laser Therapy, 4, 33-37.
80. Cheetham, M., Young, S.R. and Dyson, M. (1992) Histological effects of 820 nm laser irradiation on the healthy growth plate of the rat. Laser Therapy, 4, 59-64.
81. Whiston, R.J., Young, S.R., Lynch, J.A., Harding, K.G. and Dyson, M. Application of high frequency ultrasound to the objective assessment of healing wounds. Proc. 2nd Conference on Advances in Wound Management. Macmillan Press, London, pp.26-29.

82. Young, S.R., Lynch, J.A., Liepins, P.J. (1992) Ultrasound imaging: a non-invasive method of wound assessment. Proc. 2nd Conference on Advances in Wound Management. Macmillan Press, London, pp.26-29.
83. Dyson, M., Young, S.R., Hart, J., Lynch, J.A. and Lang, S.M. (1993) Comparison of the effect of moist and dry conditions on the process of angiogenesis during dermal repair. J. Invest. Dermatol., 99, 729-733.
84. Dyson, M. (1993) Ultrasound, lasers and soft tissue repair. J. Assoc. Chart. Physiother. Obst. Gyn., 72, 2.3
85. El Sayed, S.O. and Dyson, M. (1993) Responses of dermal mast cells to injury. J. Anat., 182, 369-376.
86. El Sayed, S.O. and Dyson, M. (1993) Histochemical heterogeneity of mast cells in rat skin. Biotech. Histochem., 68, 326-332.
87. Steinlechner, C.W.B. and Dyson, M. (1993) The effects of low-level laser therapt on macrophage-modified keratinocyte proliferation. Laser Therapy, 5, 65-73.
88. Young, S.R. and Dyson, M. (1993) The effect of light on tissue repair. Acupuncture in Medicine, 11, 17-20.
89. Dyson, M. (1993) The effect of ultrasound on the biology of soft tissue repair. In: "The soft tissues - trauma and sports injuries", edit. G.R. McLatchie and C.M.E. Lennox. Butterworth-Heinemann Ltd., Oxford, pp.200-212.
90. Young, S.R. and Dyson, M. (1993) The effect of ultrasound and light therapy on tissue repair. In "Intermittent high intensity exercise", edit. D.A.D. Macleod, R.J. Maughan, C. Williams, C.R. Madeley, J.C.M. Sharp and R.W. Nutton. Chapman and Hall, London, pp. 321-328.
91. Karim, A., Young, S.R., Lynch, J.A. and Dyson, M. (1994) A novel method of assessing skin ultrasound scans. Wounds, 6, 9-15.
92. Cervi, P., Murdock, A., Rees, D., Garner, S., Grant, D., Wright, S. and Dyson, M. (1994) Splenic Ultrasound - a new wave for immune thrombocytopenic purpura. J. Clin. Path., 47, 414-417.
93. Dyson, M. (1994) Electrotherapy: an overview. Br. J. Ther. Rehab., 1, 136-140.
94. Rajaratnam, S., Bolton,P. and Dyson, M. (1994) Macrophage responsiveness to laser therapy with varying pulsing frequencies. Laser Therapy, 6, 107-112.
95. Bolton, P., Young, S. and Dyson, M. (1995) The direct effect of 860 nm light on cell proliferation and on succinic dehydrogenase activity of human fibroblasts *in vitro*. Laser Therapy, 7, 55-60.
96. Dyson, M. (1995) Ultrasound for wound management. In: "Clinical Wound Management,", edit. P.P. Gogia, Slack, Inc. New Jersey, pp.197-204.
97. Kitchen,S. and Dyson, M. (1996) Low energy treatments: nonthermal or microthermal? In: "Clayton's Electrotherapy" 10th Edition, edit. S. Kitchen and S. Bazin, W.B. Saunders Company Ltd., London, pp. 110-115.
98. Dyson, M. (1996) Die Rolle von interaktiven Wundverbanden in de Wundheilung. In: "Wundheilung und Wundauflagen", edit. K.M. Sedlarik and H. Lippert, Wissenschaftliche Verlagsgesellschaft mbH. Stuttgart.
99. El Sayed, S. and Dyson, M. (1996) Effect of laser pulse repetition rate and pulse duration on mast cell number and degranulation. Lasers Surg. Med. 19, 433-437.
100. Young, S.R., Erian, A., and Dyson, M. (1996). High frequency diagnostic ultrasound: a noninvasive, quantitative aid for testing the efficacy of moisturizers. Internat. J. Aesthetic Restor. Surg., 4(2), 1.5.
101. Davies, J.R., Dyson, M., Mustafa, Y., Compton, F., and Perry, M. (1996) The ontogeny of adhesion molecules expressed on the vascular endothelium of the developing human skin. J. Anat., 189, 373-382.

102. Nicolopoulos, N., Cavadias, C., Karameris, A., Dyson, M, Tsourouktsoglou, Kondoyannis, P. (1996) The use of laser surgery in subtotal meniscectomy and the effect of low-level laser therapy on the healing potential of rabbit meniscus: an experimental study. Lasers Med. Sci., 11, 109-115.
103. Calvin, M., Modari, B., Young, S.R., Koffman, G. and Dyson, M. (1997) Pilot study using high-frequency diagnostic ultrasound to assess surgical wounds in renal transplant patients. Skin Res. Technol., 3, 60-65.
104. Dyson, M. (1997) Advances in wound healing physiology: the comparative perspective. Veterinary Dermatol., 8, 227-233.
105. Sussman, C. and Dyson, M. (1998) Therapeutic and Diagnostic Ultrasound. In: "Wound Care: A Collaborative Manual for Physical Therapists and Nurses", edit. C. Sussman and B.M. Bates-Jensen, Aspen Publishers, Inc., Gaithersburg, pp.427-446.
106. Webb, C., Dyson, M. and Lewis, W.H.P. (1998) Stimulatory effect of 660 nm low level laser energy on hypertrophic scar-derived fibroblasts: possible mechanisms for increase in cell counts. Lasers Surg. Med. 22, 294-301.
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