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Malcolm R. Hooper

OXYMED Australia

Hyperbaric Oxygen Therapy
Elite Sports Recovery & Performance

OXYMED

MALCOLM R. HOOPER

- Dr Malcolm Hooper

  B.App Sci 1984, D.Acup 1985, Grad Cert 1993, Grad Dip 1995, M.App Sci 1999.

  Clinic Director - OXYMED Australia

- Author

  - *Hyperbaric Medicine: The Life is in the Blood (2005, 2018 reprint)*

  - *Hyperbaric Oxygen Therapy combined with LOKOMAT (Robotic Gait Assisted Walking) assisting Neuroplasticity in Brain and Spinal Cord Injury (forthcoming October 2018)*

  - *Hyperbaric Oxygen Therapy and Cytokine Modulation (February 2019)*

- Founder Australia's first LOKOMAT – and world first combing Hyperbaric Oxygen Therapy with LOKOMAT (2006).
- International Executive Director

  Vice President, International Hyperbaric Medical Foundation (IHMF),

  Chair International Relations, International Hyperbaric Medical Association (IHMA).

- No Disclosure or Caveats

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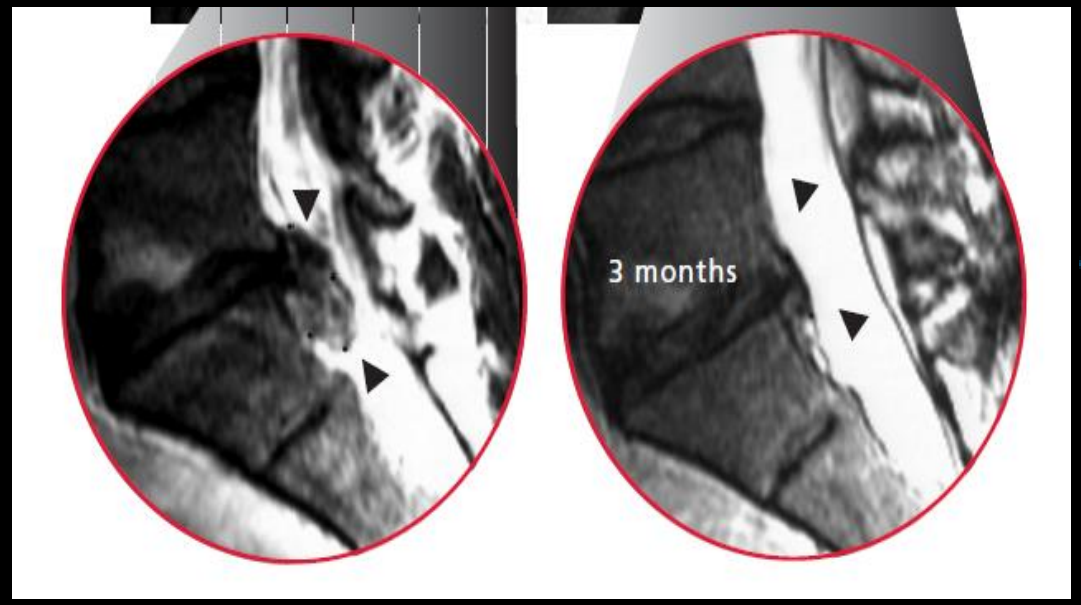
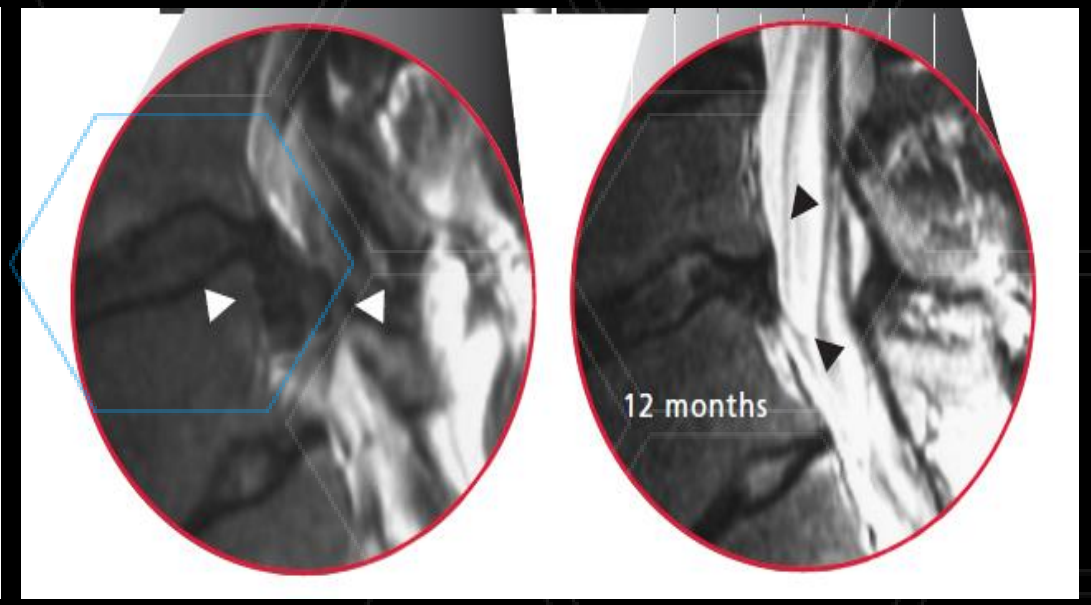
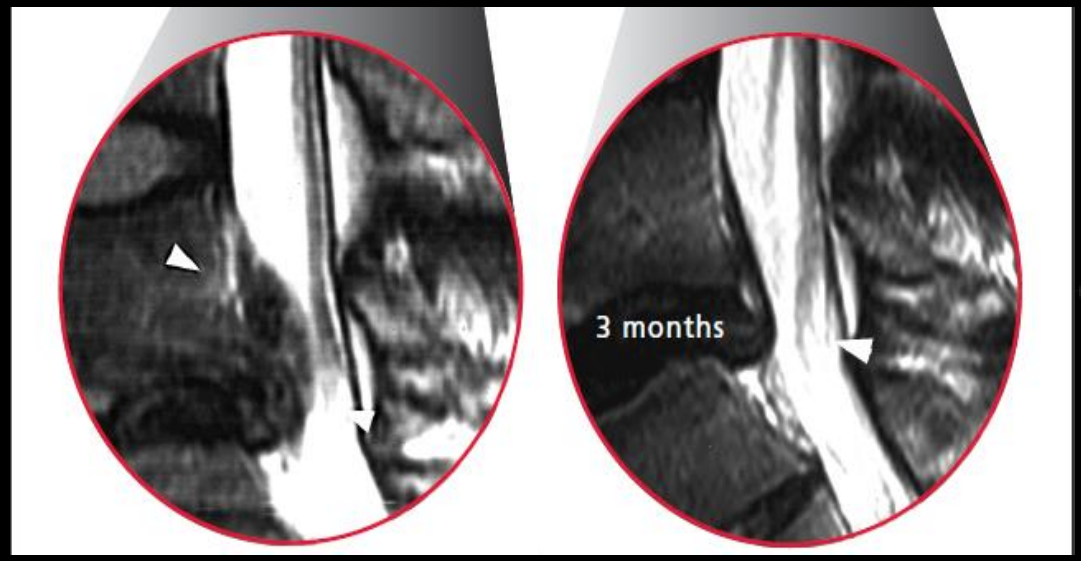
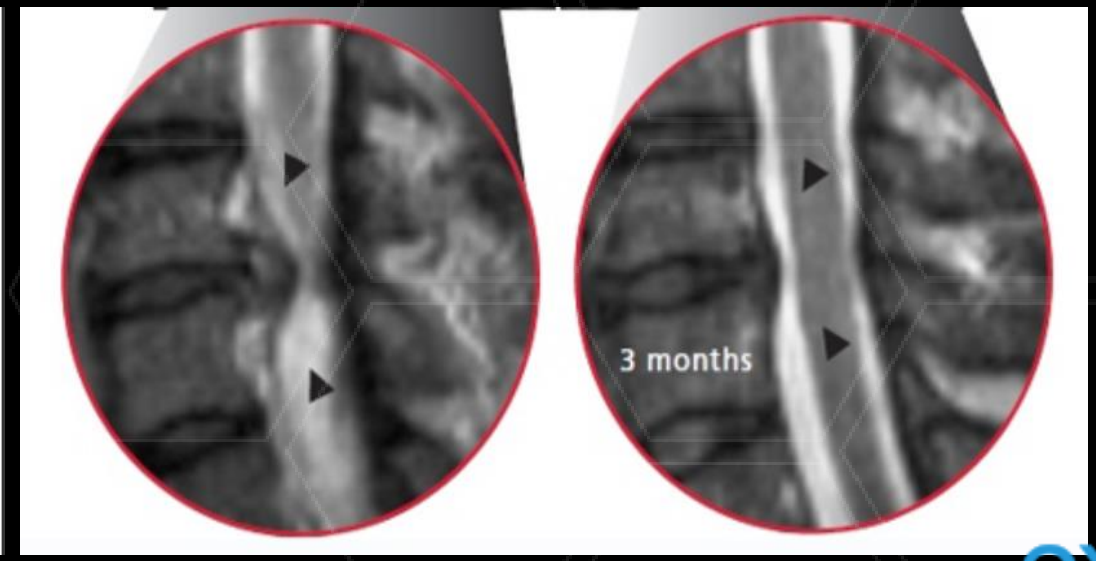
1995 - 1999: Chronic Pain, Disc Prolapse, Failed Back Surgery









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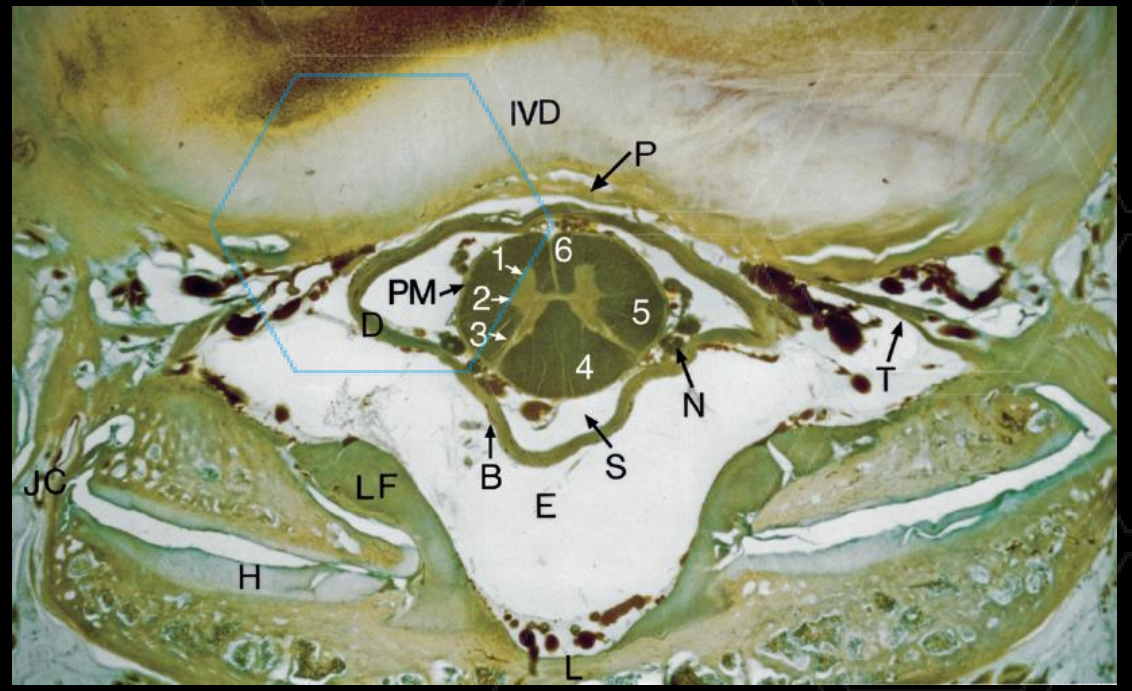
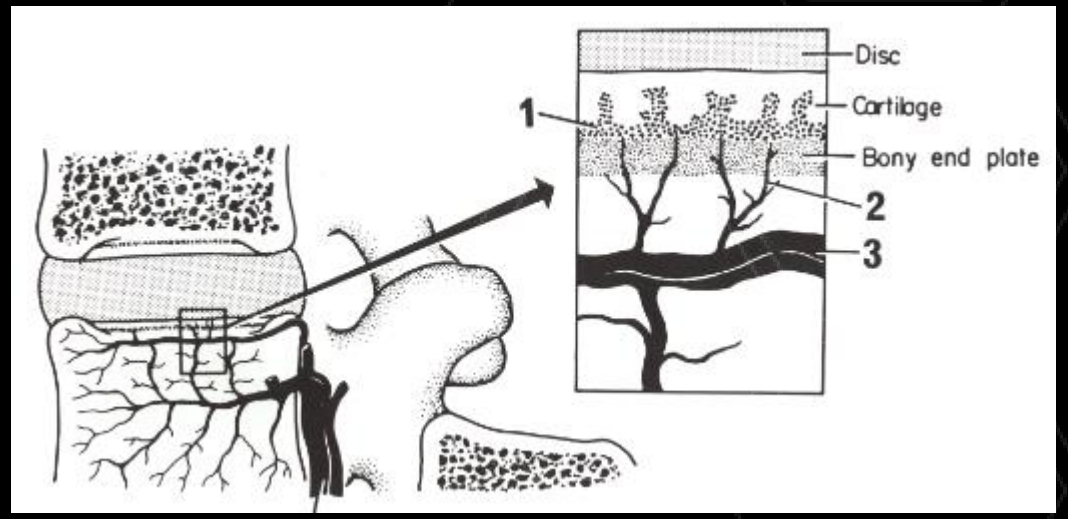
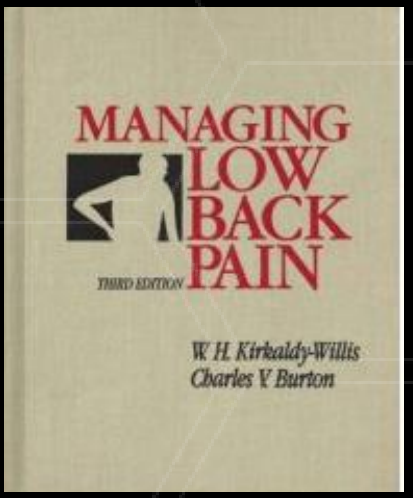
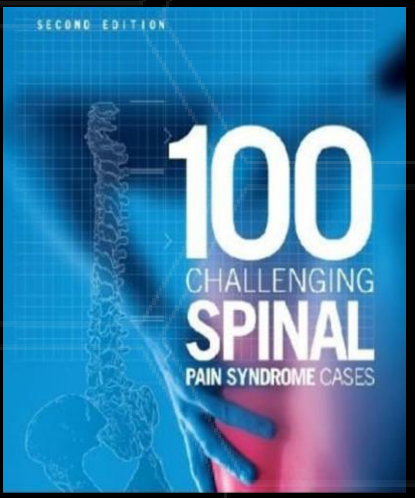
Ischemic Model of Degeneration (Kirkaldy Willis 1986, L. Giles 2nd Ed)









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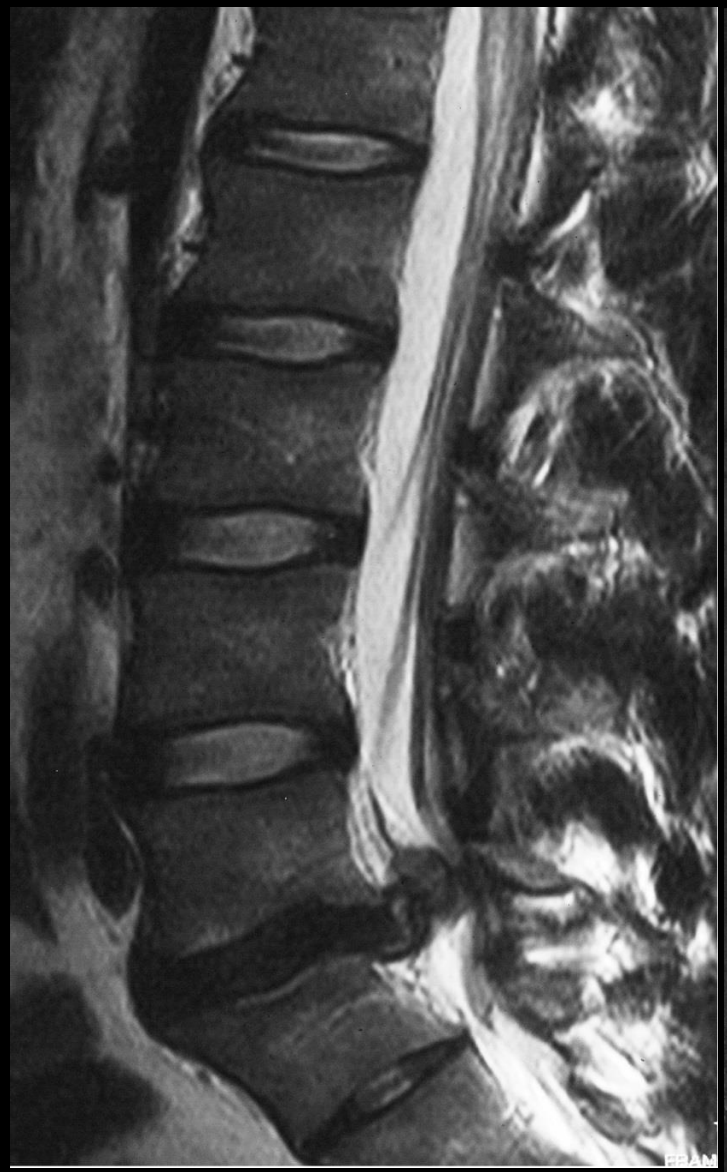
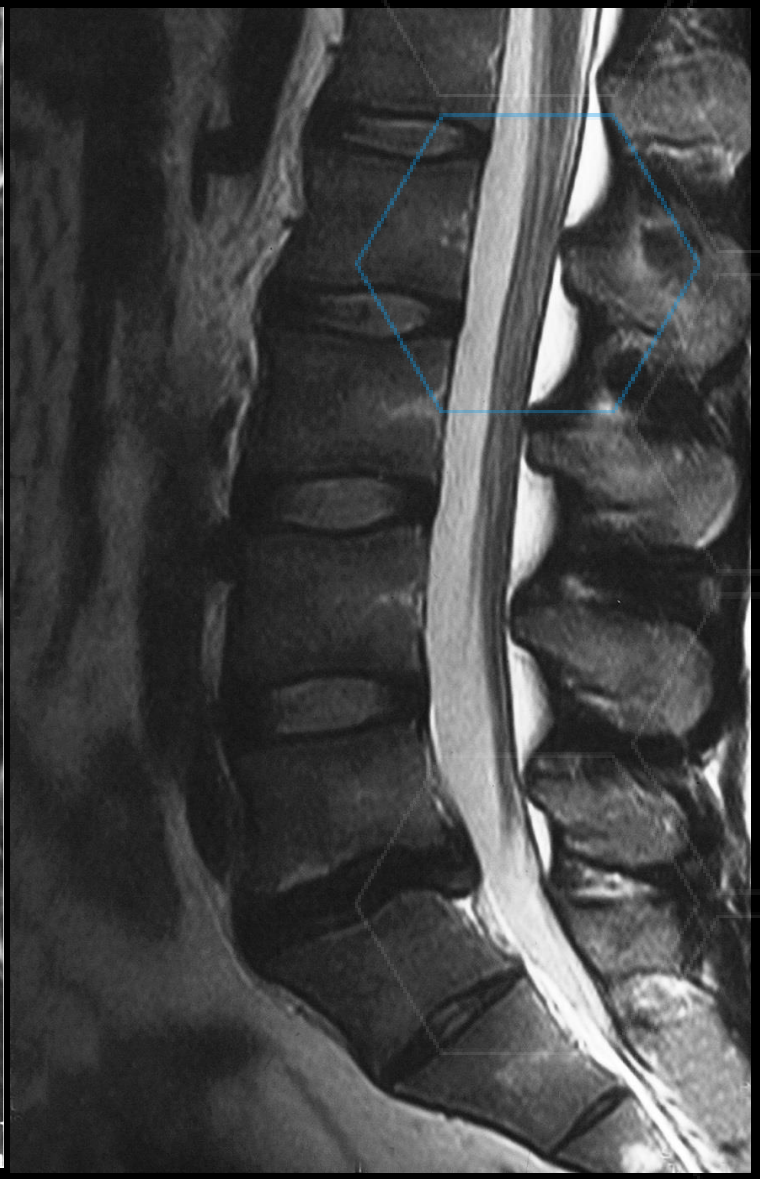
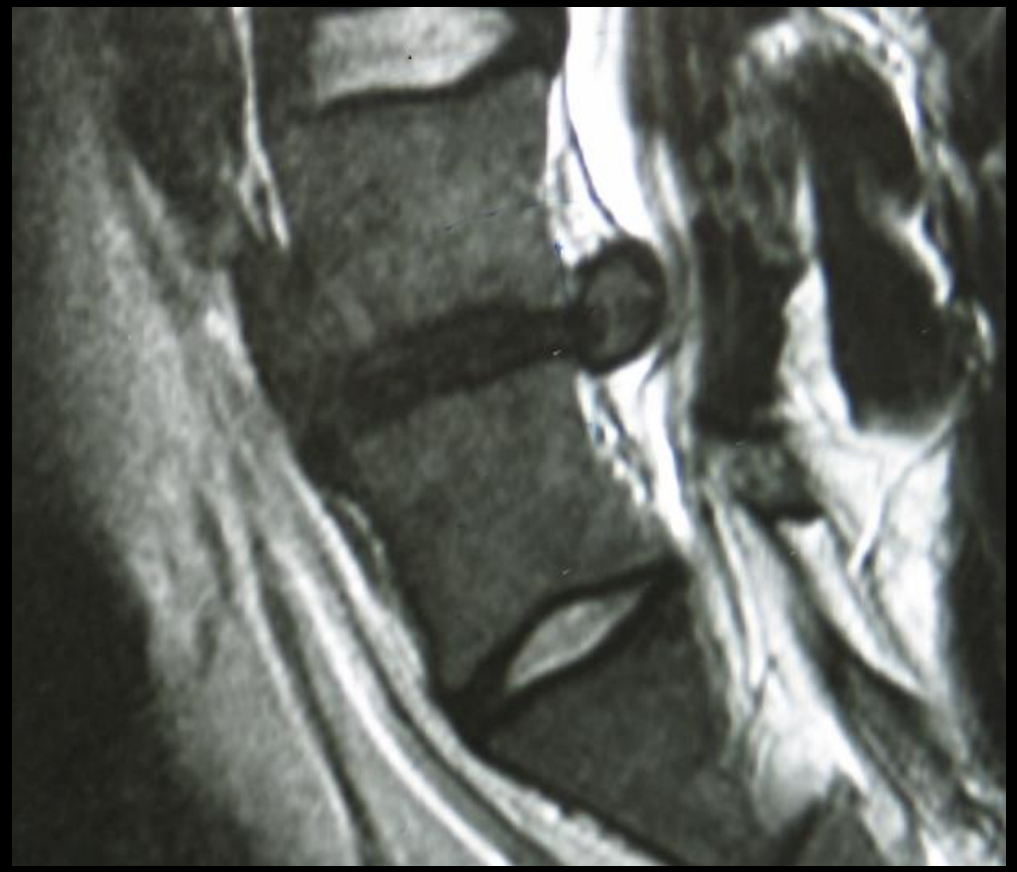
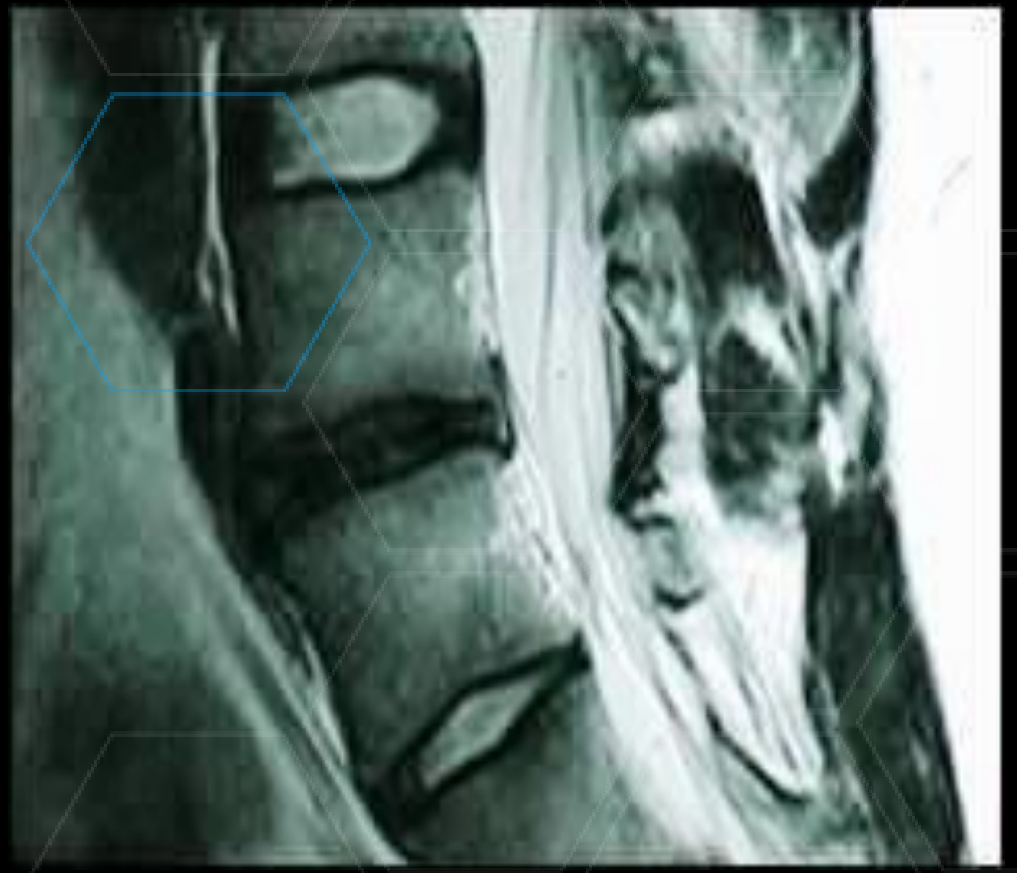
Disc Sequestration





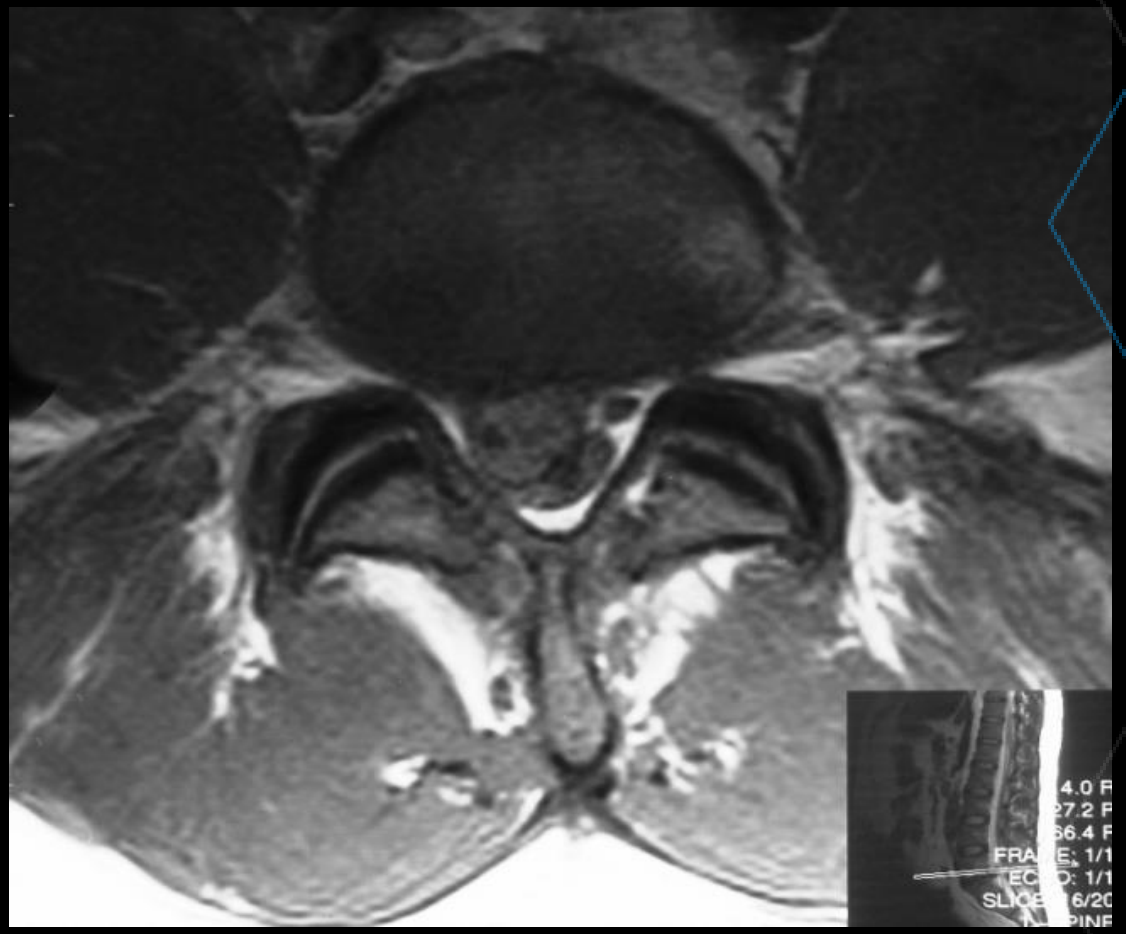
Disc Prolapse, Failed Back Surgery

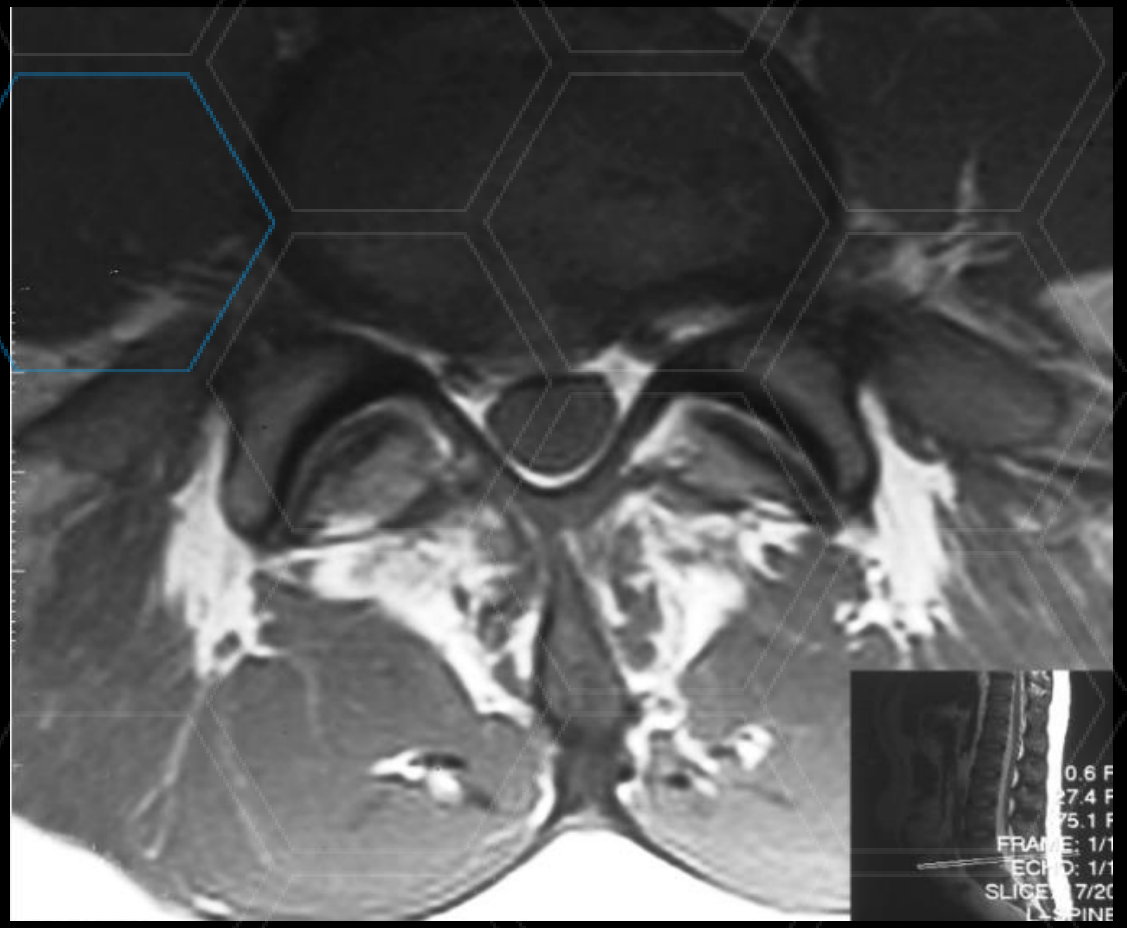




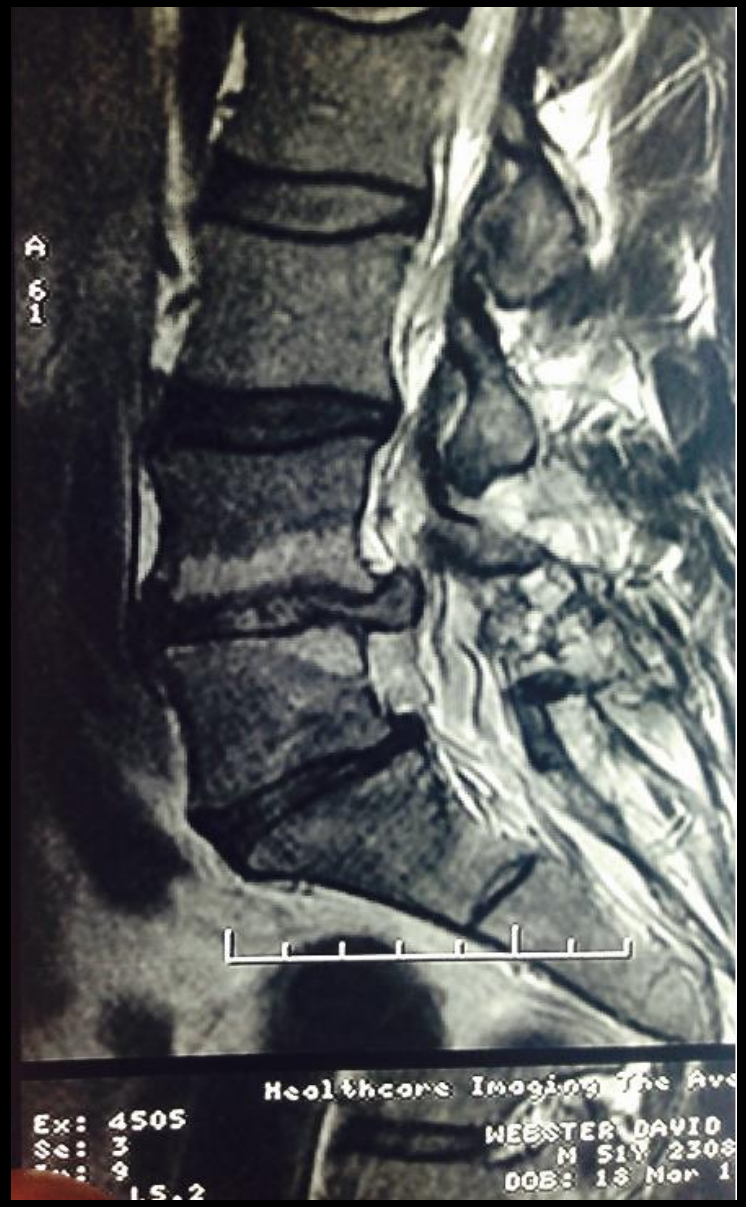
OXYMED

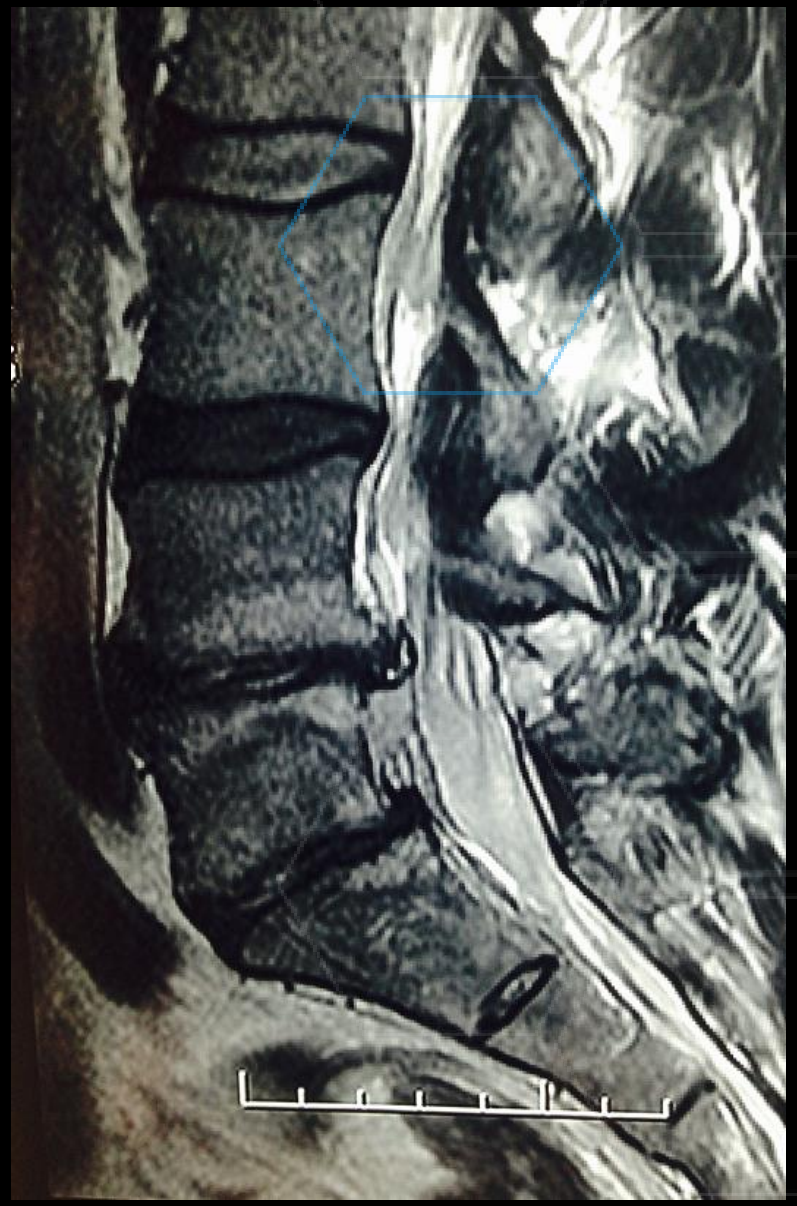
Disc Prolapse, Failed Back Surgery





Disc Prolapse, Failed Back Surgery





2011 Serbia

2008, 2011, 2012, 2013, 2015, 2016

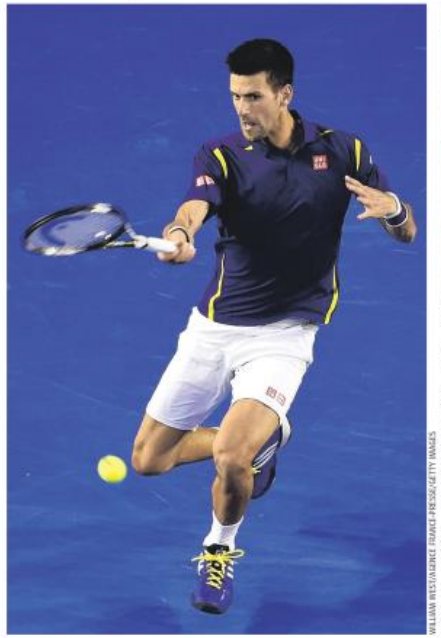
Wall Street Journal 2016

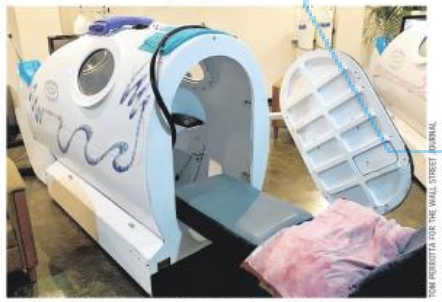
SPORTS

Tennis Players Get an Oxygen Fix

To aid recovery, tennis pros in Melbourne rent hyperbaric chambers

BY TOM PERROTTA





TOM PERROTTA FOR THE WALL STREET JOURNAL

Left, Novak Djokovic uses hyperbaric oxygen therapy to help with recovery. Above, a pod at the HyperMED clinic in Melbourne, Australia.

Melbourne, Australia

AFTER HE WON a four-hour, 32-minute match at the Australian Open on Sunday, Novak Djokovic, the world's best tennis player, showered, spoke to the media, and then hustled over to a quiet late-night spot that's popular among several players: A small clinic two miles from the tournament that has four hyperbaric oxygen pods for rent.

The machines look like deep-sea diving submarines with glass windows and hatches in the back. Inside, players slip on a plastic breathing mask attached to a long tube. Once the hatch seals they're off, compressor and regulator whirring and pressure building until they're basically 40-feet under water and breathing 100% oxygen through the mask (outside air contains just 21% oxygen).

Players have their own masks and tubes and don't share them (that's bad hygiene). They wear blue hospital booties over their socks and must leave behind their phones, watches or anything with a battery that could spark a fire. Outside each pod's front window is a television equipped with Netflix. American Bethanie Mattek-Sands binged on episodes of "Making a Murderer" before leaving Melbourne on Thursday after losing in mixed doubles. Djokovic spent an hour in the pod after his five-setter against Gilles Simon on Sunday, from around 10 p.m. to 11 p.m. He'll use it before matches too, as he did for an hour Tuesday afternoon before playing Kei Nishikori later that evening. Djokovic watched M. Night Shyamalan's "The Last Airbender."

"I either like comedy or, somebody calls it science fiction," he said. "I call it the world we still didn't explore."

The pods are located at HyperMED, a ground-floor clinic nestled between a hair salon and a bakery about a 10-minute drive from the Open. A few hotels popular among players are nearby. Djokovic, known as the most meticulous player on the tour, has used the pods here for several years with the hope of aiding recovery and preventing injury. Mattek-Sands started last year. This year, doubles star Mike Bryan became a pod regular. His twin brother, Bob, tried it too, though just once.

"It's great," Djokovic said. "It should get out there more, not just for athletes."

Mike Bryan said: "It just helps recovery. I felt a little better doing it."

Bryan also likes the VacuSport, a long tube with a skirt that seals a player's legs in a vacuum and flushes lactic acid. There is also a cryotherapy chamber, which cools to minus 150 degrees Fahrenheit for a few minutes. HyperMED's website has a picture of Milos Raonic, the Canadian star, standing in its chamber.

Oxygen sessions last anywhere from an hour to two hours and cost 150 Australian dollars (US$105). Mattek-Sands said the benefits are subtle but valuable. "It's not like you walk out of there feeling like Superman or anything," she said. "You sleep pretty good that night, I'll say that. You crash and you dream pretty heavy."

The facility is run by Malcolm Hooper, a former chiropractor, who sat in Djokovic's box in Rod Laver Arena Thursday evening as the world No. 1 beat Roger Federer and earned a spot in Sunday's Australian Open final, the sixth of his career. Hooper's clients include people with cerebral palsy, traumatic brain injuries and disabilities, as well as other athletes. During the Open, he opens his clinic day and night depending on players' needs. "Two, three in the morning, whatever the requirements are," he said.

Top athletes around the world, including football, basketball and soccer stars, use hyperbaric oxygen therapy. Djokovic said he only uses hyperbaric pods in the U.S. and Australia, because access and regulations are challenging in Europe. He said there is still a stigma about oxygen therapy, that it gives athletes who use it an unfair advantage.

"It's very sensitive, especially in the European part of the world," he said. "I wish I can have this all over the place, I wish."

Mattek-Sands said she would be thrilled if the sport's major tournaments provided pods on site. "It's just kind of the new wave for the future," she said.

Hyperbaric therapy doesn't suit everyone. Andy Murray has tried it and decided it isn't for him, according to a representative. Some question its merits. A paper published last year in PeerJ, a peer-reviewed journal, found lower lung cancer incidence among people living at high elevations, which suggests oxygen could be a driver of cancer. Kamen Simeonov, an MD-PhD trainee at the University of Pennsylvania and co-author of the paper, wrote via email, "Basically, it's exposing yourself to risks with no logical reward."

Hooper, who is an affiliate member of the International Hyperbaric Medical Association, said he has seen gains in his patients, and that research suggests hyperbaric treatment can help many ailments. "Every athlete has a growing list of injuries that may benefit," he wrote in an email.

Hooper's chiropractic license was suspended for two years in 2013 after a dispute with a former cerebral palsy patient in part over the effectiveness of treatment, though the Chiropractic Board of Australia viewed Hooper's "conduct as an error of judgment rather than a defect in character," and that he was a "true believer in the treatment that was being given." (He said he hasn't practiced as a chiropractor for 20 years and no longer has a need for the license.)

Hooper also was treating Aussie Rules football players in 2013 when they separately came under investigation for the possible use of banned substances. A Court of Arbitration for Sport panel that imposed a two-year ban on 34 players earlier this month made no mention of Hooper in its findings and didn't assign him any fault. Hooper says he tells all his clients about his history. Justin Sands, the husband of Mattek-Sands, said Hooper has been open since they first met.

"He's been nothing but an upstanding, good guy," Sands said.

Sands, who played college football in the U.S., said he used hyperbaric therapy in his playing days. He says he's surprised so few tennis players use it, given the grueling demands of the game. It might even be useful, he said, for the tour's traveling husbands and wives, though for other reasons. "It's great for a hangover," he said.

NEWS

NOVAK DJOKOVIC: 'HYPERBARIC CHAMBER IS VERY GOOD!'

Gatto Luigi - 14-01-2017 - View: 4290

Tennis - The Serbian player admits he is still using the renewed electronic system that speeds up the recovery After long matches

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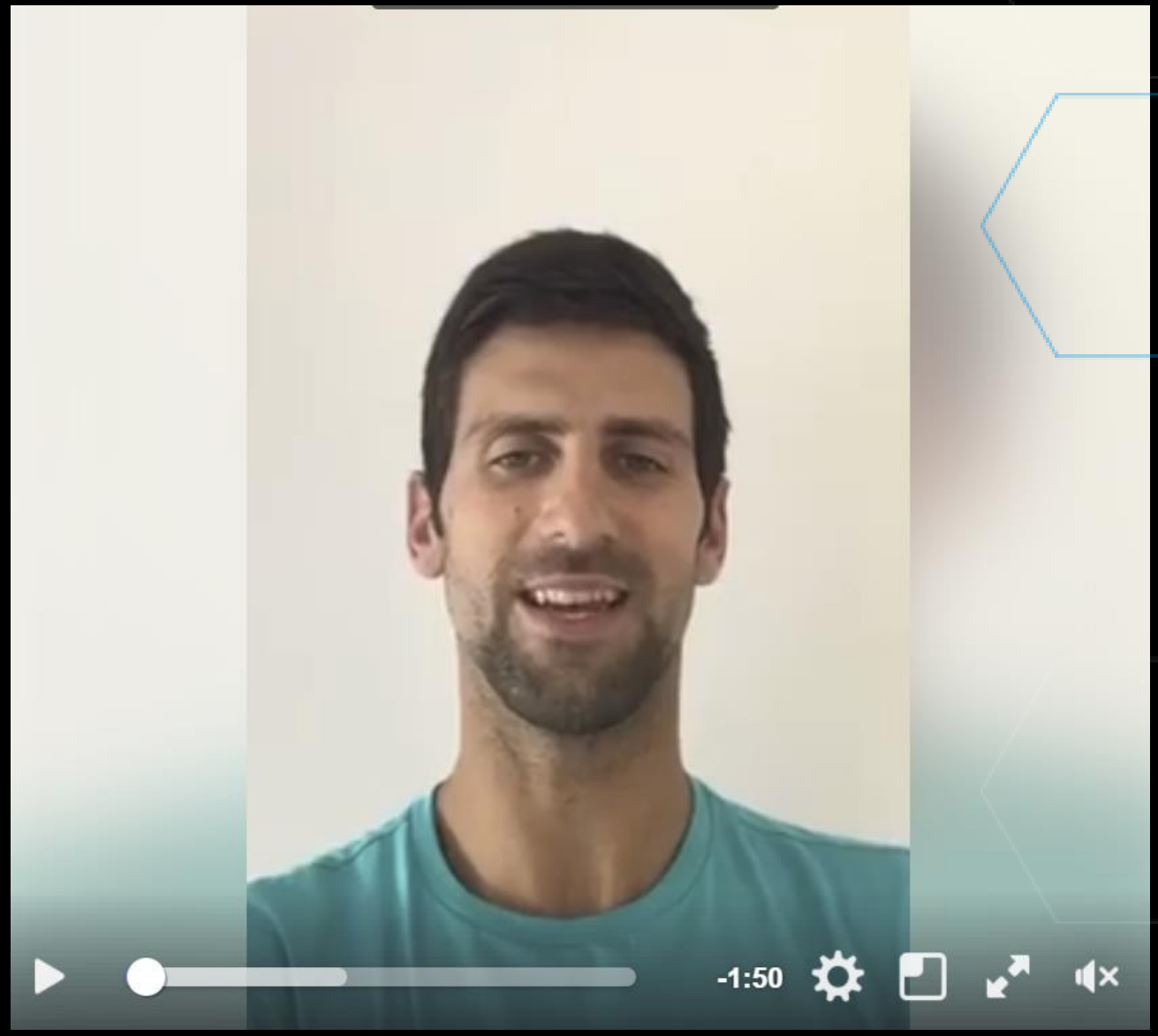
HBOT2017 – International Hyperbaric Medical Symposium



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USA Open 2018

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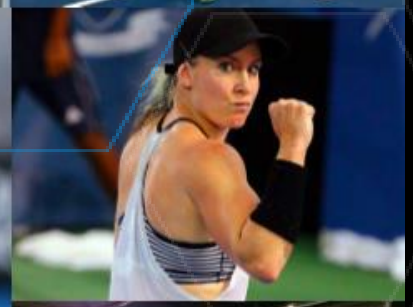
USA Open 2018

Women’s Doubles







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England

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USA

Canada

Monte Carlo

OXYMED

USA Davis Cup

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UK and Australian Rowing

Australian Rules Football (2012)

ASADA, AFL & WADA - "Period in question"

- During the period in question, the Essendon Football Club experienced 9 wins out of 11 consecutive games.
- One of the 2- games lost – the team kicked 9-goals and 1-point in the final quarter to lose the game by only 1-point.
- There were 'no reported soft tissues injuries' during the "period in question".
- The use of peptides were established in 2011, prior to attending HyperMED (OXYMED).
- So why were the players 30% up on their AFL Dream team scores during the "period in question"?
- What was the real reason for the improved performance?
- Why didn't any Australian news media discuss the role of Hyperbaric Oxygen Therapy in recovery and performance which negated the WADA decision that there was: *"no other evidence to challenge their circumstantial evidence that it must have been peptides causing the early season out performance".*
- *"Neither ASADA or WADA alleges that any players were given a banned substance at the South Yarra clinic". (Nov 2016).* http://www.theaustralian.com.au/sport/afl/news-story/f13971f9ae1a695f73c1677c52a6785

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VacuSports – Lower Body Negative Pressure

OXYMED

Cryotherapy – Minus 160C

WBV - Bones, Tendons, Ligaments

PM R. 2018 Apr 5. pii: S1934-1482(18)30172-2. doi: 10.1016/j.pmrj.2018.03.015. [Epub ahead of print]

Effects of Whole Body Vibration on Tibia Strength and Structure of Competitive Adolescent Swimmers: A Randomized Controlled Trial.

J Athl Train. 2018 Apr;53(4):355-363. doi: 10.4085/1062-6050-547-16. Epub 2018 Mar 23.

Whole-Body-Vibration Training and Balance in Recreational Athletes With Chronic Ankle Instability.

J Strength Cond Res. 2018 Jul;32(7):1809-1815. doi: 10.1519/JSC.0000000000002565.

Effects of Heavy Squat Training on a Vibration Platform on Maximal Strength and Jump Performance in Resistance-Trained Men.

Afr J Tradit Complement Altern Med. 2017 Jul 7;14(4 Suppl):19-27. doi: 10.21010/ajtcam.v14i4S.3. eCollection 2017.

RELEVANCE OF WHOLE BODY VIBRATION EXERCISE IN SPORT: A SHORT REVIEW WITH SOCCER, DIVER AND COMBAT SPORT.

Clin J Sport Med. 2018 Jul;28(4):339-349. doi: 10.1097/JSM.0000000000000466.

Efficacy of Whole-Body Vibration Board Training on Strength in Athletes After Anterior Cruciate Ligament Reconstruction: A Randomized Controlled Study.

Sports (Basel). 2017 Jan 13;5(1). pii: E7. doi: 10.3390/sports5010007.

Effect of Post-Exercise Whole Body Vibration with Stretching on Mood State, Fatigue, and Soreness in Collegiate Swimmers.

J Sports Sci Med. 2016 Feb 23;15(1):57-64. eCollection 2016 Mar.

Professional Soccer Player Neuromuscular Responses and Perceptions to Acute Whole Body Vibration Differ from Amateur Counterparts.

OXYMED

Low intensity stimuli at high frequencies

"Reduced negative effects of aging on bone, muscles and tendons"

Muscles Ligaments Tendons J. 2016 May 19;6(1):147-56. doi: 10.11138/mltj/2016.6.1.147. eCollection 2016 Jan-Mar.

Clinical applications of vibration therapy in orthopaedic practice.

Cerciello S[1], Rossi S[2], Visonà E[3], Corona K[4], Oliva F[5].

Author information

Abstract

BACKGROUND: Vibration therapy (VT) has been proposed as an option to improve physical performance and reduce the negative effects of ageing on bone, muscles and tendons. Several discrepancies exist on the type of applications, frequency and magnitude. These differences reflex on the contradictory clinical results in literature. Aim of the present study is to carry on an exhaustive review to focus on technical options on the market, clinical applications in orthopaedic practice and expected outcomes.

METHODS: a literature review using the key words "vibration therapy" and "whole-body vibration" and "orthopaedics" was performed. After checking the available abstracts 71 full text articles were evaluated.

RESULTS: fifty-one articles focused on the effects of VT on muscles and tendons reporting ways of action and clinical outcomes. In a similar way 20 studies focused on the influence of VT on bone tissue with regard on ways of action and clinical trials.

CONCLUSIONS: VT provides anabolic mechanical signals to bone and musculo-tendinous system. The best effects seem to be achieved with devices that deliver low-intensity stimuli at high frequencies providing linear horizontal displacement.

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Whole Body Cryotherapy – Cytokine markers

Cytokine. 2018 Jul 18. pii: S1043-4666(18)30310-7. doi: 10.1016/j.cyto.2018.07.018. [Epub ahead of print]

Whole-body cryotherapy (-110 °C) following high-intensity intermittent exercise does not alter hormonal, inflammatory or muscle damage biomarkers in trained males.

Krueger M[1], Costello JT[2], Achtzehn S[3], Dittmar KH[4], Mester J[5].

Author information

Abstract

PURPOSE: This study examined the acute effects of a single session of Whole-body Cryotherapy (WBC) following severe intermittent running exercise on biomarkers of inflammation, muscle damage and stress.

METHODS: Endurance-trained males (n = 11) were tested twice using a within-participant, balanced cross-over design that consisted of 5 × 5 min of high-intensity running (HIR) followed by either 3 min of WBC at -110 °C or a passive control condition (CON). Before the HIR and after 60 min of recovery a ramp-test was completed. At seven time points up to 24 hrs post exercise venous blood samples were analyzed for serum levels of interleukin 6 (IL-6), interleukin 10 (IL-10), c-reactive protein (CRP), soluble intercellular adhesion molecule-1 (sICAM-1), myoglobin, cortisol, and testosterone.

RESULTS: HIR induced significant increases in all biomarkers except sICAM-1 in both recovery conditions, respectively. Compared to the CON condition WBC did not attenuate exercise- induced changes in IL-6, IL-10, sICAM-1, myoglobin, cortisol, testosterone or their ratio. Increased levels of cortisol following exercise were negatively correlated with subsequent running performance in both conditions (WBC: r = -0.61, p = 0.04; CON: r = -0.64, p = 0.04).

CONCLUSION: The results of this study suggest that the postulated physiological mechanisms by which WBC is proposed to improve recovery, i.e. reductions in inflammation and muscle damage, may not be accurate.

OXYMED

Cryotherapy – post exercise recovery, DOMS

Front Physiol. 2017 May 2;8:258. doi: 10.3389/fphys.2017.00258. eCollection 2017.

Whole-Body Cryotherapy in Athletes: From Therapy to Stimulation. An Updated Review of the Literature.

Lombardi G[1], Ziemann E[2], Banfi G[1,3].

Author information

Abstract

Nowadays, whole-body cryotherapy is a medical physical treatment widely used in sports medicine. Recovery from injuries (e.g., trauma, overuse) and after-season recovery are the main purposes for application. However, the most recent studies confirmed the anti-inflammatory, anti-analgesic, and anti-oxidant effects of this therapy by highlighting the underlying physiological responses. In addition to its therapeutic effects, whole-body cryotherapy has been demonstrated to be a preventive strategy against the deleterious effects of exercise-induced inflammation and soreness. Novel findings have stressed the importance of fat mass on cooling effectiveness and of the starting fitness level on the final result. Exposure to the cryotherapy somehow mimics exercise, since it affects myokines expression in an exercise-like fashion, thus opening another possible window on the therapeutic strategies for metabolic diseases such as obesity and type 2 diabetes. From a biochemical point of view, whole-body cryotherapy not always induces appreciable modifications, but the final clinical output (in terms of pain, soreness, stress, and post-exercise recovery) is very often improved compared to either the starting condition or the untreated matched group. Also, the number and the frequency of sessions that should be applied in order to obtain the best therapeutic results have been deeply investigated in the last years. In this article, we reviewed the most recent literature, from 2010 until present, in order to give the most updated insight into this therapeutic strategy, whose rapidly increasing use is not always based on scientific assumptions and safety standards.

OXYMED

Cold Water Emersion

J Athl Train. 2016 Jul;51(7):540-9. doi: 10.4085/1062-6050-51.9.01. Epub 2016 Aug 30.

Use of Cold-Water Immersion to Reduce Muscle Damage and Delayed-Onset Muscle Soreness and Preserve Muscle Power in Jiu-Jitsu Athletes.

Fonseca LB[1], Brito CJ[2,3], Silva RJ[1], Silva-Grigoletto ME[1], da Silva WM Junior[1], Franchini E[3].

Author information

Abstract

CONTEXT: Cold-water immersion (CWI) has been applied widely as a recovery method, but little evidence is available to support its effectiveness.

OBJECTIVE: To investigate the effects of CWI on muscle damage, perceived muscle soreness, and muscle power recovery of the upper and lower limbs after jiu-jitsu training.

DESIGN: Crossover study.

SETTING: Laboratory and field.

PATIENTS OR OTHER PARTICIPANTS: A total of 8 highly trained male athletes (age = 24.0 ± 3.6 years, mass = 78.4 ± 2.4 kg, percentage of body fat = 13.1% ± 3.6%) completed all study phases.

INTERVENTION(S): We randomly selected half of the sample for recovery using CWI (6.0°C ± 0.5°C) for 19 minutes; the other participants were allocated to the control condition (passive recovery). Treatments were reversed in the second session (after 1 week).

MAIN OUTCOME MEASURE(S): We measured serum levels of creatine phosphokinase, lactate dehydrogenase (LDH), aspartate aminotransferase, and alanine aminotransferase enzymes; perceived muscle soreness; and recovery through visual analogue scales and muscle power of the upper and lower limbs at pretraining, postrecovery, 24 hours, and 48 hours.

RESULTS: Athletes who underwent CWI showed better posttraining recovery measures because circulating LDH levels were lower at 24 hours postrecovery in the CWI condition (441.9 ± 81.4 IU/L) than in the control condition (493.6 ± 97.4 IU/L; P = .03). Estimated muscle power was higher in the CWI than in the control condition for both upper limbs (757.9 ± 125.1 W versus 695.9 ± 56.1 W) and lower limbs (53.7 ± 3.7 cm versus 35.5 ± 8.2 cm; both P values = .001). In addition, we observed less perceived muscle soreness (1.5 ± 1.1 arbitrary units [au] versus 3.1 ± 1.0 au; P = .004) and higher perceived recovery (8.8 ± 1.9 au versus 6.9 ± 1.7 au; P = .005) in the CWI than in the control condition at 24 hours postrecovery.

CONCLUSIONS: Use of CWI can be beneficial to jiu-jitsu athletes because it reduces circulating LDH levels, results in less perceived muscle soreness, and helps muscle power recovery at 24 hours postrecovery.

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Cryotherapy

J Sports Sci Med. 2017 Jun 1;16(2):272-279. eCollection 2017 Jun.

5000 Meter Run Performance is not Enhanced 24 Hrs After an Intense Exercise Bout and Cold Water Immersion.

BMJ Case Rep. 2017 Oct 13;2017. pii: bcr-2017-221431. doi: 10.1136/bcr-2017-221431.

Transient global amnesia following a whole-body cryotherapy session.

Carrard J[1], Lambert AC[1], Genné D[1].

Author information

Abstract

Whole-body cryotherapy (WBC), which consists of a short exposure to very cold and dry air in special 'cryo-chambers', is believed to reduce inflammation and musculoskeletal pain as well as improve athletes' recovery. This is the case of a 63-year-old male, who presented with transient global amnesia (TGA) after undertaking a WBC session. TGA is a clinical syndrome characterised by a sudden onset of anterograde amnesia, sometimes coupled with a retrograde component, lasting up to 24 hours without other neurological deficits. Even though the patient completely recovered, as expected, in 24 hours, this case highlights that WBC is potentially not as risk free as thought to be initially. To conclude, before WBC can be medically recommended, well-conducted studies investigating the possible adverse events are required.

Front Physiol. 2018 May 30;9:659. doi: 10.3389/fphys.2018.00659. eCollection 2018.

Unchanged Erythrocyte Profile After Exposure to Cryogenic Temperatures in Elder Marathon Runners.

J Hum Kinet. 2018 Jun 13;62:55-63. doi: 10.1515/hukin-2017-0158. eCollection 2018 Jun.

Thermal Sensations during a Partial-Body Cryostimulation Exposure in Elite Basketball Players.

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Hyperbaric Oxygen Therapy – Placebo?

PLoS One. 2016 Mar 9;11(3):e0150517. doi: 10.1371/journal.pone.0150517. eCollection 2016.

The Effects of Hyperbaric Oxygen Therapy on Post-Training Recovery in Jiu-Jitsu Athletes.

Branco BH[1,2], Fukuda DH[3], Andreato LV[4], Santos JF[1,2], Esteves JV[5], Franchini E[1,2].

Author information

Abstract

OBJECTIVES: The present study aimed to evaluate the effects of using hyperbaric oxygen therapy during post-training recovery in jiu-jitsu athletes.

METHODS: Eleven experienced Brazilian jiu-jitsu athletes were investigated during and following two training sessions of 1h30min. Using a cross-over design, the athletes were randomly assigned to passive recovery for 2 hours or to hyperbaric oxygen therapy (OHB) for the same duration. After a 7-day period, the interventions were reversed. Before, immediately after, post 2 hours and post 24 hours, blood samples were collected to examine hormone concentrations (cortisol and total testosterone) and cellular damage markers [creatine kinase (CK), aspartate aminotransferase (AST), alanine aminotransferase (ALT), and lactate dehydrogenase (LDH)]. Moreover, the rating of perceived exertion (RPE) and recovery (RPR) scales were applied.

RESULTS: Final lactate [La] values (control: 11.9 ± 1.4 mmol/L, OHB: 10.2 ± 1.4 mmol/L) and RPE [control: 14 (13-17 a.u.), OHB: 18 (17-20 a.u.)] were not significantly different following the training sessions. Furthermore, there was no difference between any time points for blood lactate and RPE in the two experimental conditions ($P>0.05$). There was no effect of experimental conditions on cortisol ($F1,20 = 0.1$, $P = 0.793$, $\eta2 = 0.00$, small), total testosterone ($F1,20 = 0.03$, $P = 0.877$, $\eta2 = 0.00$, small), CK ($F1,20 = 0.1$, $P = 0.759$, $\eta2 = 0.01$, small), AST ($F1,20 = 0.1$, $P = 0.761$, $\eta2 = 0.01$, small), ALT ($F1,20 = 0.0$, $P = 0.845$, $\eta2 = 0.00$, small) or LDH ($F1,20 = 0.7$, $P = 0.413$, $\eta2 = 0.03$, small). However, there was a difference between the two experimental conditions in RPR with higher values at post 2 h and 24 h in OHB when compared to the control condition ($P<0.05$).

CONCLUSIONS: Thus, it can be concluded that OHB exerts no influence on the recovery of hormonal status or cellular damage markers. Nonetheless, greater perceived recovery, potentially due to the placebo effect, was evident following the OHB condition.

OXYMED

HBOT placebo? Requires larger cohort clinical trials

PLoS One. 2016 Mar 9;11(3):e0150517. doi: 10.1371/journal.pone.0150517. eCollection 2016.

The Effects of Hyperbaric Oxygen Therapy on Post-Training Recovery in Jiu-Jitsu Athletes.

Ther Adv Musculoskelet Dis. 2011 Apr;3(2):111-21. doi: 10.1177/1759720X11399172.

Hyperbaric oxygen effects on sports injuries.

Barata P[1], Cervaens M, Resende R, Camacho O, Marques F.

Author information

Abstract

In the last decade, competitive sports have taken on a whole new meaning, where intensity has increased together with the incidence of injuries to the athletes. Therefore, there is a strong need to develop better and faster treatments that allow the injured athlete to return to competition faster than with the normal course of rehabilitation, with a low risk of re-injury. Hyperbaric therapies are methods used to treat diseases or injuries using pressures higher than local atmospheric pressure inside a hyperbaric chamber. Within hyperbaric therapies, hyperbaric oxygen therapy (HBO) is the administration of pure oxygen (100%) at pressures greater than atmospheric pressure, i.e. more than 1 atmosphere absolute (ATA), for therapeutic reasons. The application of HBO for the treatment of sports injuries has recently been suggested in the scientific literature as a modality of therapy either as a primary or an adjunct treatment. Although results have proven to be promising in terms of using HBO as a treatment modality in sports-related injuries, these studies have been limited due to the small sample size, lack of blinding and randomization problems. HBO seems to be promising in the recovery of injuries for high-performance athletes; however, there is a need for larger samples, randomized, controlled, double-blinded clinical trials combined with studies using animal models so that its effects and mechanisms can be identified to confirm that it is a safe and effective therapy for the treatment of sports injuries.

OXYMED

HBOT _ DOMS, Fatigue

Sports Med. 2006;36(9):781-96.

Using recovery modalities between training sessions in elite athletes: does it help?

Sports Med. 2005;35(9):739-46.

Hyperbaric oxygen as an adjuvant for athletes.

Ishii Y[1], Deie M, Adachi N, Yasunaga Y, Sharman P, Miyanaga Y, Ochi M.

Author information

Abstract

There has recently been a resurgence in interest in hyperbaric oxygen (HBO) treatment in sports therapy, especially in Japan. Oxygen naturally plays a crucial role in recovery from injury and physiological fatigue. By performing HBO treatment, more oxygen is dissolved in the plasma of the pulmonary vein via the alveolar, increasing the oxygen reaching the peripheral tissues. HBO treatment is therefore expected to improve recovery from injury and fatigue. HBO treatment has been reported to reduce post-injury swelling in animals, and in humans; swelling was also mitigated, but to a lesser extent. Positive results have also been reported regarding tissue remodelling after injury, with injuries involving bones, muscles and ligaments showing improved recovery. Furthermore, HBO treatment has effectively increased recovery from fatigue. This was clearly seen at the Nagano Winter Olympics, where sports players experiencing fatigue were successfully treated, enabling the players to continue performing in the games. Despite its potential, HBO treatment does have its risks. Increasing oxygen levels in tissues poses a risk to DNA through oxidative damage, which can lead to pathological changes in the CNS and the lungs. Regarding the operating of HBO systems, safer administration should be advised. Further research into HBO treatment is required if this therapy is to become more widespread. It should become possible to tailor treatment to an individual's condition in order to use HBO treatment efficiently.

OXYMED

Satellite cell proliferation, IGF1 healing

J Appl Physiol (1985). 2014 Jan 15;116(2):149-55. doi: 10.1152/japplphysiol.00235.2013. Epub 2013 Dec 12.

Enhancement of satellite cell differentiation and functional recovery in injured skeletal muscle by hyperbaric oxygen treatment.

Horie M[1], Enomoto M, Shimoda M, Okawa A, Miyakawa S, Yagishita K.

Author information

Abstract

Recently, the use of hyperbaric oxygen (HBO) treatments by elite athletes to accelerate recovery from muscle injuries has become increasingly popular. However, the mechanism of promoting muscle regeneration under HBO conditions has not yet been defined. In this study, we investigated whether HBO treatments promoted muscle regeneration and modulated muscle regulatory factor expression in a rat skeletal muscle injury model. Muscle injury was induced by injecting cardiotoxin (CTX) into the tibialis anterior (TA) muscles. As the HBO treatment, rats were placed in an animal chamber with 100% oxygen under 2.5 atmospheres absolute for 2 h/day, 5 days/wk for 2 wk. We then performed histological analyses, measured the maximum force-producing capacity of the regenerating muscle fibers, and performed quantitative RT-PCR analysis of muscle regulatory factor mRNAs. The cross-sectional areas and maximum force-producing capacity of the regenerating muscle fibers were increased by HBO treatment after injury. The mRNA expression of MyoD, myogenin, and IGF-1 increased significantly in the HBO group at 3 and 5 days after injury. The number of Pax7(+)/MyoD(+), Pax7(-)/MyoD(+), and Pax7(+)/BrdU(+)-positive nuclei was increased by HBO treatment. In this study, we demonstrated that HBO treatment accelerated satellite cell proliferation and myofiber maturation in rat muscle that was injured by a CTX injection. These results suggest that HBO treatment accelerates healing and functional recovery after muscle injury.

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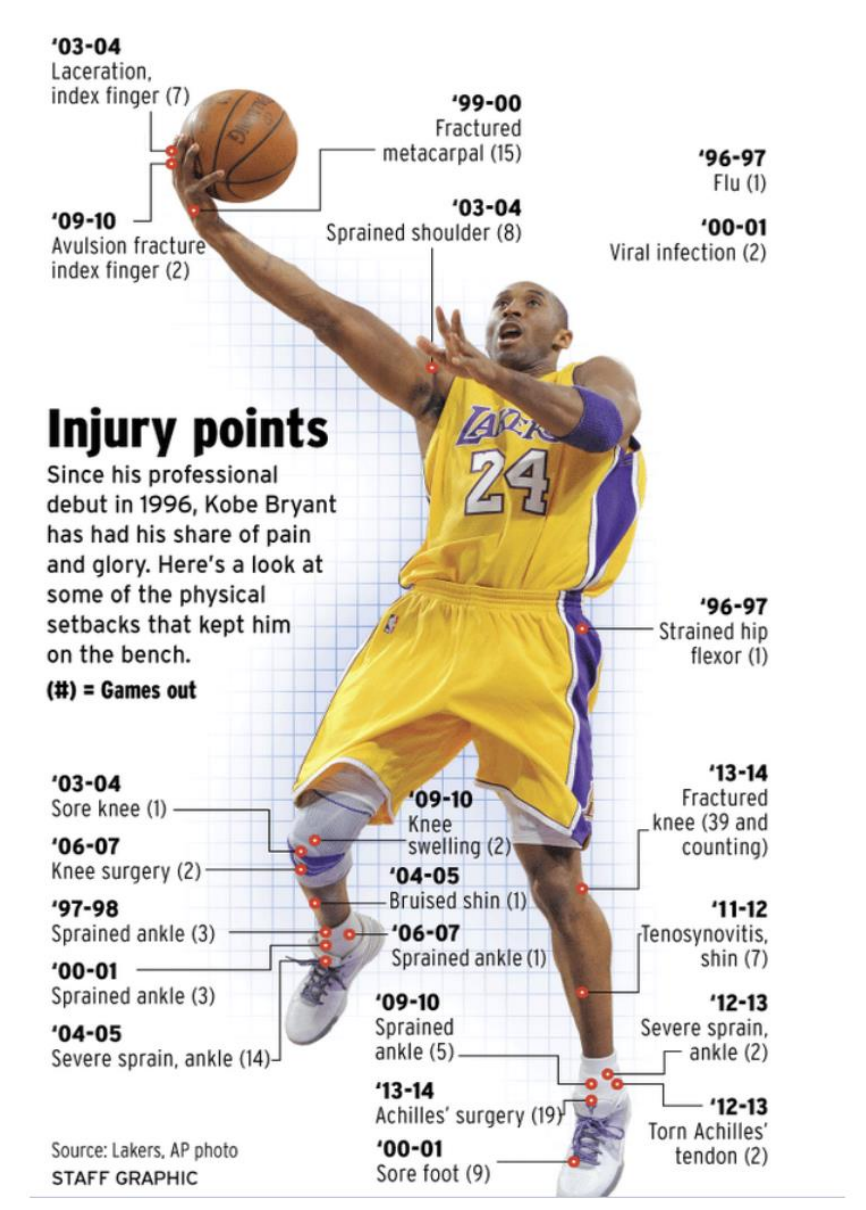
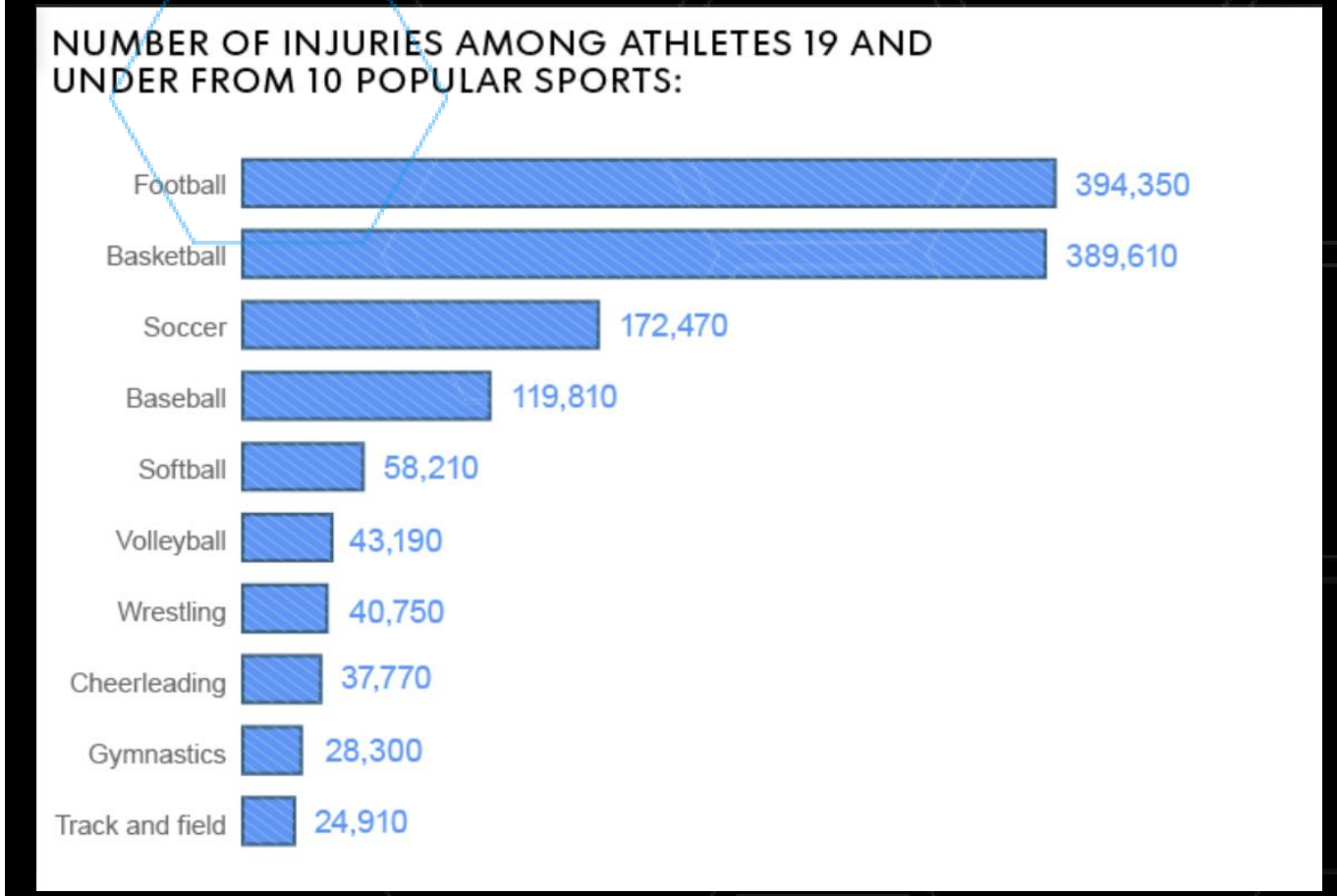
Growing injury list - performance, recovery, travel – the endless loop





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Chronic Traumatic Encephalopathy (CTE)

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Burn Out

- **Multidimensional Cognitive Affective Syndrome** – “emotional, physical exhaustion, reduced sense of accomplishment and sport devaluation”. Burn-out affects 1-10% of all athletes (figure probably much higher).
- Negative impact – social relationship breakdowns; mental anxiety, eating disorders, self abuse, PTSD etc.

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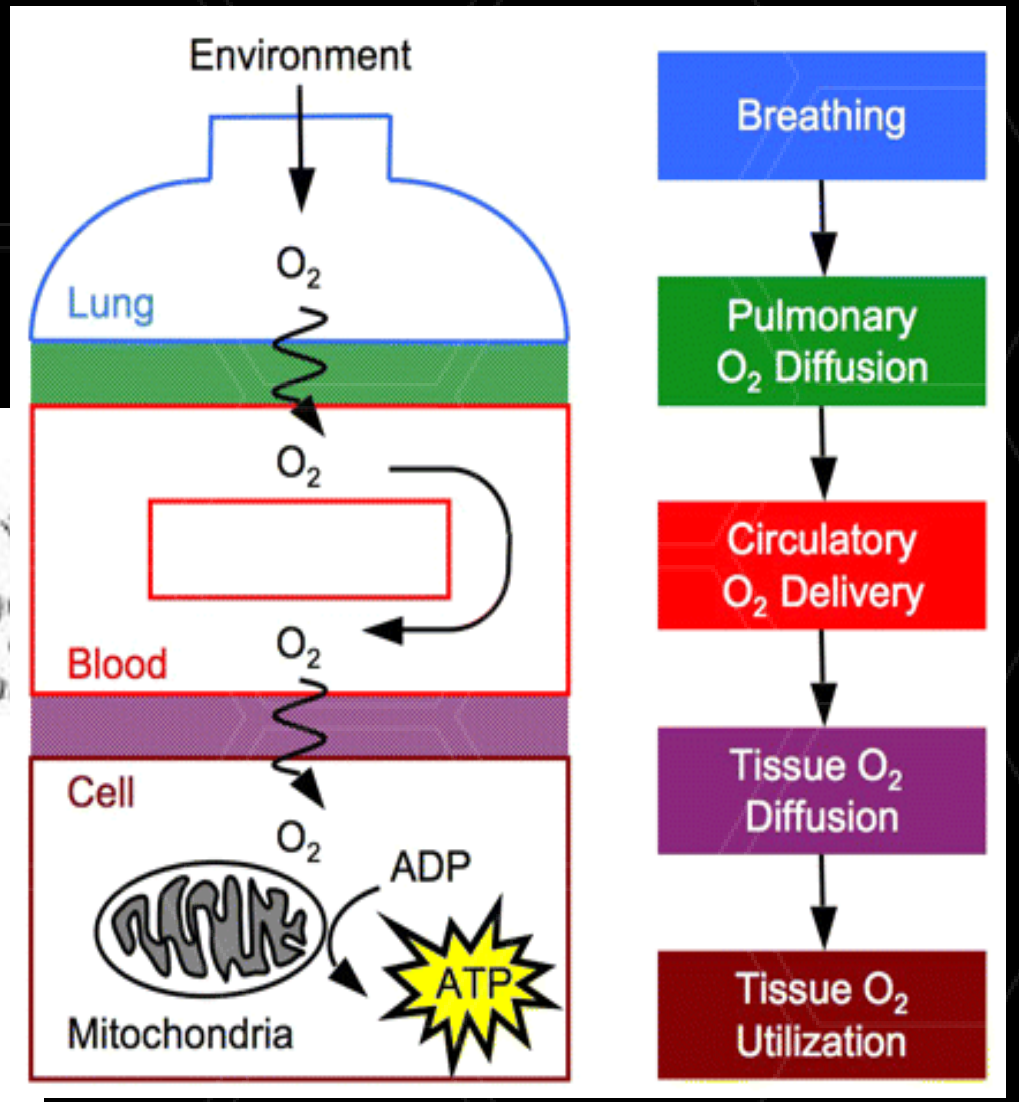
What happens if you get the “fuel” wrong?



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What is plan B?

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Show Me The Money!

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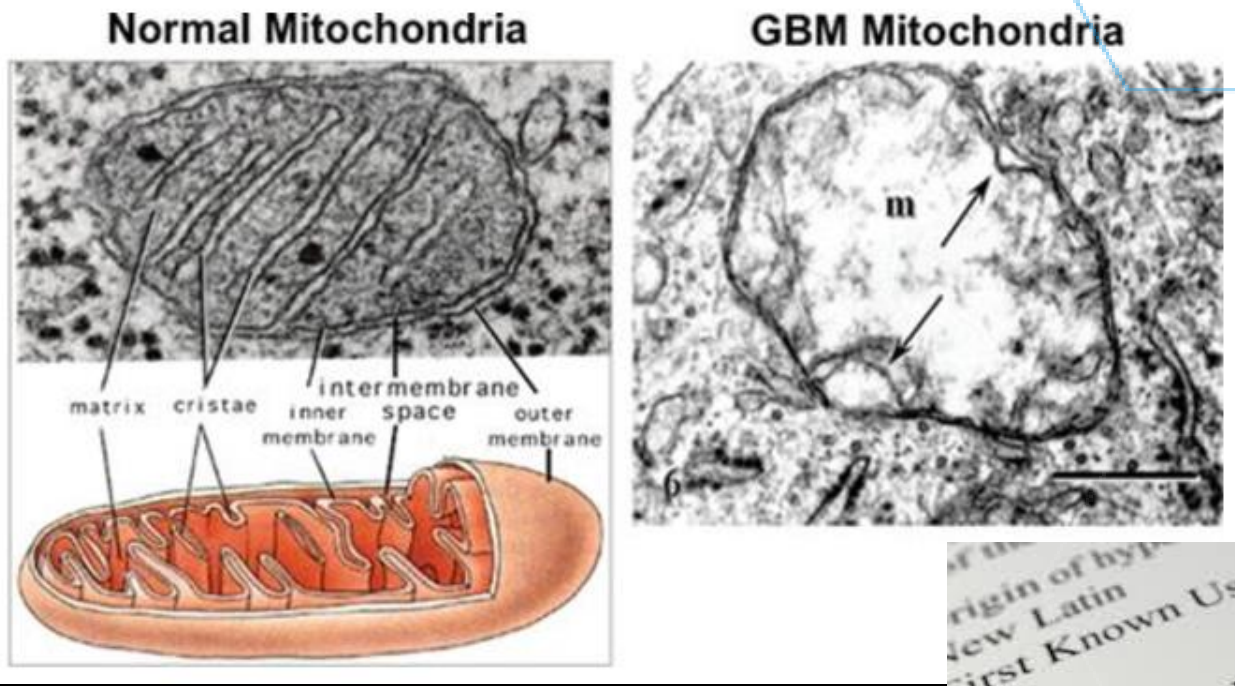
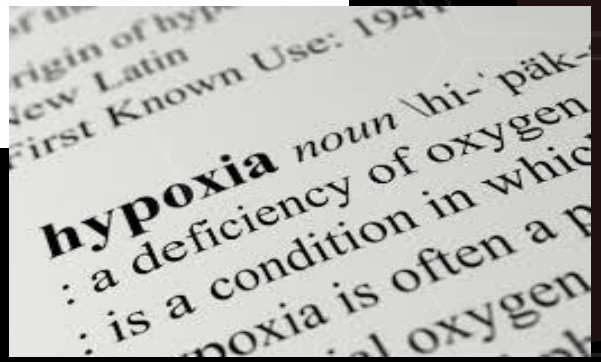
“The Mitochondria Oxygen Money Meter”

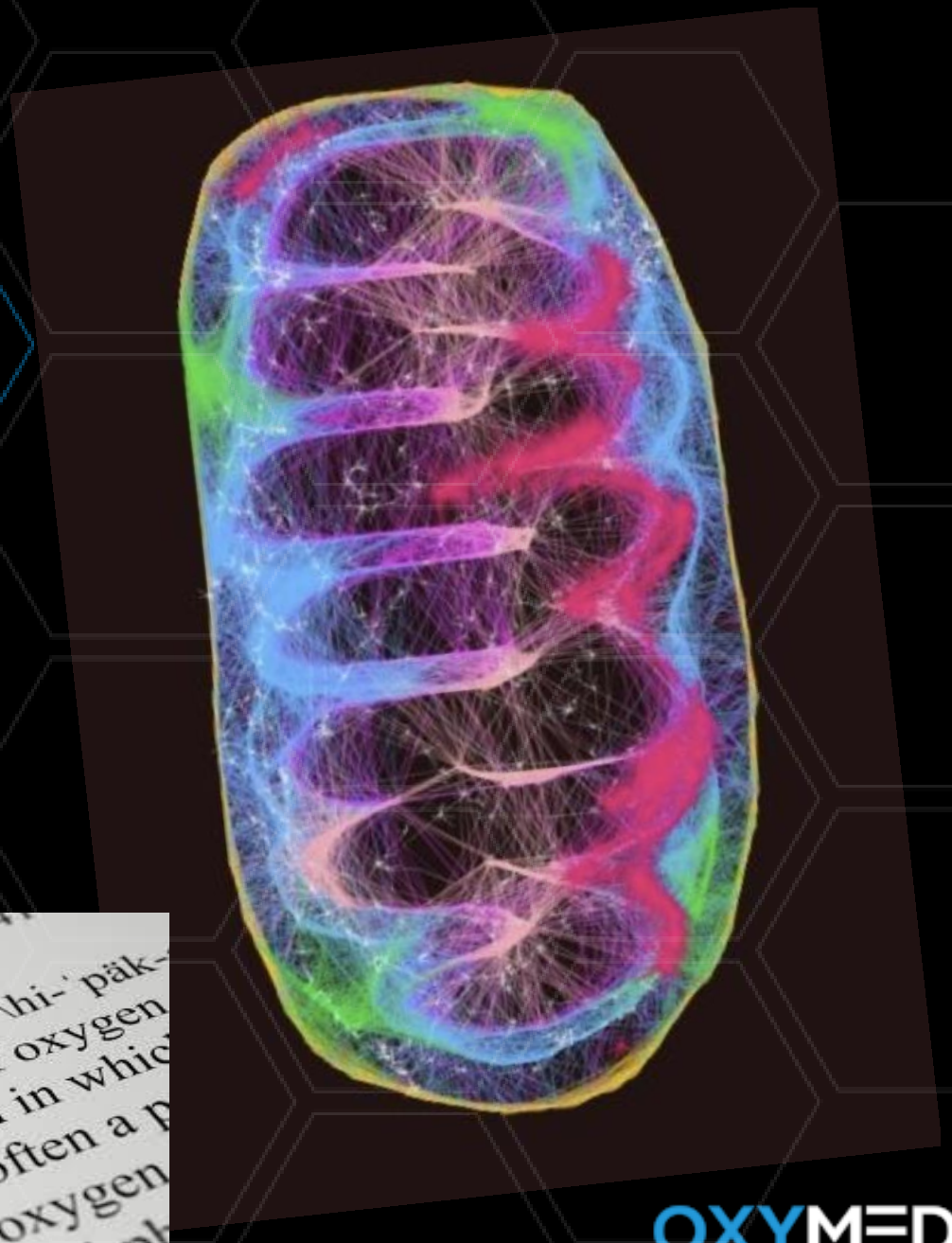
Cancer as a metabolic disease: implications for novel therapeutics

Thomas N. Seyfried ✉, Roberto E. Flores, Angela M. Poff, Dominic P. D’Agostino

Carcinogenesis, Volume 35, Issue 3, 1 March 2014, Pages 515–527,







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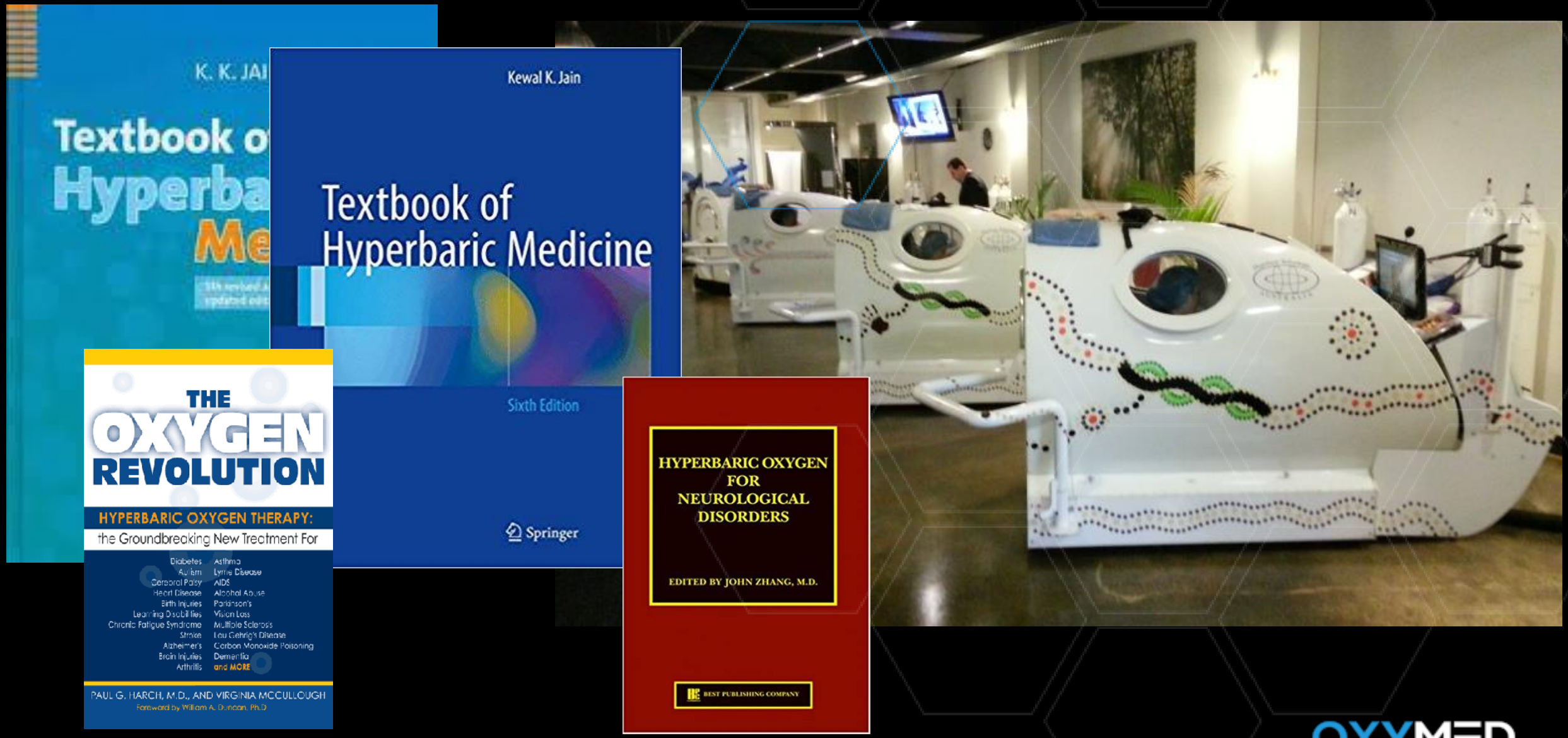
OXYMED Hyperbaric Oxygen Therapy



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Tel Aviv | The Sagol Hyperbaric Center

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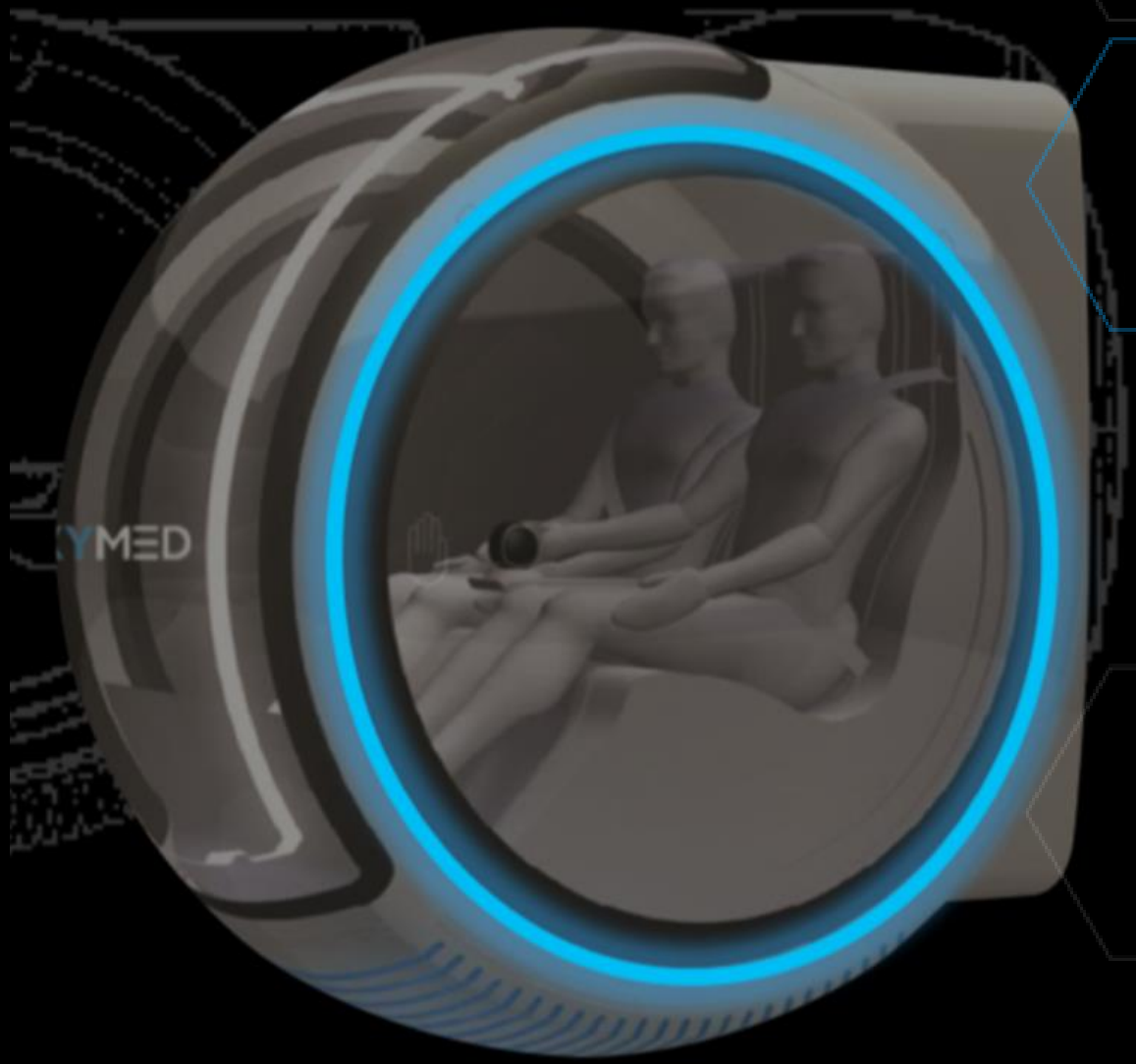
OXYMED International Development



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Advertised “Benefits” of Hyperbaric

Source: www ….

- Preconditioning against injury
- Shortens recovery time after extreme exercise, injury or surgery
- Revitalizes by improving blood flow and oxygen to all organs
- Regenerates small blood vessels (capillaries), nerves and bones
- Improved performance
- Increased strength
- Enhanced endurance
- Energy boost preventing exhaustion
- Reducing inflammation, swelling, pain
- Reducing fatigue and recovery time
- Speeding up healing of muscles, ligaments and fractured bones
- Rejuvenates by releasing stem cells from bone marrow for tissue repair
- Reducing and preventing infection
- Reducing scar tissue formation
- Cleansing blood from toxins and toxic substances
- Maintaining general health

Why do many athletes feel flat or even come down with ‘flu like’ symptoms after HBOT?

OXYMED

Cytokines Gene Signalling – The Cellular Landscape

Cytokine Gene Expression Testing is at the forefront of medical advances and immunotherapy interventions.

Type into **Google search** - the ‘health condition’ and ‘cytokines’.

- Why aren't governments paying for Cytokine testing enabling the public to access these specific advances with view of how your body is actually functioning at a ‘cellular level’?

The answer is simple.

- Cytokine testing places focus on the "individual" cellular gene expression with the view that your health is simply not another ‘generic drug for another symptom’.

Circulating Blood

- The cytokine biomarker blood tests are specific indicators of what is happening in your circulating blood.
- However, it does not necessarily reflect what is actually happening in the deeper tissues where cells are in a lowered respiration (low metabolic) state.
- Cells in a hypoxic state are the cells that “over secrete” pro-inflammatory cytokines and inflammatory gene expressions.

- Inflammatory responses are required in the **acute phase** of illness but become destructive (apoptosis) with **chronic long term expression** leading to neurodegeneration and autoimmune related illness.

OXYMED

PubMED – “Cytokines Athletes & Injuries”

Clin Biochem. 2018 Feb;52:142-147. doi: 10.1016/j.clinbiochem.2017.11.018. Epub 2017 Dec 2.

Impact of long distance rowing on biological health: A pilot study.

Frias MA[1], Virzi J[2], Golaz O[3], Gencer B[4], Mach F[4], Vuilleumier N[2].

Author information

Abstract

OBJECTIVES: To determine the impact of long distance rowing (160km, nonstop) on standard biological parameters and to study the relation between inflammation, myocardial necrosis, lipid profile, heart rate and energy expenditure.

J Sports Sci. 2018 Oct;36(19):2226-2234. doi: 10.1080/02640414.2018.1448570. Epub 2018 Mar 6.

Inflammatory and apoptotic signalling pathways and concussion severity: a genetic association study.

J Sports Med Phys Fitness. 2016 Oct;56(10):1113-1119. Epub 2015 Nov 11.

Acute physiological changes in elite free-style wrestlers during a one-day tournament.

PLoS One. 2016 Jul 26;11(7):e0159929. doi: 10.1371/journal.pone.0159929. eCollection 2016.

Altered Blood Biomarker Profiles in Athletes with a History of Repetitive Head Impacts.

J Sports Med Phys Fitness. 2016 Jun;56(6):665-77. Epub 2015 Feb 18.

Differing cytokine responses by ethnic groups to a bout of exercise-induced muscle damage: a preliminary report.

Starzak DE[1], Semple SJ, Smith LL, McKune AJ.

Author information

Abstract

BACKGROUND: Strenuous exercise has been shown to alter immune and inflammatory responses potentially predisposing athletes to infection and injury. Ethnic disparities have been demonstrated in athletic performance and in the way individuals respond to exercise as well as in the predisposition towards certain diseases however, the information relating to immune and inflammatory responses to exercise between ethnic groups is still limited. The aim of this study was to investigate whether serum cytokine levels respond differently to eccentrically-biased exercise in African and Caucasian males.

OXYMED

Cytokine Modulators

J Sports Sci. 2018 Aug;36(16):1897-1901. doi: 10.1080/02640414.2018.1424491. Epub 2018 Jan 9.

Melatonin therapy for blunt trauma and strenuous exercise: A mechanism involving cytokines, NFκB, Akt, MAF_{BX} and MURF-1.

Maarman GJ[1], Reiter RJ[2].

Author information

Abstract

Muscle injury occurs due to trauma, strenuous exercise or sports activities; most people affected are athletes. Ineffectively treated muscle injury can negatively affect sports careers and quality of life after retirement from sports. Reports have indicated that the current therapeutic management of muscle injury, particularly anti-inflammatory drugs, are not necessarily effective. Therefore, better therapies are required. Accumulating evidence has demonstrated melatonin's potent antioxidant and anti-inflammatory actions against muscle pathology in sarcopenia or atrophy in systemic disease. However, the underlying mechanisms for the protective effect of melatonin in the context of trauma/strenuous exercise are multifactorial and not well described. This paper reviews data on melatonin's impact on muscle injury and findings that points toward the mechanisms through which melatonin achieves muscle protection. The general concept described in this review is that melatonin inhibits NFκB, reduces cytokine expression, increases Akt that downregulates the ratio of MAF_{BX} and MURF-1 in order to limit the extent of muscle injury and promote muscle recovery post-injury. The work discussed in this review supports the notion that melatonin may be considered a possible therapy against trauma/sports related muscle injury. Inclusion of melatonin as a therapy in sports medicine could therefore provide a better treatment option for injured athletes and sports individuals.

OXYMED

Dietary influences Cytokines and sports

J Int Soc Sports Nutr. 2017 Feb 28;14:7. doi: 10.1186/s12970-017-0165-z. eCollection 2017.

Effects of cranberry (*Vaccinum macrocarpon*) supplementation on iron status and inflammatory markers in rowers.

J Int Soc Sports Nutr. 2014 Oct 1;11(1):48. doi: 10.1186/s12970-014-0048-5. eCollection 2014.

Effect of supplementation with chokeberry juice on the inflammatory status and markers of iron metabolism in rowers.

CONCLUSION: Supplementation with chokeberry juice results in an increase in the antioxidant activity of plasma and contributes significantly to reducing the TNF-alpha level.

OXYMED

Ketogenic Diet Transition

J Int Soc Sports Nutr. 2018 Jul 9;15(1):31. doi: 10.1186/s12970-018-0236-9.

Efficacy of ketogenic diet on body composition during resistance training in trained men: a randomized controlled trial.

J Sports Med Phys Fitness. 2018 Apr 4. doi: 10.23736/S0022-4707.18.08318-4. [Epub ahead of print]

Low-carbohydrate, ketogenic diet impairs anaerobic exercise performance in exercise-trained women and men: a randomized-sequence crossover trial.

Wroble KA[1], Trott MN[1], Schweitzer GG[1,2], Rahman RS[1], Kelly PV[3], Weiss EP[4,3].

Author information

Abstract

BACKGROUND: Low-carbohydrate, ketogenic diets cause mild, sub-clinical systemic acidosis. Anaerobic exercise performance is limited by acidosis. Therefore, we evaluated the hypothesis that a low-carbohydrate, ketogenic diet impairs anaerobic exercise performance, as compared to a high-carbohydrate diet.

METHODS: Sixteen men and women (BMI, 23±1 kg/m2, age 23±1 yr) participated in a randomized-sequence, counterbalanced crossover study in which they underwent exercise testing after four days of either a low-carbohydrate, ketogenic diet (LC; <50 g/day and <10% of energy from carbohydrates) or a high-carbohydrate diet (HC; 6-10 g/kg/day carbohydrate). Dietary compliance was assessed with nutrient analysis of diet records, and with measures of urine pH and ketones. Anaerobic exercise performance was evaluated with the Wingate anaerobic cycling test and the yo-yo intermittent recovery test.

RESULTS: The diets were matched for total energy (LC: 2333±158 kcal/d; HC: 2280±160 kcal/d; p=0.65) but differed in carbohydrate content (9±1 vs. 63±2% of energy intake; p<0.001). LC resulted in lower urine pH (5.9±0.1 vs. 6.3±0.2, p=0.004) and the appearance of urine ketones in every participant. LC resulted in 7% lower peak power (801±58 vs. 857±61 watts, p=0.008) and 6% lower mean power (564±50 vs. 598±51 watts, p=0.01) during the Wingate test. Total distance ran in the yo-yo intermittent recovery test was 15% less after LC diet (887±139 vs. 1045±145 meters, p=0.02).

CONCLUSIONS: Short-term low-carbohydrate, ketogenic diets reduce exercise performance in activities that are heavily dependent on anaerobic energy systems. These findings have clear performance implications for athletes, especially for high-intensity, short duration activities and sports.

OXYMED

Ketogenic benefits

"enhanced well being, improved recovery, improvements in skin, reduced inflammation"

J Int Soc Sports Nutr. 2017 Jul 12;14:22. doi: 10.1186/s12970-017-0180-0. eCollection 2017.

Ketogenic diet benefits body composition and well-being but not performance in a pilot case study of New Zealand endurance athletes.

Zinn C[1], Wood M[1], Williden M[1], Chatterton S[1], Maunder E[1].

Author information

Abstract

BACKGROUND: Low-carbohydrate, high-fat and ketogenic diets are increasingly adopted by athletes for body composition and sports performance enhancements. However, as yet, there is no consensus on their efficacy in improving performance. There is also no comprehensive literature on athletes' experiences while undertaking this diet. The purpose of this pilot work was two-fold: i. to examine the effects of a non-calorie controlled ketogenic diet on body composition and performance outcomes of endurance athletes, and ii. to evaluate the athletes' experiences of the ketogenic diet during the 10-week intervention.

METHODS: Using a case study design, five New Zealand endurance athletes (4 females, 1 male) underwent a 10-week ketogenic dietary intervention. Body composition (sum of 8 skinfolds), performance indicators (time to exhaustion, VO_2 max, peak power and ventilatory threshold), and gas exchange thresholds were measured at baseline and at 10 weeks. Mean change scores were calculated, and analysed using t-tests; Cohen's effect sizes and 90% confidence limits were applied to quantify change. Individual interviews conducted at 5 weeks and a focus group at 10 weeks assessed athletes' ketogenic diet experiences. Data was transcribed and analysed using thematic analysis.

RESULTS: All athletes increased their ability to utilise fat as a fuel source, including at higher exercise intensities. Mean body weight was reduced by 4 kg ± SD 3.1 (p = 0.046; effect size (ES):0.62), and sum of 8 skinfolds by 25.9 mm ± SD 6.9; ES: 1.27; p = 0.001). Mean time to exhaustion dropped by ~2 min (±SD 0.7; p = 0.004; ES: 0.53). Other performance outcomes showed mean reductions, with some increases or unchanged results in two individuals (VO2 Max: -1.69 ml.kg.min ± SD 3.4 (p = 0.63); peak power: -18 W ± SD 16.4 (p = 0.07), and VT2: -6 W ± SD 44.5 (p = 0.77). Athletes reported experiencing reduced energy levels initially, followed by a return of high levels thereafter, especially during exercise, but an inability to easily undertake high intense bouts. Each athlete reported experiencing enhanced well-being, included improved recovery, improvements in skin conditions and reduced inflammation.

CONCLUSIONS: Despite performance decrements and some negative experiences, athletes were keen to pursue a modified low-carbohydrate, high-fat eating style moving forward due to the unexpected health benefits they experienced.

OXYMED

PRP & Cytokines vs HBOT Plus PRP

Nutrition. 2016 May;32(5):539-45. doi: 10.1016/j.nut.2015.11.002. Epub 2015 Dec 7.

Effects of pomegranate juice in circulating parameters, cytokines, and oxidative stress markers in endurance-based athletes: A randomized controlled trial.

J Equine Sci. 2017;28(2):31-39. doi: 10.1294/jes.28.31. Epub 2017 Jul 6.

Does the injection of platelet-rich plasma induce changes in the gene expression and morphology of intact Thoroughbred skeletal muscle?

Curr Rev Musculoskelet Med. 2015 Jun;8(2):145-53. doi: 10.1007/s12178-015-9259-x.

Platelet-rich plasma for muscle injuries: game over or time out?

Mosca MJ[1], Rodeo SA.

Author information

Abstract

Muscle injuries are common and may be associated with impaired functional capacity, especially among athletes. The results of healing with conventional therapy including rest, ice, compression, and elevation (RICE) are often inadequate, generating substantial interest in the potential for emerging technologies such as platelet-rich plasma (PRP) to enhance the process of soft-tissue healing and to decrease time to recovery. In vitro studies and animal research have suggested that PRP may have benefits associated with the increased release of cytokines and growth factors resulting from supraphysiological concentrations of platelets that facilitate muscle repair, regeneration, and remodeling. Despite the promise of basic science, there is a paucity of clinical data to support the theoretical benefits of PRP. The only double-blind controlled clinical trial was recently reported and showed no benefit of PRP in the time to resume sports activity among athletes with hamstring muscle injury. This review examines the current evidence and the theoretical framework for PRP and muscle healing. Scientific gaps and technological barriers are discussed that must be addressed if the potential promise of PRP as a therapeutic modality for muscle injury is to be realized.

OXYMED

Cytokines ‘orchestrate’ immune modulation

- Cytokines 'orchestrate' & modulate immune response & gene expression.

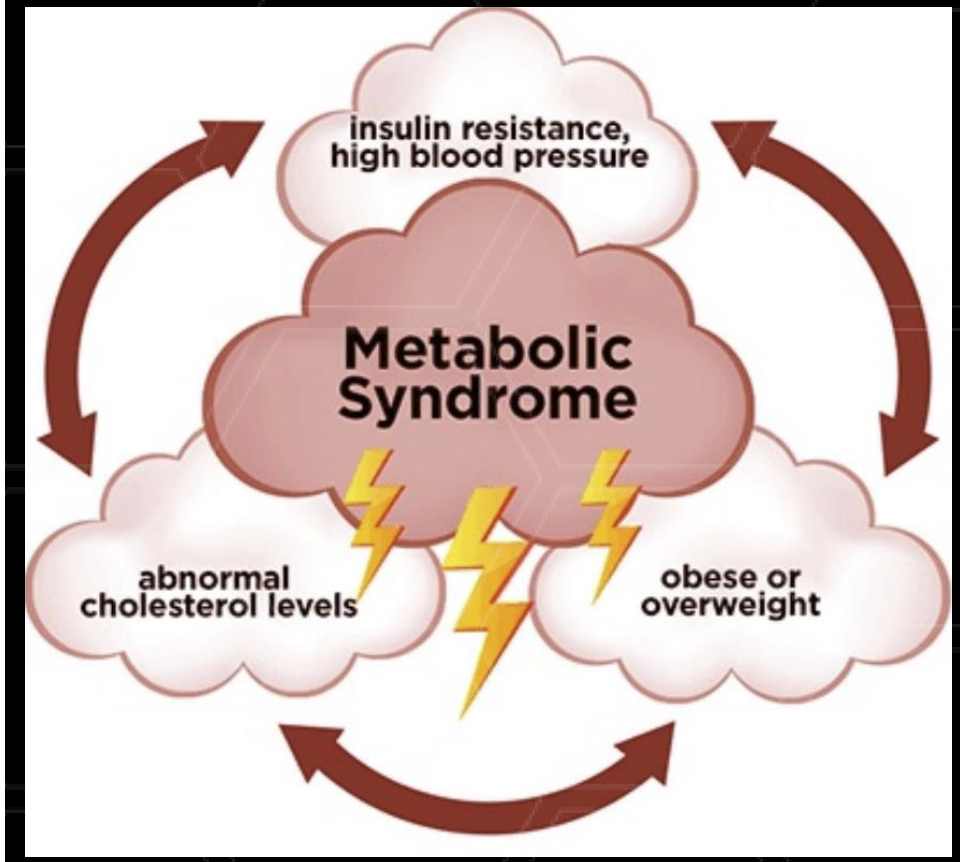
- Cytokine modulate cell growth and differentiation, cell death (apoptosis), angiogenesis, normal development and neuromodulations.
- They are known as either **pro-inflammatory** or **anti-inflammatory.**

- **“Autophagy Cross Talk”** - Cells ‘talk’ to other cells – mediated by pro-inflammatory Cytokines and other glycoproteins including HIF other factors including NFkB, TGFb.

Hypoxic Cross-Talk

- Hypoxia drives molecular crosstalk of multiple pro-inflammatory loops. NFkB is also another key regulator in the crosstalk among the pathways leading to: arthritis, inflammatory bowel disease (IBD), colorectal cancer (CRC) and type 2 diabetes mellitus (T2DM), systemic inflammation.
- ‘Hypoxic Cross Talk’ plays a major role in linking inflammation to cancer development through its ability to up regulate inflammatory tumor promoting cytokines - IL6, IL1α and TNFα and genes like BCL2 and BCLXL.

- Chronic ‘over-expression’ and ‘cross-talk’ of **HIF, TGFβ, NFKB, TNFα, IL1, IL6, IL8** could be considered as the matchmaker between inflammation, IBD, cancer and diabetes – Metabolic Syndrome



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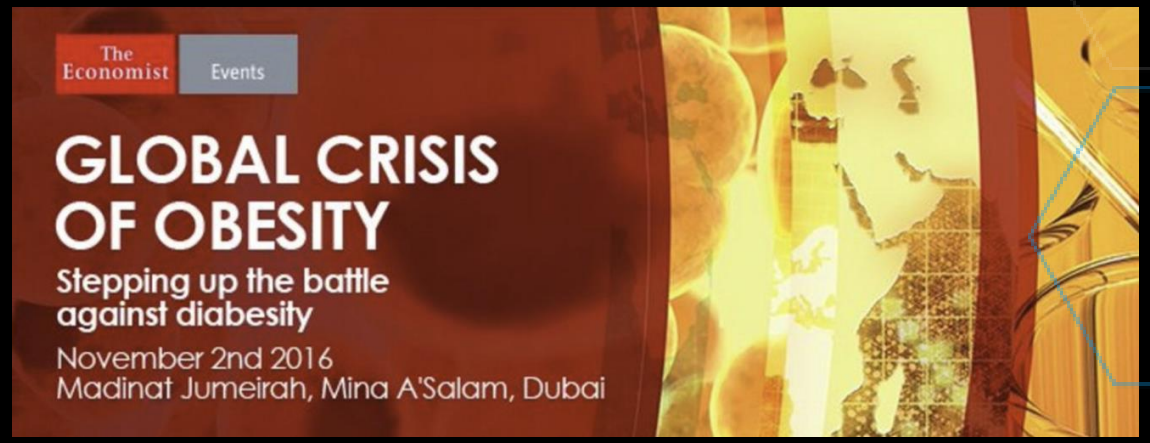
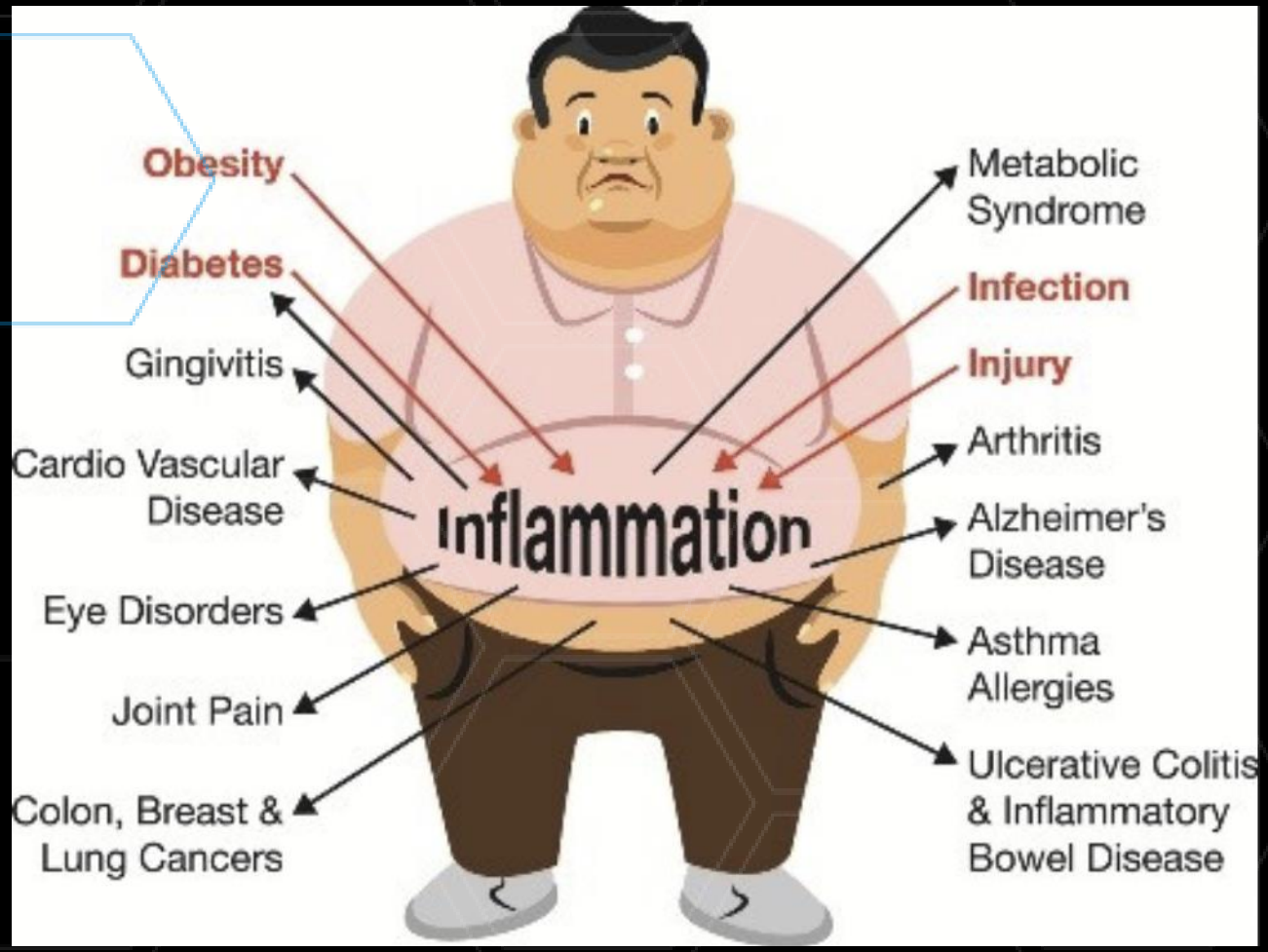
Global Crisis





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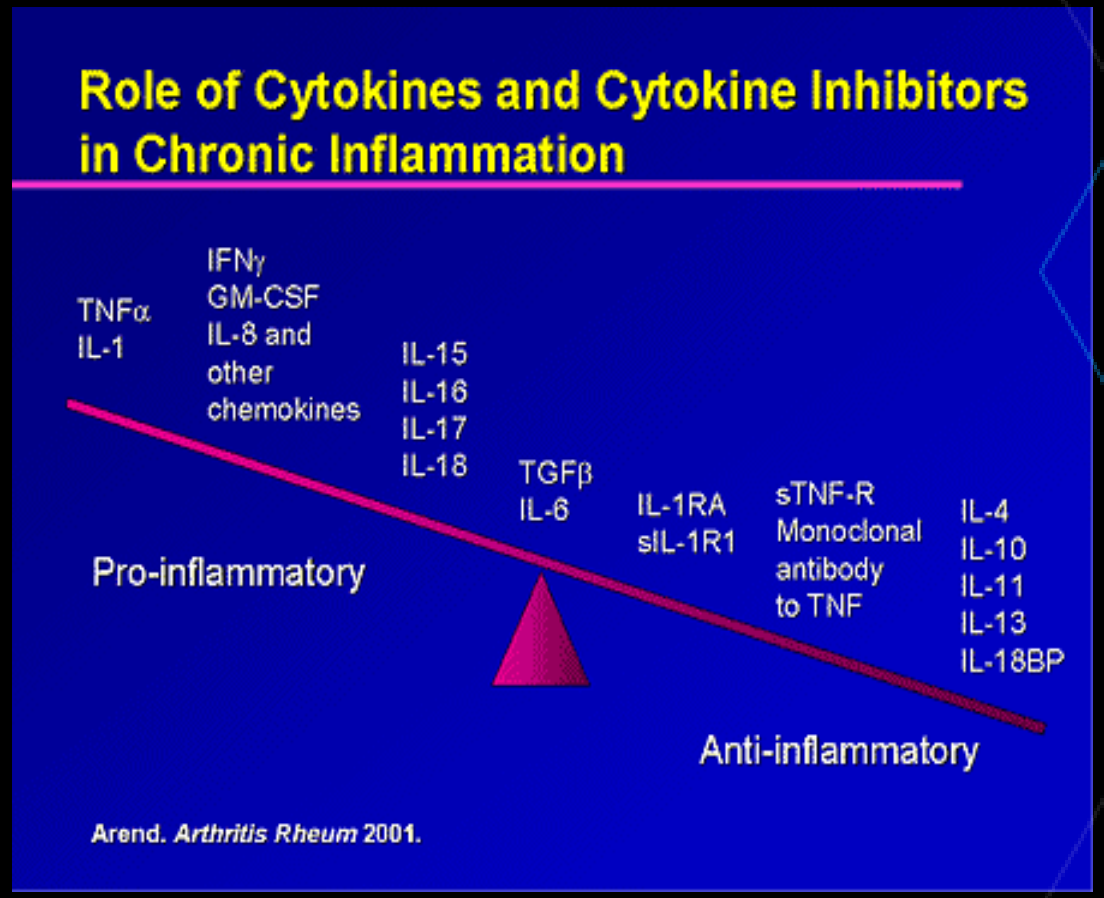
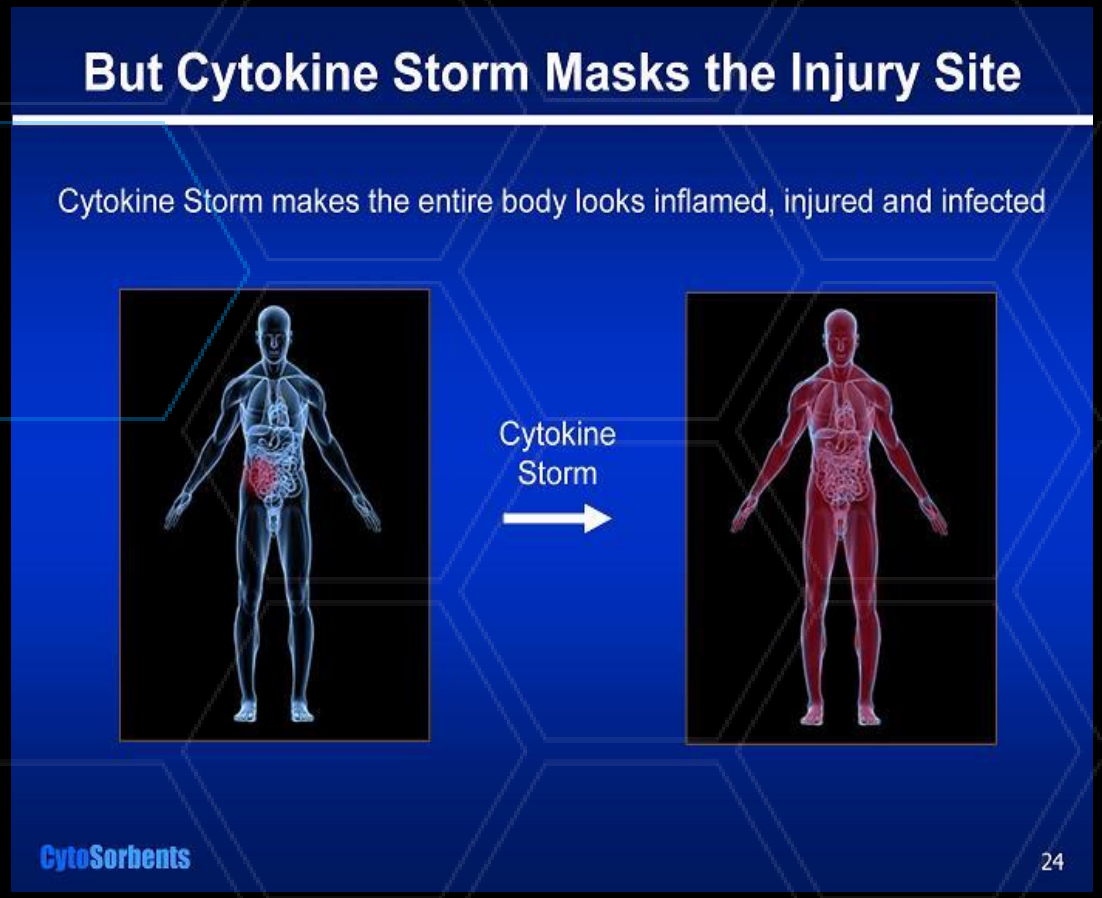
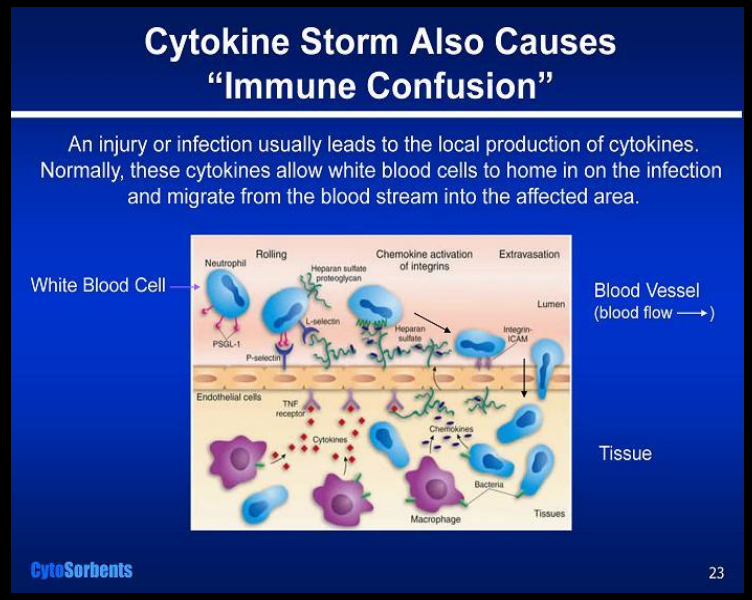
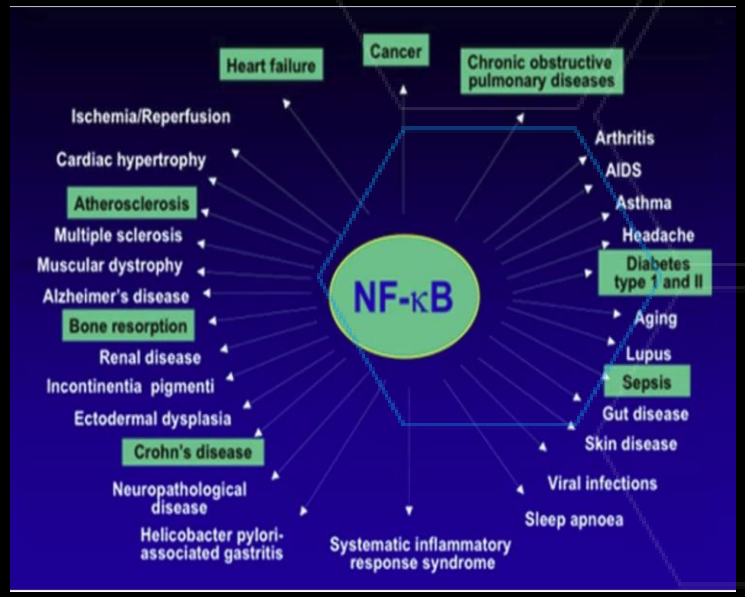
Cytokine Balance

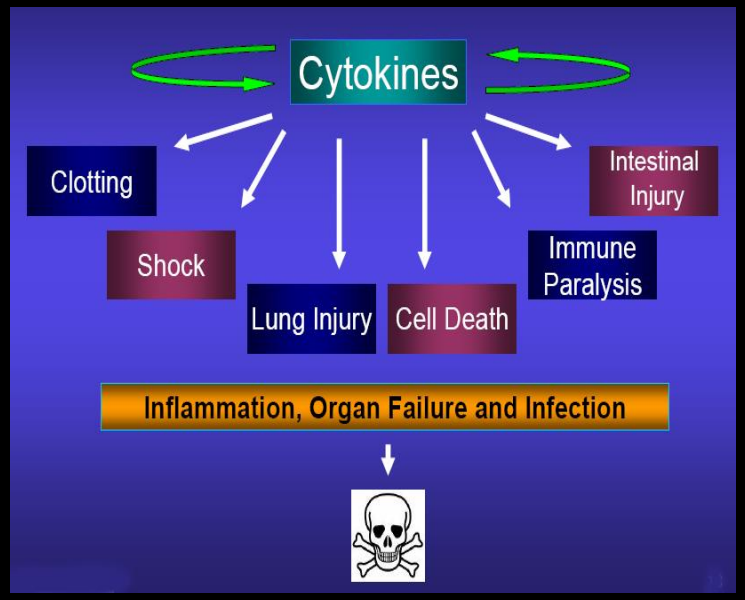


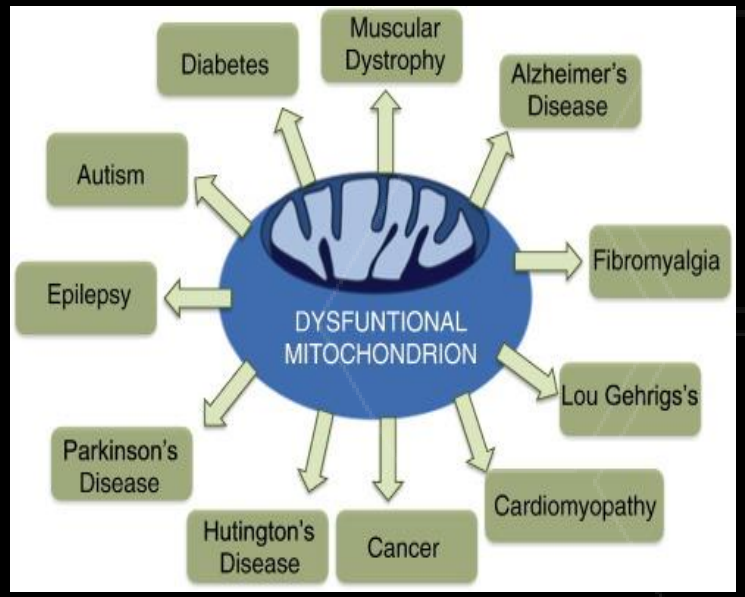


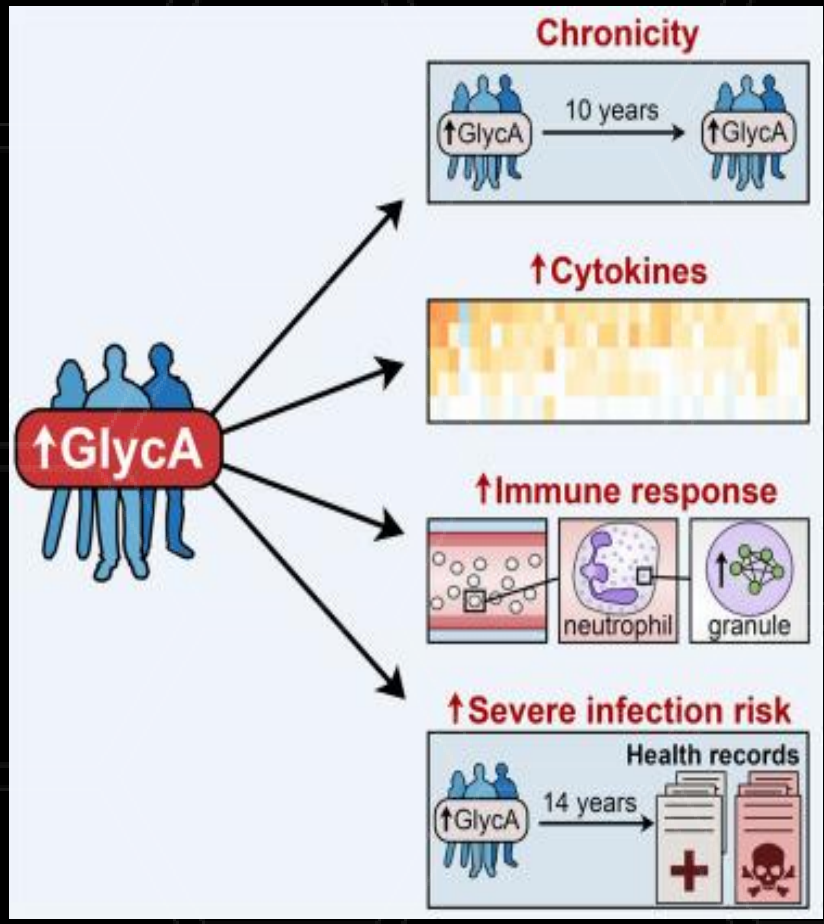
Cytokine Storm





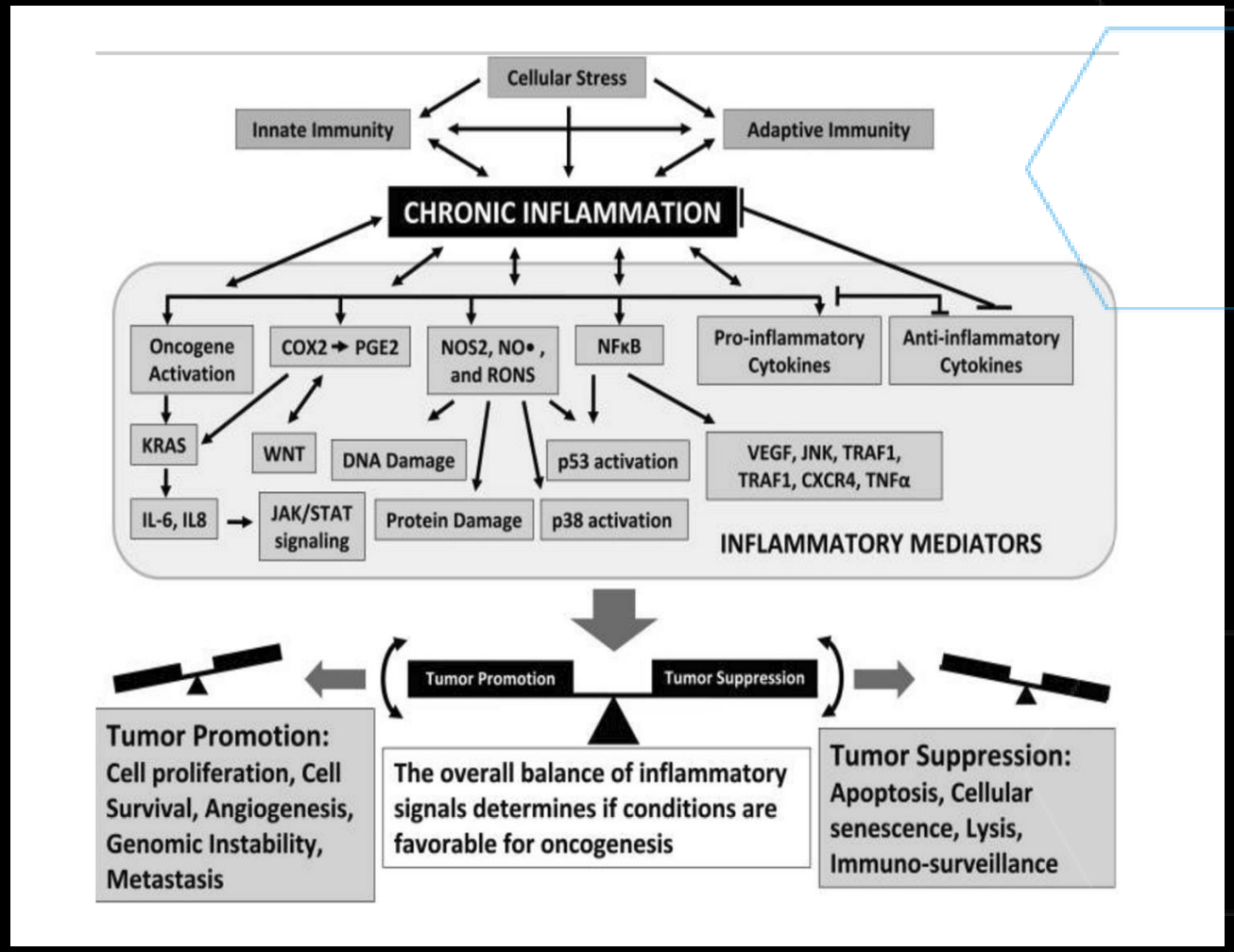


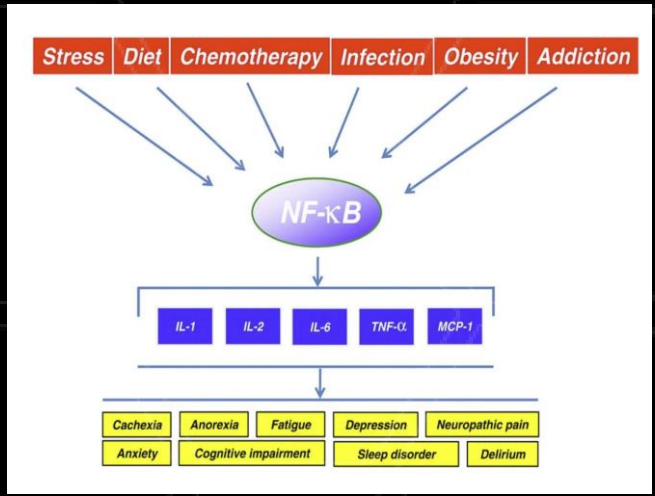


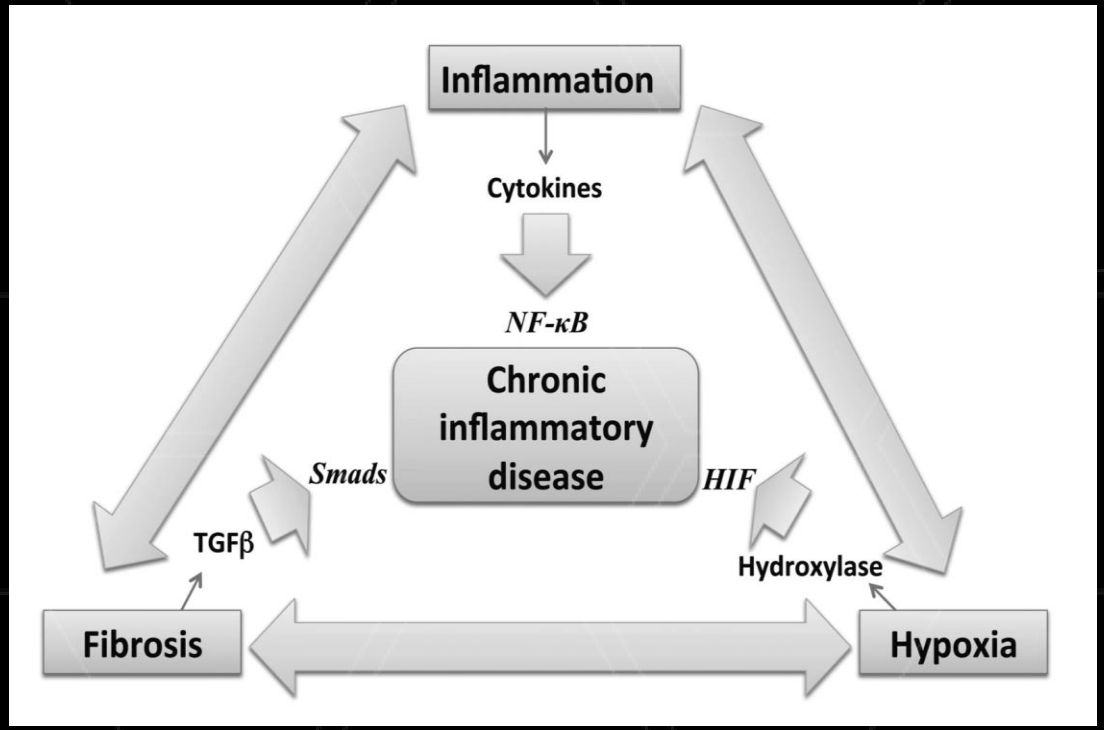


OXYMED

Cytokines Autophagy

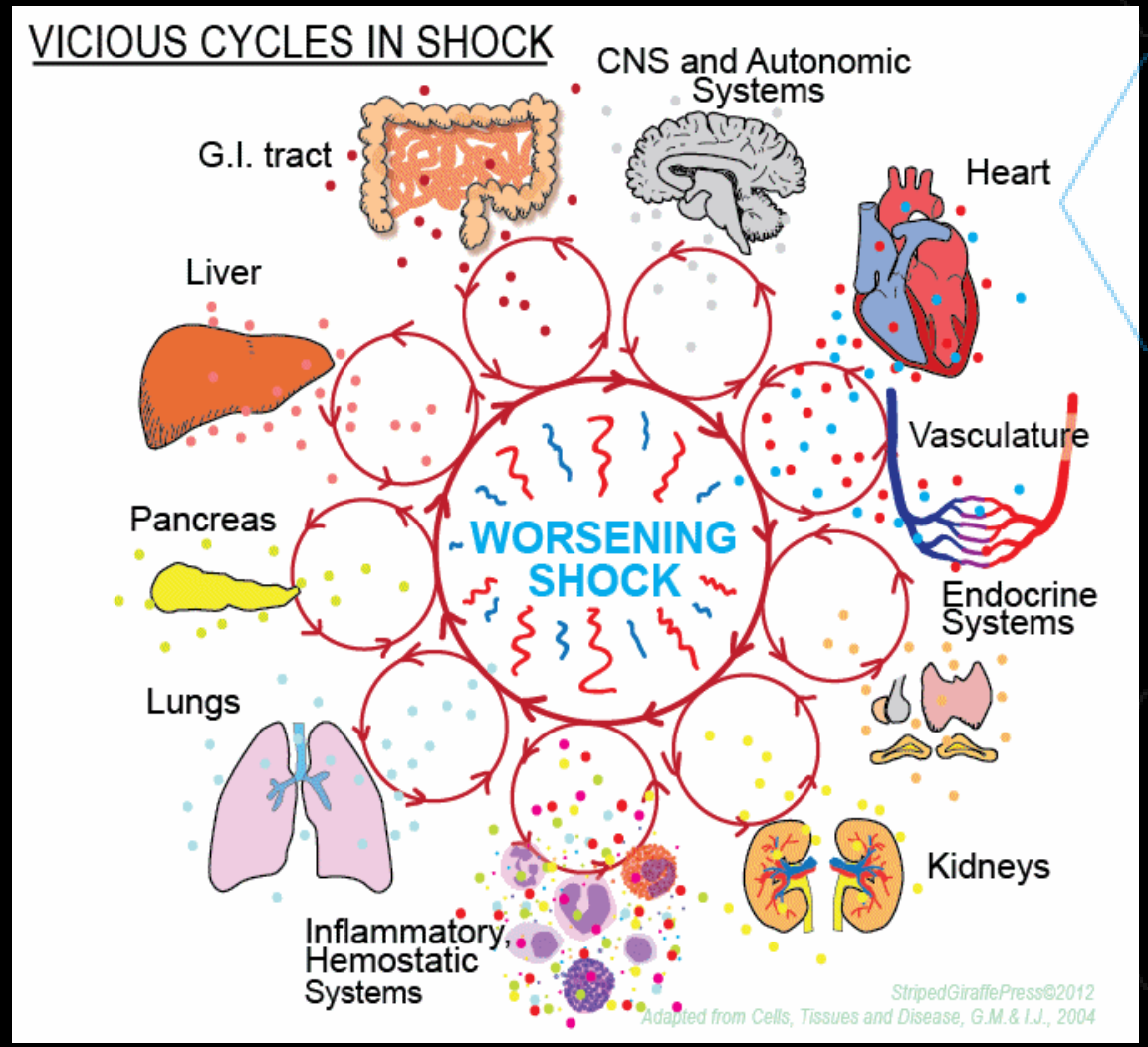






OXYMED

Cytokine Shock



Case 1: Athlete January 2016

OXYMED HEALTH

Date of Birth : 29-Apr-1978
Sex : M
Collected : 20-Jan-2016
C/O HYPERMED 643 CHAPEL STREET
SOUTH YARRA VIC 3141
Lab id: UR#:

HYPERMED
643 CHAPEL STREET
SOUTH YARRA VIC 3141

INTEGRATIVE MEDICINE

BLOOD - SERUM	Result	Range	Units
Hyperbaric Oxygen Therapy (HBO)	0.0		Hours
CYTOKINES,Extensive Panel			
ProInflammatory Cytokines (TH1)			
Interleukin 1	*161.0* *H	0.0 - 2.8	pg/mL
Interleukin 6	*35.0* *H	0.0 - 11.0	pg/mL
Interleukin 7	*23.3* *H	0.0 - 16.0	pg/mL
Interleukin 8	*>2500.0* *H	0.0 - 28.0	pg/mL
TNFa	*39.10* *H	0.00 - 13.00	pg/mL
TNFb	149.0	0.0 - 156.0	pg/mL
AntiInflammatory Cytokines (TH2)			
GM-CSF	77.7	0.0 - 80.0	pg/mL
Interleukin 2	6.8	0.0 - 10.0	pg/mL
Interleukin 4	*26.2* *H	0.0 - 19.0	pg/mL
Interleukin 5	1.6	0.0 - 13.0	pg/mL
Interleukin 10	*10.1* *H	0.0 - 7.0	pg/mL
Interleukin 12	7.5	0.0 - 14.0	pg/mL
Interleukin 13	*13.2* *H	0.0 - 6.0	pg/mL
INFg	*75.2* *H	0.0 - 28.0	pg/mL
TGFb	50.6	28.0 - 64.0	pg/mL

OXYMED

Case 1: Athlete January 2017

OXYMED HEALTH

Date of Birth : 29-Apr-1978
Sex : M
Collected : 24-Jan-2017
C/O HYPERMED 643 CHAPEL STREET
SOUTH YARRA VIC 3141
Lab id: UR#:

OXYMED
643 CHAPEL STREET
SOUTH YARRA VIC 3141

INTEGRATIVE MEDICINE

BLOOD - SERUM	Result	Range	Units
Hyperbaric Oxygen Therapy (HBO)			Hours
CYTOKINES,Extensive Panel			
ProInflammatory Cytokines (TH1)			
Interleukin 1	82.2 *H	0.0 - 2.8	pg/mL
Interleukin 6	137.6 *H	0.0 - 11.0	pg/mL
Interleukin 7	8.9	0.0 - 16.0	pg/mL
Interleukin 8	<0.6	0.0 - 28.0	pg/mL
Interleukin 17	6.9	< 13.0	pg/mL
TNFa	46.50 *H	0.00 - 13.00	pg/mL
TNFb	91.0	0.0 - 156.0	pg/mL
AntiInflammatory Cytokines (TH2)			
GM-CSF	298.2 *H	0.0 - 80.0	pg/mL
Interleukin 2	2.7	0.0 - 10.0	pg/mL
Interleukin 3	88.4 *H	< 5.0	pg/mL
Interleukin 4	<3.7	0.0 - 19.0	pg/mL
Interleukin 5	1.8	0.0 - 13.0	pg/mL
Interleukin 10	<3.0	0.0 - 7.0	pg/mL
Interleukin 12	3.1	0.0 - 14.0	pg/mL
Interleukin 13	10.9 *H	0.0 - 6.0	pg/mL
INFg	9.2	0.0 - 28.0	pg/mL
TGFb	44.0	28.0 - 64.0	pg/mL

OXYMED

Case 2: Athlete March 2018

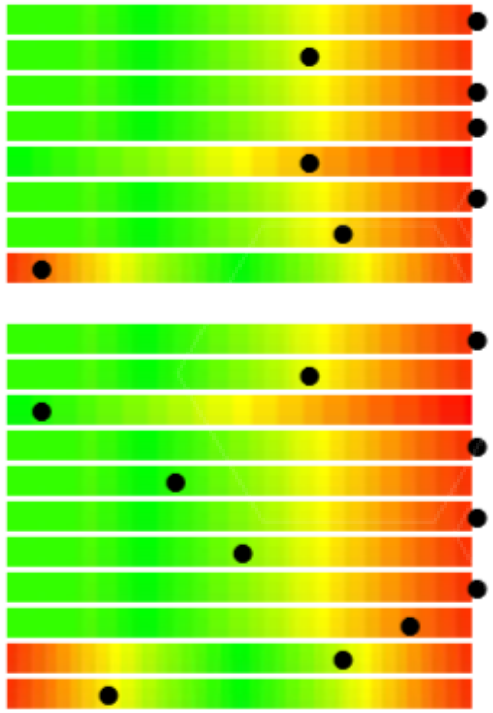
OXYMED HEALTH

Date of Birth : 25-May-1979
Sex : M
Collected : 20-Mar-2018
643 CHAPEL STREET
SOUTH YARRA VIC 3141
Lab id: UR#:

OXYMED, 643 CHAPEL STREET
SOUTH YARRA VIC 3141

INTEGRATIVE MEDICINE

BLOOD - SERUM	Result	Range	Units
Hyperbaric Oxygen Therapy (HBO)	0.0		Hours
CYTOKINES,Extensive Panel			
ProInflammatory Cytokines (TH1)			
Interleukin 1	*5.6* *H	0.0 - 2.8	pg/mL
Interleukin 6	7.9	0.0 - 11.0	pg/mL
Interleukin 7	*28.6* *H	0.0 - 16.0	pg/mL
Interleukin 8	*172.7* *H	0.0 - 28.0	pg/mL
Interleukin 17	*44.3* *H	< 13.0	pg/mL
TNFa	*19.10* *H	0.00 - 13.00	pg/mL
TNFb	139.0	0.0 - 156.0	pg/mL
S100B	*20.0* *L	60.0 - 100.0	pg/mL
AntiInflammatory Cytokines (TH2)			
GM-CSF	*448.5* *H	0.0 - 80.0	pg/mL
Interleukin 2	7.8	0.0 - 10.0	pg/mL
Interleukin 3	1.4	< 5.0	pg/mL
Interleukin 4	*104.0* *H	0.0 - 19.0	pg/mL
Interleukin 5	2.6	0.0 - 13.0	pg/mL
Interleukin 10	*56.3* *H	0.0 - 7.0	pg/mL
Interleukin 12	6.2	0.0 - 14.0	pg/mL
Interleukin 13	*22.1* *H	0.0 - 6.0	pg/mL
INFg	*32.3* *H	0.0 - 28.0	pg/mL
TGFb	60.8	28.0 - 64.0	pg/mL
Brain Derived Neurotrophic Factor BDNF	21.0	20.0 - 50.0	ng/mL



OXYMED

Case 2: Athlete May 2018

OXYMED HEALTH

Date of Birth : 25-May-1979
Sex : M
Collected : 21-May-2018

Lab id: UR#:

OXYMED, 643 CHAPEL STREET
SOUTH YARRA VIC 3141

INTEGRATIVE MEDICINE

BLOOD - SERUM	Result	Range	Units
Hyperbaric Oxygen Therapy (HBO)	90.0		Hours
CYTOKINES,Extensive Panel			
ProInflammatory Cytokines (TH1)			
Interleukin 1	2.5	0.0 - 2.8	pg/mL
Interleukin 6	5.7	0.0 - 11.0	pg/mL
Interleukin 7	16.0	0.0 - 16.0	pg/mL
Interleukin 8	19.0	0.0 - 28.0	pg/mL
Interleukin 17	*14.8* *H	< 13.0	pg/mL
TNFa	*15.90* *H	0.00 - 13.00	pg/mL
TNFb	109.0	0.0 - 156.0	pg/mL
S100B	*39.0* *L	60.0 - 100.0	pg/mL
AntiInflammatory Cytokines (TH2)			
GM-CSF	*200.4* *H	0.0 - 80.0	pg/mL
Interleukin 2	4.3	0.0 - 10.0	pg/mL
Interleukin 3	<1.0	< 5.0	pg/mL
Interleukin 4	<1.0	0.0 - 19.0	pg/mL
Interleukin 5	1.9	0.0 - 13.0	pg/mL
Interleukin 10	*21.8* *H	0.0 - 7.0	pg/mL
Interleukin 12	1.3	0.0 - 14.0	pg/mL
Interleukin 13	*12.5* *H	0.0 - 6.0	pg/mL
INFg	9.6	0.0 - 28.0	pg/mL
TGFb	30.0	28.0 - 64.0	pg/mL
Brain Derived Neurotrophic Factor BDNF	*65.0* *H	20.0 - 50.0	ng/mL

OXYMED

Case 3: Athlete Ketogenic Program

OXYMED HEALTH

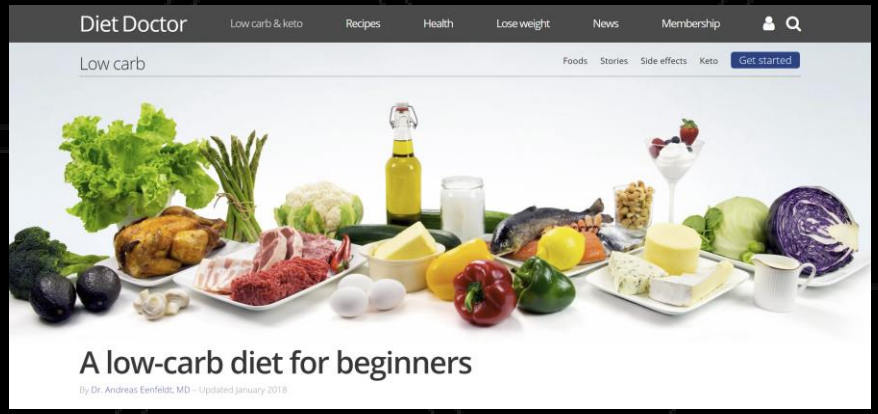
Date of Birth : 18-Sep-1976
Sex : M
Collected : 6/Jun/2018
Received: 07-Jun-2018

Lab id : 3542900 UR#:

OXYMED, 643 CHAPEL STREET
SOUTH YARRA VIC 3141

INTEGRATIVE MEDICINE

BLOOD - SERUM	Result	Range	Units
Hyperbaric Oxygen Therapy (HBO)	0.0		Hours
CYTOKINES,Extensive Panel			
ProInflammatory Cytokines (TH1)			
Interleukin 1	<1.0	0.0 - 2.8	pg/mL
Interleukin 6	1.2	0.0 - 11.0	pg/mL
Interleukin 7	14.4	0.0 - 16.0	pg/mL
Interleukin 8	12.2	0.0 - 28.0	pg/mL
Interleukin 17	2.2	< 13.0	pg/mL
TNFa	13.00	0.00 - 13.00	pg/mL
TNFb	93.0	0.0 - 156.0	pg/mL
S100B	*<10.0* *L	60.0 - 100.0	pg/mL
AntiInflammatory Cytokines (TH2)			
GM-CSF	*195.1* *H	0.0 - 80.0	pg/mL
Interleukin 2	1.3	0.0 - 10.0	pg/mL
Interleukin 3	1.5	< 5.0	pg/mL
Interleukin 4	<1.0	0.0 - 19.0	pg/mL
Interleukin 5	<1.0	0.0 - 13.0	pg/mL
Interleukin 10	*7.4* *H	0.0 - 7.0	pg/mL
Interleukin 12	1.7	0.0 - 14.0	pg/mL
Interleukin 13	3.5	0.0 - 6.0	pg/mL
INFg	4.3	0.0 - 28.0	pg/mL
TGFb	34.0	28.0 - 64.0	pg/mL
Brain Derived Neurotrophic Factor BDNF	*68.0* *H	20.0 - 50.0	ng/mL



OXYMED

Blood, Salvia, Urine

Diagnostics (Basel). 2017 Apr 9;7(2). pii: E21. doi: 10.3390/diagnostics7020021.

Salivary IL-8, IL-6 and TNF-α as Potential Diagnostic Biomarkers for Oral Cancer.

Sahibzada HA[1], Khurshid Z[2], Khan RS[3], Naseem M[4], Siddique KM[5], Mali M[6], Zafar MS[7,8].

Abstract

Saliva has been useful as a liquid biopsy for the diagnosis of various oral or systemic diseases, and oral squamous cell carcinoma (OSCC) is no exception.

- Salivary cytokines expression, specifically Interleukin-8 (IL-8), Interleukin-6 (IL-6) and Tumor necrosis factor (TNF-α), contribute to the pathogenesis of cancer and these cytokines serve as potential biomarkers.
- Their excessive production plays a role in cancer progression and establishment of angiogenesis. However, other inflammatory or immunological conditions may affect the levels of cytokines in saliva.

Apart from serum, the saliva-based test can be a cost-effective tool in the follow-up and diagnosis of OSCC. Moreover, large-scale investigations are still needed for the validation of salivary cytokines.

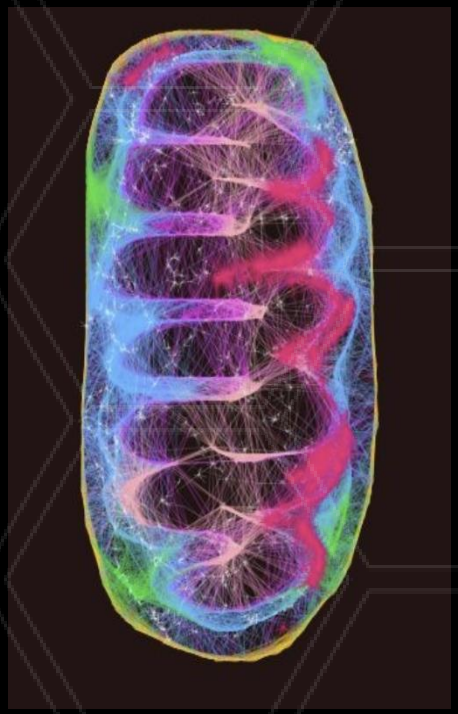
OXYMED

“Washout” – ‘Oxygen In Garbage out’

Adv Drug Deliv Rev. 2017 Feb 16. pii: S0169-409X(17)30028-5. doi: 10.1016/j.addr.2017.02.001. [Epub ahead of print]

Mimicking oxygen delivery and waste removal functions of blood.

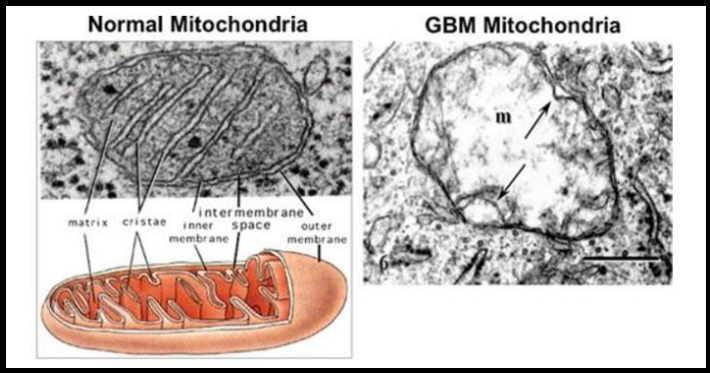
Zhang H[1], Barralet JE[2].

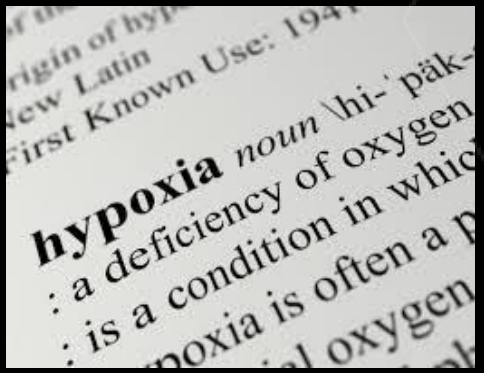


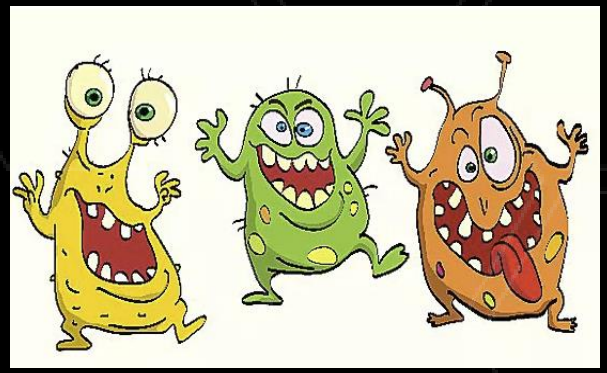
Oxygen In - Garbage Out - ‘Wash-out’ (Herxheimer's Effect)

Blood delivers oxygen to cells and tissues and removes metabolic wastes.
Oxygen is crucial for the long-term survival of tissues and cells in vertebrates.

- Metabolic waste accumulation is another issue in biological systems when blood flow is insufficient. Metabolic wastes change the microenvironment of cells and tissues, influence the metabolic activities of cells, and ultimately cause cell death.
- This review examines advances in blood mimicking systems in the field of biomedical engineering in terms of oxygen delivery and metabolic waste removal.



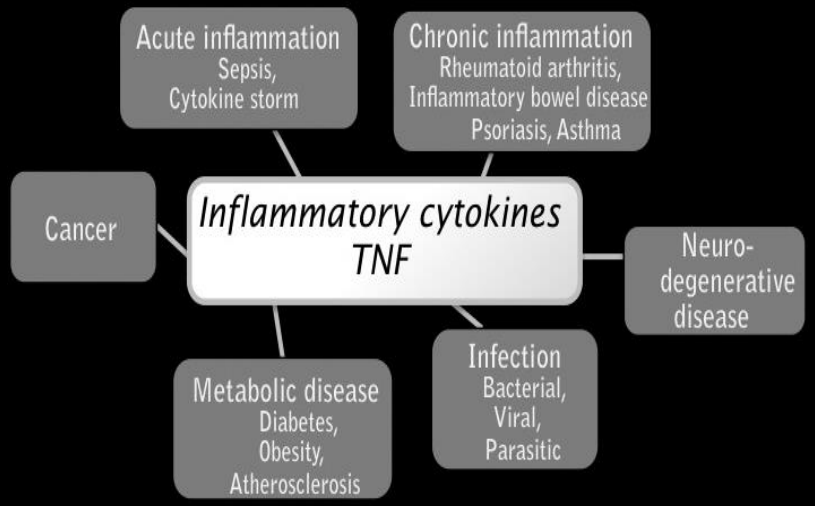


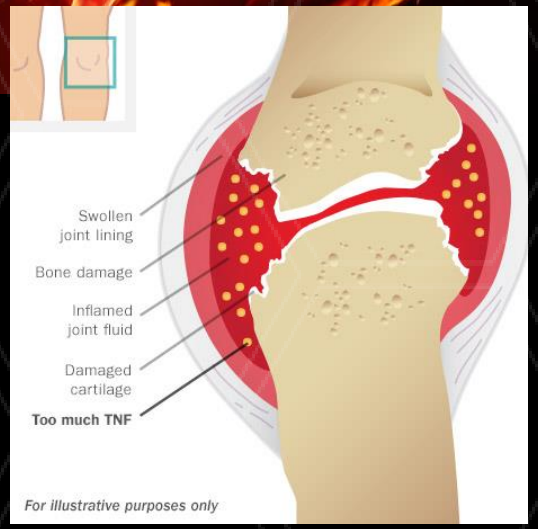


OXYMED

Tumor Necrosis Factor Alpha

- TNFα is a cytokine produced by white blood cells, released during the acute inflammatory response. It plays a pivotal role in systemic inflammation as it induces the synthesis of C-reactive protein, vasodilatation, and vascular permeability.
- Master regulator of the human inflammatory response.
- Linked with autoimmune disorders including chronic pain, arthritis, tendinitis.
- **Elevated TNFα in the brain hippocampus results in atrophy** and associated with traumatic brain injuries, post traumatic stress disorders, concussion syndrome and conditions depression, psychosis, addiction and dementia.
- Elevated pro-inflammatory cytokines (IL1, IL6, IL7, IL8, S100B) and TNFα are linked with chronic and progressive neurodegenerative disease - often referred to as **Cytokine Storm leading to multisystem inflammatory cascade** (autoimmune erosion). The body due to autoimmune dysfunction - attacks itself!
- Reason for many **'retired' athletes combating depression and other progressive mental health issues.**





OXYMED

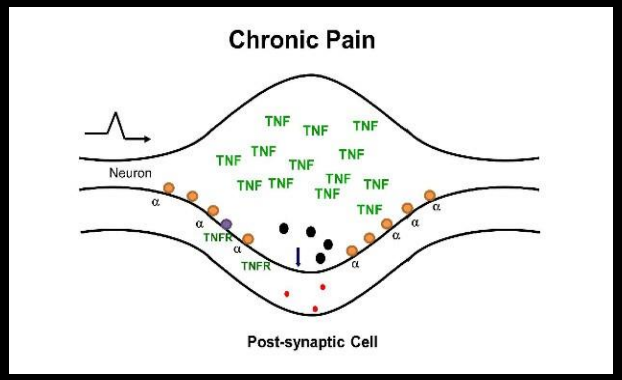
Tumor Necrosis Factor Alpha

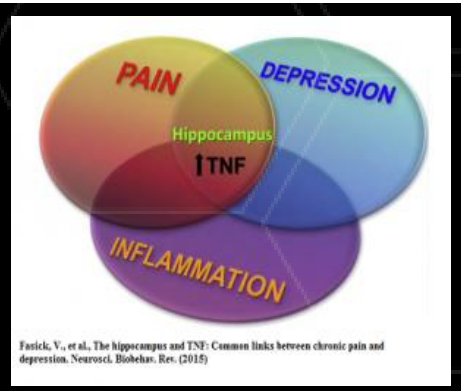
Pain. Author manuscript; available in PMC 2013 Sep 1.
Published in final edited form as:
Pain. 2012 Sep; 153(9): 1871–1882.
Published online 2012 Jul 4. doi: 10.1016/j.pain.2012.05.028

PMCID: PMC3417838
NIHMSID: NIHMS391887
PMID: 22770843

Increasing TNF Levels Solely in the Rat Hippocampus Produces Persistent Pain-like Symptoms

- This review discusses the interaction between depression and chronic pain, emphasizing the fundamental **role of the hippocampus** in the development and maintenance of both disorders. **Reduced CBF to the hippocampus results in TNFα overexpression** coupled with other proinflammatory chemokines.
- Hippocampus expressed TNFα serves as a therapeutic target for management of chronic pain and major depressive disorder.
- **A novel method of reducing TNF** - increasing BDNF levels.
- In animal subjected to **chronic stress and pain** demonstrated **elevated TNFα associated with decreased BDNF expression in the hippocampus.** Administration BDNF synthesis exerts antidepressant and analgesic effects on chronic pain via induction of BDNF in the brain.
- Increasing BDNF may be a novel treatment strategy for chronic pain associated with depression.
- **HBOT down regulates TNF and upregulates BDNF.**





OXYMED

Tumor Necrosis Factor Alpha - Etanercept

International Journal of
Molecular Sciences

MDPI

Review

A New Venue of TNF Targeting

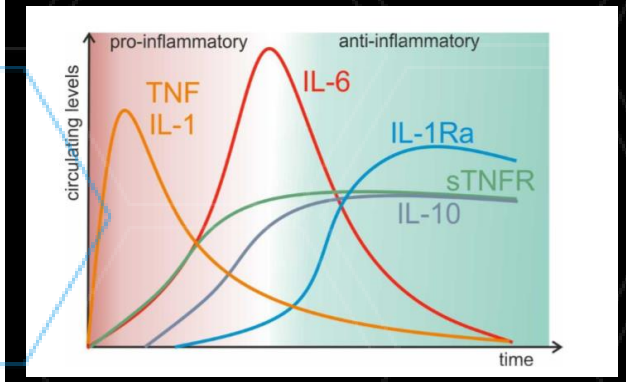
Sophie Steeland [1], Claude Libert [2] and Roosmarijn E. Vandenbroucke [1,*]

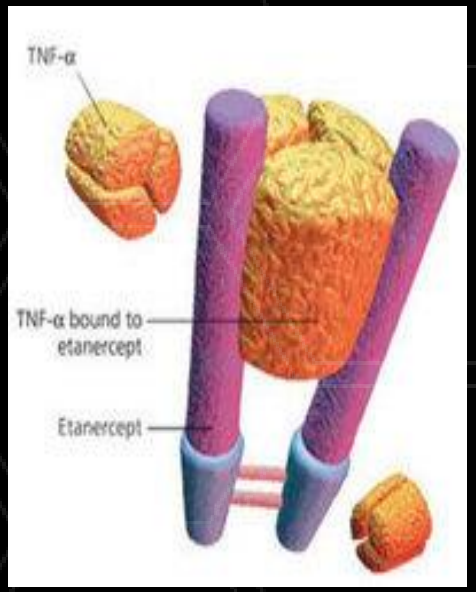
1 Barriers in Inflammation, VIB Center for Inflammation Research, Ghent, Department of Biomedical Molecular Biology, Ghent University, 9052 Ghent, Belgium; Sophie.Steeland@irc.VIB-ugent.be
2 Mouse Genetics in Inflammation, VIB Center for Inflammation Research, Ghent, Department of Biomedical Molecular Biology, Ghent University, 9052 Ghent, Belgium; Claude.Libert@irc.VIB-ugent.be
* Correspondence: Roosmarijn.Vandenbroucke@irc.VIB-ugent.be; Tel.: +32-9-331-35-87

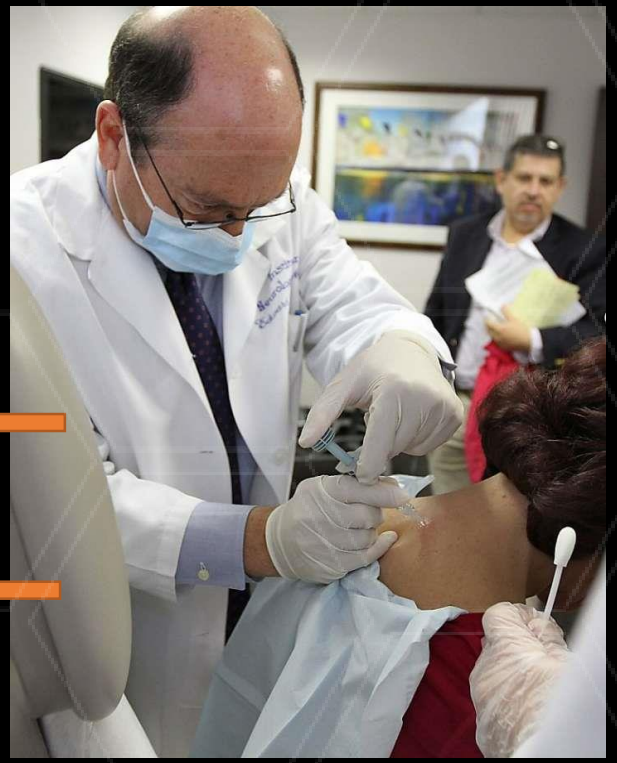
Received: 31 March 2018; Accepted: 3 May 2018; Published: 11 May 2018

check for updates

Abstract: The first Food and Drug Administration-(FDA)-approved drugs were small, chemically-manufactured and highly active molecules with possible off-target effects, followed by protein-based medicines such as antibodies. Conventional antibodies bind a specific protein and are becoming increasingly important in the therapeutic landscape. A very prominent class of biologicals are the anti-tumor necrosis factor (TNF) drugs that are applied in several inflammatory diseases that are characterized by dysregulated TNF levels. Marketing of TNF inhibitors revolutionized the treatment of diseases such as Crohn's disease. However, these inhibitors also have undesired effects, some of them directly associated with the inherent nature of this drug class, whereas others are linked with their mechanism of action, being pan-TNF inhibition. The effects of TNF can diverge at the level of TNF format or receptor, and we discuss the consequences of this in sepsis, autoimmunity and neurodegeneration. Recently, researchers tried to design drugs with reduced side effects. These include molecules with more specificity targeting one specific TNF format or receptor, or that neutralize TNF in specific cells. Alternatively, TNF-directed biologicals without the typical antibody structure are manufactured. Here, we review the complications related to the use of conventional TNF inhibitors, together with the anti-TNF alternatives and the benefits of selective approaches in different diseases.







OXYMED

TNFa vs BDNF

Neural Regen Res. 2018 Oct;13(10):1693-1704. doi: 10.4103/1673-5374.238604.

Depression following a traumatic brain injury: uncovering cytokine dysregulation as a pathogenic mechanism.

Bodnar CN[1], Morganti JM[2], Bachstetter AD[1].

Author information

Abstract

A substantial number of individuals have long-lasting adverse effects from a traumatic brain injury (TBI). Depression is one of these long-term complications that influences many aspects of life. Depression can limit the ability to return to work, and even worsen cognitive function and contribute to dementia. The mechanistic cause for the increased depression risk associated with a TBI remains to be defined. As TBI results in chronic neuroinflammation, and priming of glia to a secondary challenge, the inflammatory theory of depression provides a promising framework for investigating the cause of depression following a TBI. Increases in cytokines similar to those seen in depression in the general population are also increased following a TBI. Biomarker levels of cytokines peak within hours-to-days after the injury, yet pro-inflammatory cytokines may still be elevated above physiological levels months-to-years following TBI, which is the time frame in which post-TBI depression can persist. As tumor necrosis factor α and interleukin 1 can signal directly at the neuronal synapse, pathophysiological levels of these cytokines can detrimentally alter neuronal synaptic physiology. The purpose of this review is to outline the current evidence for the inflammatory hypothesis of depression specifically as it relates to depression following a TBI. Moreover, we will illustrate the potential synaptic mechanisms by which tumor necrosis factor α and interleukin 1 could contribute to depression. The association of inflammation with the development of depression is compelling; however, in the context of post-TBI depression, the role of inflammation is understudied. This review attempts to highlight the need to understand and treat the psychological complications of a TBI, potentially by neuroimmune modulation, as the neuropsychiatric disabilities can have a great impact on the rehabilitation from the injury, and overall quality of life.

J Stroke Cerebrovasc Dis. 2018 Aug 24. pii: S1052-3057(18)30356-2. doi: 10.1016/j.jstrokecerebrovasdis.2018.06.032. [Epub ahead of print]

Brain-Derived Neurotrophic Factor Levels are Lower in Chronic Stroke Patients: A Relation with Manganese-dependent Superoxide Dismutase ALA16VAL Single Nucleotide Polymorphism through Tumor Necrosis Factor-α and Caspases Pathways.

OXYMED

IL1

J Transl Med. 2009; 7: 97.
Published online 2009 Nov 17. doi: 10.1186/1479-5876-7-97
PMCID: PMC2780998

Inflammatory mechanisms in ischemic stroke: therapeutic approaches

- IL1 is linked with systemic inflammation including the **'gut and brain connection'.**
- Patients with chronic irritable bowel and chronic disease are often elevated with IL1 and IL8.
- In stroke patients, IL1 caused a severe reduction in cerebral blood flow and an increase in infarct volume. Blockade of endothelin-1 receptors reversed this hypoperfusion, reduced tissue damage, and improved functional outcome.
- **Post stroke, IL1 mediates inflammatory effects** (in red) – **negative cascade** - including **increased adhesion molecules, neutrophil infiltration, reduced BBB integrity, decreased blood flow.**
- Elevated IL1 on astrocytes reveal increased IL6, TNFa and other chemokines.
- Elevated IL1 inhibits stroke repair – **reduced neurogenesis.**
- Elevations in serum levels and joint fluids (synovial fluids) are detected in **rheumatoid arthritis.**

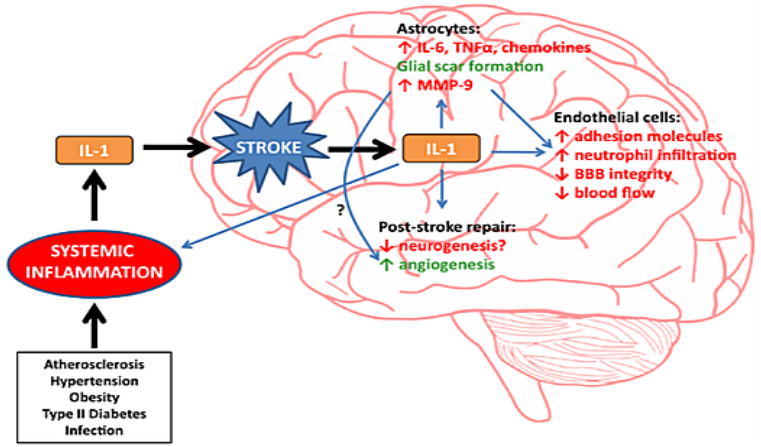


Figure. Mechanisms of interleukin-1 (IL-1) action in stroke. Stroke-related comorbidities and risk factors are associated with a raised systemic inflammatory profile, mediated in part by IL-1. Post-stroke increases in IL-1 in the brain mediate detrimental (indicated in red) inflammatory processes in the acute phase that contribute to worse outcome. In the subacute and chronic phase after stroke, certain actions of IL-1 may be beneficial (indicated in green). Figure provided and used with permission courtesy of Christopher Hoyle, University of Manchester, United Kingdom. MMP indicates matrix metalloproteinases; and TNF, tumor necrosis factor.

OXYMED

IL6

Published online 2014 Oct 13. doi: 10.1016/j.ygeno.2014.10.003

Whole Blood Gene Expression and Interleukin-6 Levels

Shock. 2013 Dec; 40(6): 471–475.
doi: 10.1097/SHK.0000000000000037

INTERLEUKIN 6 MEDIATES NEUROINFLAMMATION AND MOTOR COORDINATION DEFICITS AFTER MILD TRAUMATIC BRAIN INJURY AND BRIEF HYPOXIA IN MICE

Atherosclerosis. 2018 Aug 24. pii: S0021-9150(18)31247-4. doi: 10.1016/j.atherosclerosis.2018.07.037. [Epub ahead of print]

Corrigendum to "Interleukin-6 is an independent predictor of progressive atherosclerosis in the carotid artery: The Tromsø study" [Atherosclerosis 271 (April 2018) 1-8].

- IL6 is an important biomarker in monitoring inflammatory responses. IL-6 is involved in the induction of acute phase responses and induction of fever.
- Elevated serum levels of IL6 are also found in patients with chronic inflammatory arthritis and traumatic arthritis. IL6 is a cytokine with a wide variety of biological functions. It is a potent lymphoid cell growth factor that stimulates the growth and survivability of certain B cells and T cells. It plays an essential role in the final differentiation of b-cells into Ig-secreting cells, it induces myeloma and plasmacytoma growth, it induces nerve cells differentiation and in hepatocytes it induces acute phase reactants.
- IL6 can increase up to a 1,000-fold during trauma and infection. Chronic elevation of serum IL-6 is associated with the progression of atherosclerosis in patients with vascular risk factors.
- Elevated IL6 but not CRP in midlife, predicts cognitive decline and dementia.
- IL6 elevation associated with chronic lumbar radicular pain. Persistent increase of the pro-inflammatory substances IL6 and IL8 in serum after disc herniation.

- IL6 is a growth and survival factor in human glioblastoma cells and plays an important role in malignant progression. Its increased levels have been associated with elevated cancer risk, and also these levels have been found to be a prognostic factor for several cancer types. In addition, increased levels have been found in coronary heart disease, insulin resistant patients, advance stage cancer patients, atopy/asthma and in patients with blood circulating micro metastasis (circulating tumour cells).

OXYMED

IL7 - promotes tumour development and progression

Interleukin-7 (IL-7) and IL-7 receptor (IL-7R) signalling complex in human solid tumours

M.A.A. Al-Rawi, R.E. Mansel and W.G. Jiang
Metastasis Research Group, University Department of Surgery, University of Wales College of Medicine, Heath Park, Cardiff, UK

Interleukin–7: from bench to clinic

Terry J. Fry and Crystal L. Mackall

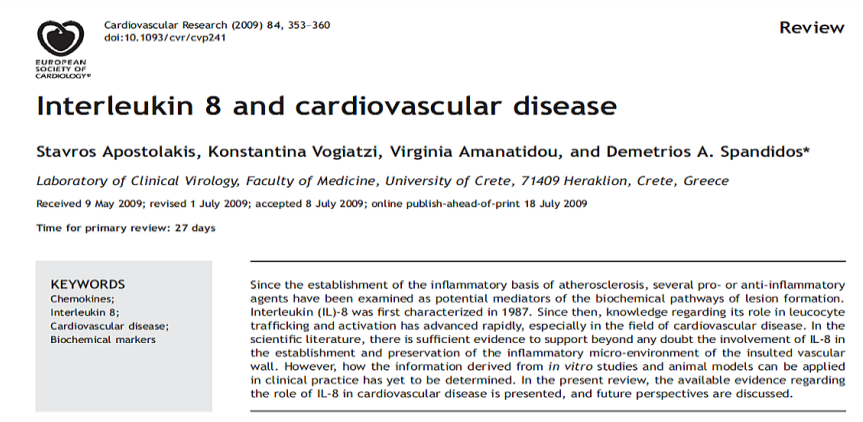
Blood 2002 99:3892–3904; doi: https://doi.org/10.1182/blood.V99.11.3892

- Hematopoietic growth factor secreted by red marrow and thymus.
- Stimulates the differentiation of multipotent (pluripotent) hematopoietic stem cells.
- Elevated levels detected in the plasma of HIV-infected patients.
- Elevated IL7 promotes tumour development and progression.
- Linked with NFkB in **malignancies (acute lymphoblastic leukemia).**
- Proliferative and trophic cytokine that induces the development and proliferation of haematopoietic cells and malignancies.
- The production of IL7 linked in the process of tumourgenesis upregulated in several solid tumours including breast, lung, prostate, renal, ovarian, melanomas as well as head and neck tumours.

- An important marker in **cancer activity**.

OXYMED

IL8 - Cardiovascular Disease



Cardiovascular Research (2009) 84, 353–360
doi:10.1093/cvr/cvp241

Review

Interleukin 8 and cardiovascular disease

Stavros Apostolakis, Konstantina Vogiatzi, Virginia Amanatidou, and Demetrios A. Spandidos*

Laboratory of Clinical Virology, Faculty of Medicine, University of Crete, 71409 Heraklion, Crete, Greece

Received 9 May 2009; revised 1 July 2009; accepted 8 July 2009; online publish-ahead-of-print 18 July 2009

Time for primary review: 27 days

KEYWORDS
Chemokines;
Interleukin 8;
Cardiovascular disease;
Biochemical markers

Since the establishment of the inflammatory basis of atherosclerosis, several pro- or anti-inflammatory agents have been examined as potential mediators of the biochemical pathways of lesion formation. Interleukin (IL)-8 was first characterized in 1987. Since then, knowledge regarding its role in leucocyte trafficking and activation has advanced rapidly, especially in the field of cardiovascular disease. In the scientific literature, there is sufficient evidence to support beyond any doubt the involvement of IL-8 in the establishment and preservation of the inflammatory micro-environment of the insulted vascular wall. However, how the information derived from *in vitro* studies and animal models can be applied in clinical practice has yet to be determined. In the present review, the available evidence regarding the role of IL-8 in cardiovascular disease is presented, and future perspectives are discussed.

Interleukin 8 and cardiovascular disease FREE

Stavros Apostolakis, Konstantina Vogiatzi, Virginia Amanatidou, Demetrios A. Spandidos

Cardiovascular Research, Volume 84, Issue 3, 1 December 2009, Pages 353–360,

- IL8 was first characterized in 1987.
- Identified with systemic inflammation of the blood and involved in cerebrovascular disorders and cardiovascular disease.
- IL8 and other chemokines are produced in several tissues upon infection, inflammation, ischemia, trauma etc - main cause of local neutrophil accumulation.
- Gross overproduction of IL8 from Endothelial cells in presence of hypoxia.
- Chronic systemic inflammation including: Progressive vascular disease, atherosclerosis lesions, main source for atherosclerosis plagues, predictive biomarker for ischemia induced oxidative stress.
- Pathogenesis of hypertension, and in the progression of ischemic induced necrosis.
- IL8 elevated in ventricular fibrillation complicating myocardial infarction.
- IL8 is a powerful independent predictive factor for cardiovascular disease and overall mortality in patients with end stage renal disease. Biomarker of outcome following cardiopulmonary arrest.
- SPECT Imaging (Single Photon Emission Computed Tomography) demonstrates regions of cerebral hypoperfusion. Typically, these are the regions of the brain associated with chronic ‘over expression’ of pro-inflammatory cytokines including IL1, IL8, TNFa, S100B and lowered BDNF.

OXYMED

IL8 – Neurodegenerative disorders

- IL8 linked with neuroinflammation associated with activated microglia resulting in neurotoxicity in the inflamed brain.
- IL8 are significantly elevated in neurodegenerative disease.
- Activated microglia increased levels of IL8 which contribute to a positive feedback process amplifying and sustaining inflammatory reactivity in Alzheimer's Disease brain.
- Chronic microglial activation is associated with sustained cellular production of a milieu of inflammatory mediators including pro-inflammatory cytokines including IL8, reactive oxygen species and matrix metalloproteinases which cause abnormalities to blood vessels (weak VEGF) and neurotoxicity.

Alzheimers Dement. 2018 Aug 8. pii: S1552-5260(18)33035-8. doi: 10.1016/j.jalz.2018.06.2857. [Epub ahead of print]

Blood-brain barrier breakdown, neuroinflammation, and cognitive decline in older adults.

Front Immunol. 2018 Jul 30;9:1767. doi: 10.3389/fimmu.2018.01767. eCollection 2018.

Adipocytes Promote Early Steps of Breast Cancer Cell Dissemination *via* Interleukin-8.

Curr Neurovasc Res. 2018 Jul 17. doi: 10.2174/1567202615666180717161807. [Epub ahead of print]

Pivotal pathogenic and biomarker role of Chlamydia Pneumoniae in neurovascular diseases.

OXYMED

IL8 – Hypoxia Cancer Proliferation

Hyperbaric Oxygen: Does it promote growth or recurrence of malignancy?

J. FELDMEIER[1], U. CARL[2], K. HARTMANN[3], P. SMINIA[4].

[1]Radiation Oncology Department, Medical College of Ohio, Toledo, OH, USA;[2]Department of Radiation Oncology and Nuclear Medicine, Diakoniekrankenhaus Rotenberg, Germany; [3]Department of Radiation Oncology, Heinrich Heine University, Duesseldorf, Germany; [4]Department of Radiation Oncology, VU University Medical Center, The Netherlands

iii. Interleukin-8 release is increased by hypoxia[68] This has been demonstrated in human glioblastoma cells in culture. IL-8 has been shown to have angiogenic properties in this model. The work of Shi and associates[69] confirms an increase in IL-8 by hypoxia and acidosis and suggest this contributes significantly to the aggressive biology of pancreatic cancer.

- Hypoxia, IL8 and acidity contributes to the aggressive biology of pancreatic cancer growth (UHMS 2003)

Vaccines (Basel). 2016 Jun 24;4(3). pii: E22. doi: 10.3390/vaccines4030022.

The IL-8/IL-8R Axis: A Double Agent in Tumor Immune Resistance.

David JM[1], Dominguez C[2], Hamilton DH[3], Palena C[4].

- Tumorigenesis analysis proved that patients with higher serum IL-8 levels grew faster than those with lower IL-8 levels.

Arterioscler Thromb Vasc Biol. 2017 Oct;37(10):1819-1827. doi: 10.1161/ATVBAHA.117.309794. Epub 2017 Aug 3.

Clots Are Potent Triggers of Inflammatory Cell Gene Expression: Indications for Timely Fibrinolysis.

J Pediatr. 2017 Jan;180:116-123.e1. doi: 10.1016/j.jpeds.2016.09.054. Epub 2016 Oct 24.

Circulating Inflammatory-Associated Proteins in the First Month of Life and Cognitive Impairment at Age 10 Years in Children Born Extremely Preterm.

OXYMED

IL17 – tendinopathy, rotator cuff injuries

SCIENTIFIC REPORTS

OPEN **IL-17A mediates inflammatory and tissue remodelling events in early human tendinopathy**

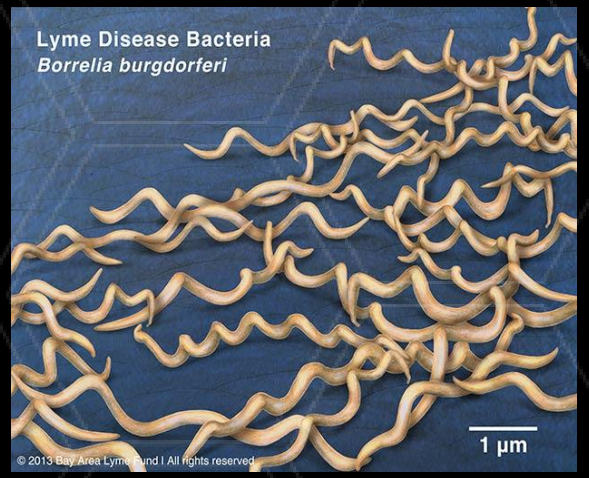
IL17A interleukin 17A [*Homo sapiens* (human)]

Gene ID: 3605, updated on 13-Aug-2017

J Leukoc Biol. 2016 Sep;100(3):589-98. doi: 10.1189/jlb.4A0715-331R. Epub 2016 Apr 21.

Autophagy suppresses host adaptive immune responses toward Borrelia burgdorferi.

Buffen K[1], Oosting M[1], Li Y[2], Kanneganti TD[3], Netea MG[1], Joosten LA[4].



- High levels of this cytokine IL17 are associated with several **chronic inflammatory diseases** including rheumatoid arthritis, psoriasis and multiple sclerosis.
- IL17 is an inflammatory infiltrate in **tendinopathy, rotator cuff injuries and repetitive strain injuries**. Increased levels of **Il17 coupled with TNF-α, IL-6** in torn supraspinatus promoting tissue destruction and degeneration during inflammation.
- IL17 has been linked with inflammatory arthritis and more recently associated with chronic symptoms associated with Lyme like illness.
- High levels of IL17 have been found in patients with confirmed, severe, **chronic 'borreliosis'** in conjunction with elevated IL1, IL6, TNFa.
- **Oral high dose vitamin D intake** reducing IL17 levels in **MS patients** in a double blind randomized clinical trial. 94 patients with a diagnosis of relapsing remitting multiple sclerosis (RRMS). 50,000 IU vitamin D3 every five days for 12 weeks showed significant reduction change in RRMS patients.

Curr Rheumatol Rep. 2016 Jun;18(6):33. doi: 10.1007/s11926-016-0585-9.

The Bench-to-Bedside Story of IL-17 and the Therapeutic Efficacy of its Targeting in Spondyloarthritis.

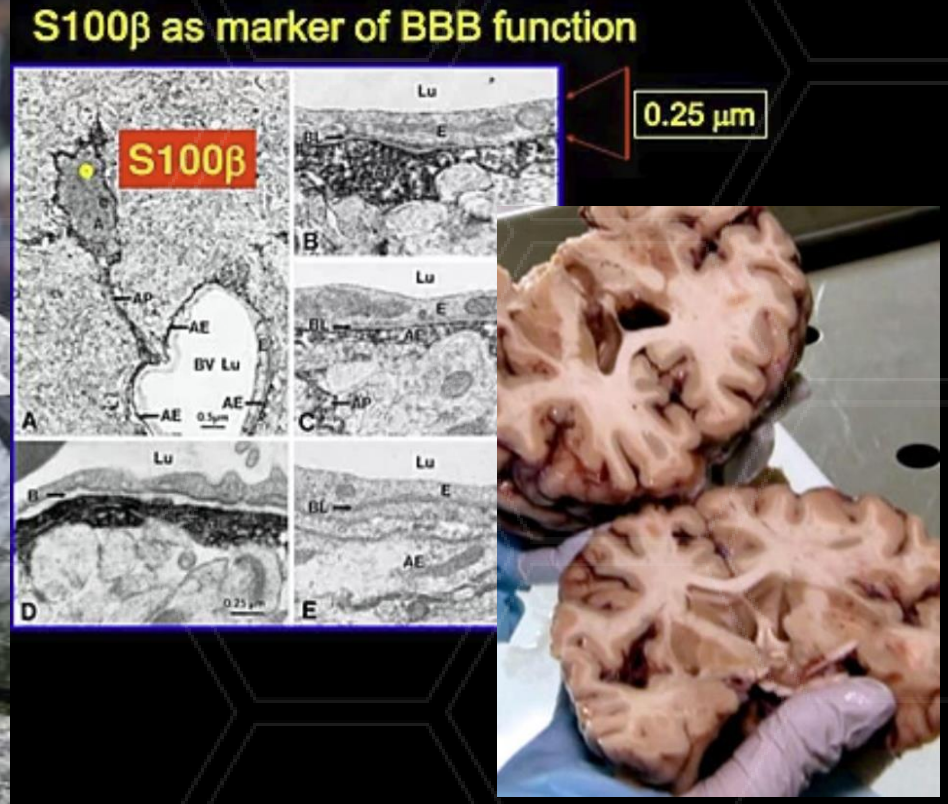
J Neuroimmunol. 2015 Aug 15;285:125-8. doi: 10.1016/j.jneuroim.2015.05.022. Epub 2015 Jun 12.

Effect of high dose vitamin D intake on interleukin-17 levels in multiple sclerosis: a randomized, double-blind, placebo-controlled clinical trial.

OXYMED

S100B – Astrocyte Neurotrophic Cytokine

- S100B is a **neurobiochemical marker of brain damage** and used as a measure of BBB dysfunction.
- Elevated with mental health issues including dementia, Alzheimer's but also a marker of other systemic issues including circulatory arrest, stroke and traumatic brain injury.
- Elevated with **Chronic Traumatic Brain Injury** (TBI and CTE), Post Traumatic Stress Disorders (PTSD), shock blast injury, concussions syndromes, blunt head injury and progressive neurodegeneration disorders



OXYMED

S100B and the Blood Brain Barrier

Neurol Sci. 2016 Apr;37(4):533-9. doi: 10.1007/s10072-016-2521-1. Epub 2016 Feb 29.

Re-exposure to the hypobaric hypoxic brain injury of high altitude: plasma S100B levels and the possible effect of acclimatisation on blood-brain barrier dysfunction.

Winter CD[1,2], Whyte T[3], Cardinal J[4,5], Kenny R[6], Ballard E[7].

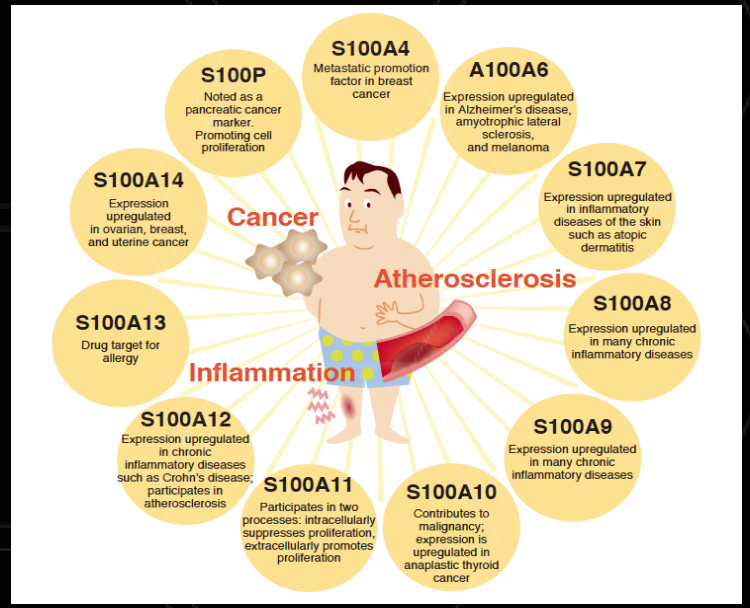
- Hypobaric hypoxic brain injury results in elevated peripheral S100B levels which may relate to blood-brain barrier (BBB) dysfunction.

Curr Med Chem. 2016;23(15):1571-96.

Identifying S100B as a Biomarker and a Therapeutic Target For Brain Injury and Multiple Diseases.

Chong ZZ[1], Changyaleket B, Xu H, Dull RO, Schwartz DE.

- S100B has attracted great attention as a biomarker for a variety of diseases.
- S100B is mainly expressed in glial cells.
- The levels of S100B in the blood may function to predict the progress or the prognosis of many kinds of diseases, such as cerebrovascular diseases, neurodegenerative diseases, motor neuron diseases, traumatic brain injury, schizophrenia, depression, diabetes mellitus, myocardial infarction, cancer, and infectious diseases.
- S100B has been implicated in the pathological process of these diseases, S100B should not be simply regarded as a biomarker, it may also function as therapeutic target for these diseases.
- The role of S100B may formulate innovative therapeutic strategies for multiple diseases.



OXYMED

S100B – neuroinflammation regardless of location

J Neurochem. 2018 Aug 24. doi: 10.1111/jnc.14574. [Epub ahead of print]

The S100B story: From biomarker to active factor in neural injury.

Michetti F[1,2], D'Ambrosi N[3], Toesca A[1], Puglisi MA[1], Serrano A[1], Marchese E[1], Corvino V[1], Geloso MC[1].

Author information

Abstract

S100B is a Ca^{2+} -binding protein mainly concentrated in astrocytes. Its levels in biological fluids (cerebrospinal fluid, peripheral and cord blood, urine, saliva, amniotic fluid) are recognized as a reliable biomarker of active neural distress. Although the wide spectrum of diseases in which the protein is involved (acute brain injury, neurodegenerative diseases, congenital/perinatal disorders, psychiatric disorders) reduces its specificity, its levels remain an important aid in monitoring the trend of the disorder. Mounting evidence now points to S100B as a Damage Associated Molecular Pattern molecule which, when released at high concentration, through its Receptor for Advanced Glycation Endproducts, triggers tissue reaction to damage in a series of different neural disorders. This review addresses this novel scenario, presenting data indicating that S100B levels and/or distribution in the nervous tissue of patients and/or experimental models of different neural disorders, for which the protein is used as a biomarker, are directly related to the progress of the disease: acute brain injury (ischemic/haemorrhagic stroke, traumatic injury), neurodegenerative diseases (Alzheimer's disease, Parkinson's disease, amyotrophic lateral sclerosis, multiple sclerosis), congenital/ perinatal disorders (Down syndrome, spinocerebellar ataxia-1), psychiatric disorders (schizophrenia, mood disorders), inflammatory bowel disease. In many cases, overexpression/administration of the protein induces worsening of the disease, while its deletion/inactivation produces amelioration. This review points out that the pivotal role of the protein resulting from these data, opens the perspective that S100B may be regarded as a therapeutic target for these different diseases, which appear to share some common features reasonably attributable to neuroinflammation, regardless their origin.

OXYMED

S100B – Astrocyte Neurotrophic Cytokine

Biol Trace Elem Res. 2018 Aug 14. doi: 10.1007/s12011-018-1463-2. [Epub ahead of print]

Chronic Oral Arsenic Exposure and Its Correlation with Serum S100B Concentration.

Hum Mol Genet. 2018 Aug 2. doi: 10.1093/hmg/ddy288. [Epub ahead of print]

Targeting RAGE as a potential therapeutic approach to Duchenne muscular dystrophy.

Eur Arch Psychiatry Clin Neurosci. 2018 Aug 6. doi: 10.1007/s00406-018-0928-9. [Epub ahead of print]

Cerebrospinal fluid markers analysis in the differential diagnosis of dementia with Lewy bodies and Parkinson's disease dementia.

Cancer Lett. 2018 Aug 1. pii: S0304-3835(18)30498-1. doi: 10.1016/j.canlet.2018.07.034. [Epub ahead of print]

S100B Suppression Alters Polarization of Infiltrating Myeloid-Derived Cells in Gliomas and Inhibits Tumor Growth.

Ageing Res Rev. 2018 Jul 30. pii: S1568-1637(18)30093-X. doi: 10.1016/j.arr.2018.07.004. [Epub ahead of print]

Towards frailty biomarkers: Candidates from genes and pathways regulated in aging and age-related diseases.

Oncol Rep. 2018 Sep;40(3):1574-1582. doi: 10.3892/or.2018.6527. Epub 2018 Jun 27.

S100B promotes chemoresistance in ovarian cancer stem cells by regulating p53.

Alcohol Clin Exp Res. 2018 Jun 28. doi: 10.1111/acer.13796. [Epub ahead of print]

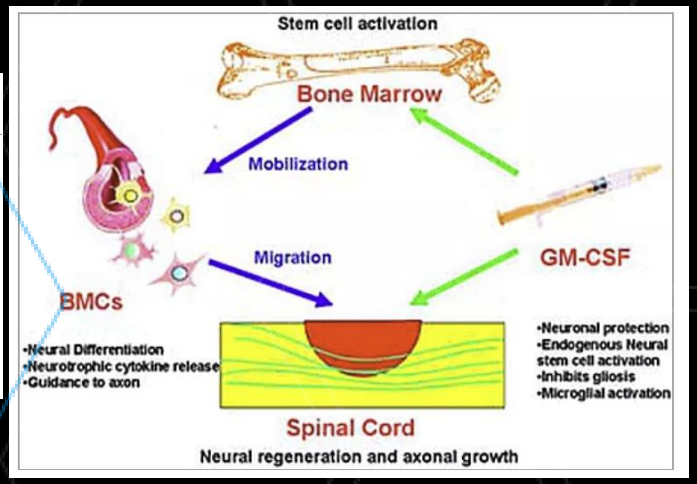
S100B and Inflammatory Cytokine Levels in Blood as Potential Markers of Blood-Brain Barrier Damage and Psychiatric Impairment in Comorbid Hepatitis C Viral Infection and Alcohol Use Disorder.

OXYMED

Granulocyte Colony Stimulating Factor (GMCSF)

STEM CELLS
TRANSLATIONAL AND CLINICAL RESEARCH

Complete Spinal Cord Injury Treatment Using Autologous Bone Marrow Cell Transplantation and Bone Marrow Stimulation with Granulocyte Macrophage-Colony Stimulating Factor: Phase I/II Clinical Trial



- GMCSF are glycoprotein growth factors.
- Stimulates blood stem cells to produce more white blood cells (neutrophils, eosinophils, basophils, and monocytes) to reduce the risk of infection notably after types of cancer treatment.
- GMCSF stimulates bone marrow and make stem cells move from the bone marrow into the blood.
- GMCSF can have intrinsic spinal cord repair mechanisms including neuroprotection from apoptosis, endogenous stem cell activation, inhibition of glial scar formation, and microglial cell activation.
- GMCSF decreased neuronal apoptosis and improved the functional outcome in SCI animal models.
- GMCSF stimulates microglial cells to increase brain-derived neurotrophic factor (BDNF) synthesis.
- The total number of recruited white blood cells in the peripheral blood was elevated after GMCSF (SGS) administration. The number of white blood cells in patients showing improved neurologic function was significantly higher.
- Following GMCSF - Spinal MRI Findings - 42.9% of patients in the GMCSF treated group showed an increase in the diameter of the spinal cord at the cell transplantation site. 28.6% showed evidence of spinal cord enhancement.

OXYMED

GMCSF

Eur J Neurosci. 2018 Aug 11. doi: 10.1111/ejn.14106. [Epub ahead of print]

Granulocyte-macrophage colony-stimulating factor improves mouse peripheral nerve regeneration following sciatic nerve crush.

Cytotherapy. 2010;12(1):50-9. doi: 10.3109/14653240903300682.

Consistent bone marrow-derived cell mobilization following repeated short courses of granulocyte-colony-stimulating factor in patients with amyotrophic lateral sclerosis: results from a multicenter prospective trial.

J Neuropathol Exp Neurol. 2006 Aug;65(8):816-25.

Intrathecal upregulation of granulocyte colony stimulating factor and its neuroprotective actions on motor neurons in amyotrophic lateral sclerosis.

J Vis Exp. 2018 Aug 10;(138). doi: 10.3791/57365.

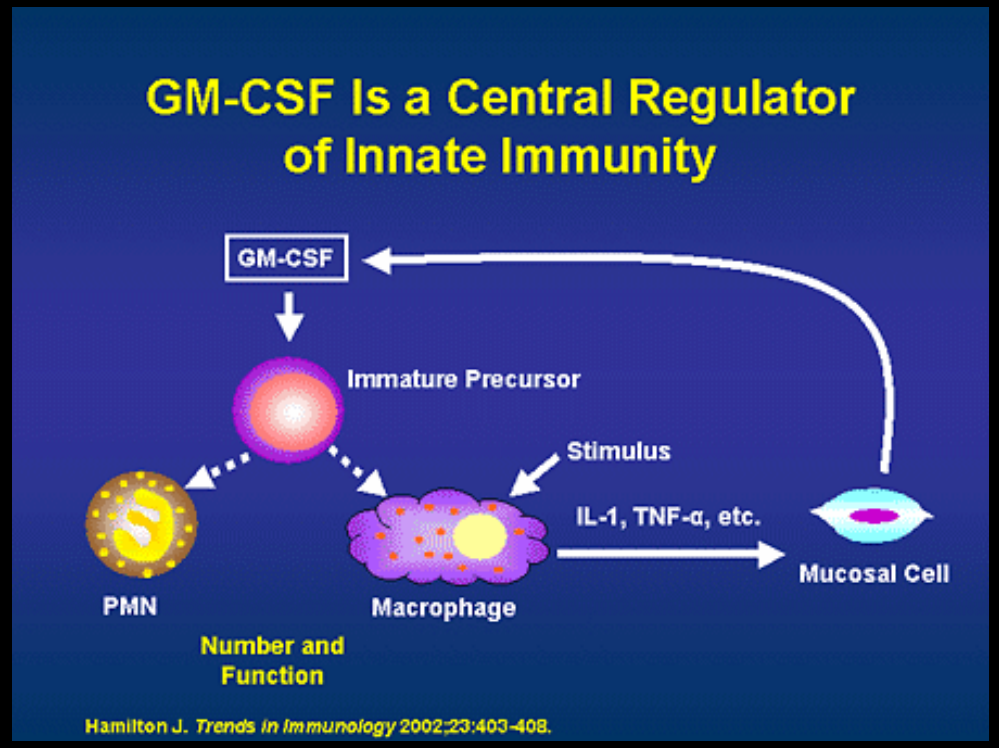
Generation of Large Numbers of Myeloid Progenitors and Dendritic Cell Precursors from Murine Bone Marrow Using a Novel Cell Sorting Strategy.

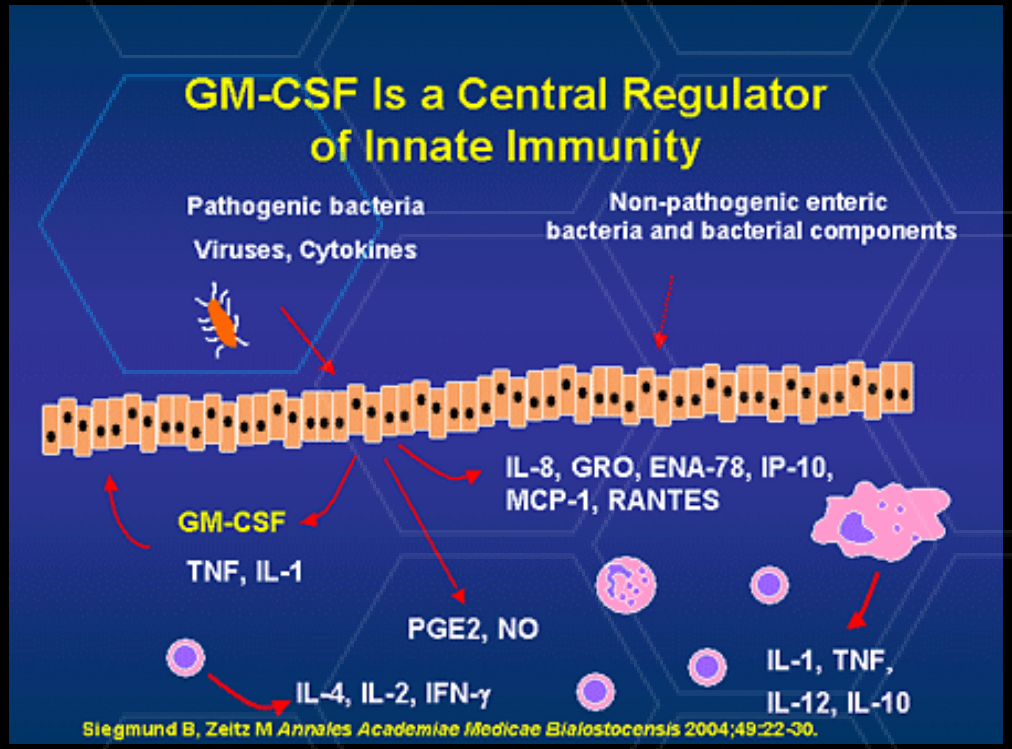
Int J Inflam. 2018 Aug 1;2018:2027856. doi: 10.1155/2018/2027856. eCollection 2018.

Association of Elevated Serum GM-CSF, IFN-γ, IL-4, and TNF-α Concentration with Tobacco Smoke Induced Chronic Obstructive Pulmonary Disease in a South Indian Population.

OXYMED

GMCSF – Innate Immunity





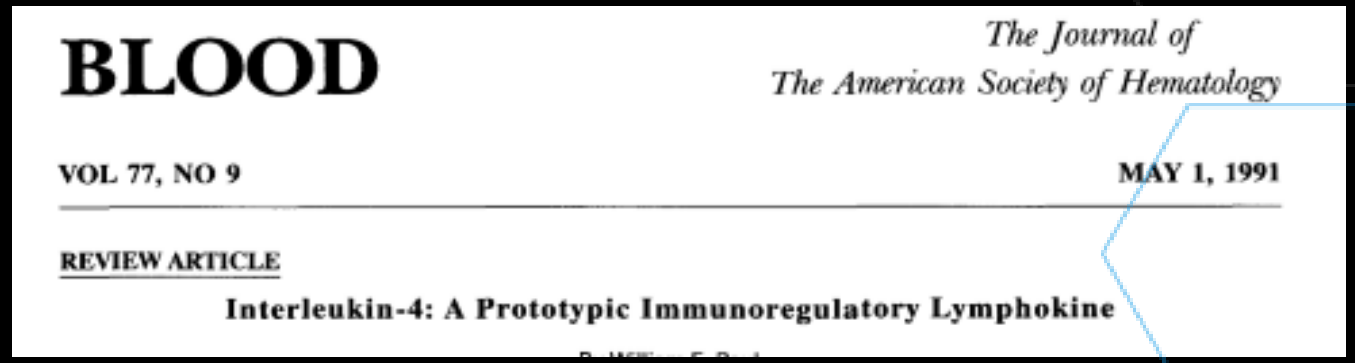
Filgrastim - Drug Information - Chemocare

chemocare.com/chemotherapy/drug-info/filgrastim.aspx ▾

Filgrastim is used to stimulate the production of granulocytes (a type of white blood cell) in patients undergoing therapy that will cause low white blood cell counts. This medication is used to prevent infection and neutropenic (low white blood cells) fevers caused by chemotherapy.

OXYMED

IL4 – Diverse Immune Modulation



BLOOD

The Journal of The American Society of Hematology

VOL 77, NO 9 MAY 1, 1991

REVIEW ARTICLE

Interleukin-4: A Prototypic Immunoregulatory Lymphokine

INTERLEUKIN-4, INTERLEUKIN-10, AND INTERLEUKIN-13

- Can all be regarded as *inhibitory, anti-inflammatory,* or *counter-regulatory* cytokines.
- All three of these cytokines are produced by TH2 cells
- serve to modulate the production and effects of proinflammatory cytokines such as TNF and IL-1.

- Studies of IL-4 have revealed a wealth of information on the diverse roles of this cytokine in homeostatic regulation and disease pathogenesis.
- IL4 is an Th2 anti-inflammatory cytokine, acting synergistic with **IL10 and IL13** responsible for cell growth factor that stimulates the growth and survivability of B cells and T cells.
- IL4 inhibits the production of pro-inflammatory cytokines including TNF, IL1, and IL6.
- IL4 is an immune-stimulating molecule. More recent targets being studied for new asthma treatments.
- IL4 has striking **antitumor activities** expressing potent biologic agents to enhance immune elimination of certain tumor cells.
- Ameliorates **non-resolving neuro-inflammation that causes neuropathic pain** after nerve injury (crush injury).

Neural Regen Res. 2018 Oct;13(10):1743-1752. doi: 10.4103/1673-5374.238615.

Release of interleukin-10 and neurotrophic factors in the choroid plexus: possible inductors of neurogenesis following copolymer-1 immunization after cerebral ischemia.

Acta Cir Bras. 2018 Jun;33(6):491-498. doi: 10.1590/s0102-865020180060000003.

Interleukin-4 protects from chemotherapy-induced peripheral neuropathy in mice modal via the stimulation of IL-4/STAT6 signaling.

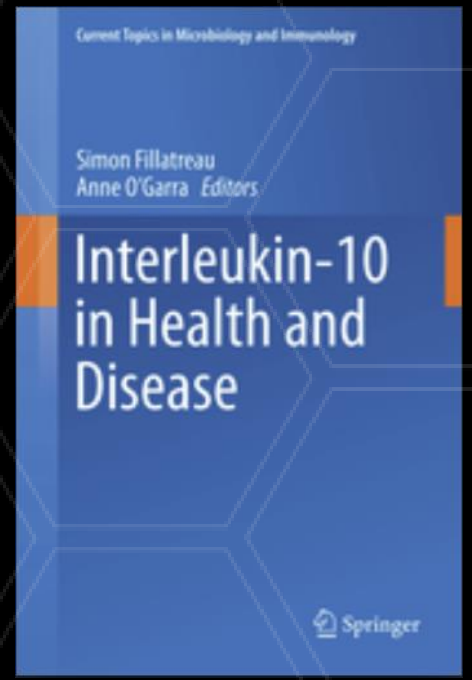
OXYMED

IL10

0031-6997/03/5502-241–260$7.00
PHARMACOLOGICAL REVIEWS
Copyright © 2003 by The American Society for Pharmacology and Experimental Therapeutics
Pharmacol Rev 55:241–269, 2003

Vol. 55, No. 2
0/1067026
Printed in U.S.A.

Interleukin-10 Therapy—Review of a New Approach



- Potent anti-inflammatory TH2 cytokine that has a critical role in limiting the immune response to pathogens to prevent host damage.
- Strong inhibitor of inflammation.
- Elevated levels in **parasitic infection,** high expression levels of IL10 are also found in retroviral infections inducing immunodeficiency.
- The immunosuppressive properties of IL10 suggest a possible clinical use in suppressing rejections of grafts after organ transplantations.
- Patients with Crohn's disease react favourably to treatment with bacteria producing recombinant IL10.
- Pre conditioning elevation of IL10 induces a resistance of the brain cells to ischemia-evoked damages.
- This protective effect in cultured hippocampal cells is developed rapidly after application of IL10, capable to exert the rapid neuroprotective effects through transcription-independent modulation of ischemia-induced intracellular Ca(2+) responses in the brain cells.
- IL10 **upregulates BNDF** production.

Int J Mol Sci. 2018 Aug 28;19(9). pii: E2550. doi: 10.3390/ijms19092550.

Schwann Cell Transplantation Subdues the Pro-Inflammatory Innate Immune Cell Response after Spinal Cord Injury.

OXYMED

IL10

Neural Regen Res. 2018 Sep;13(9):1650-1656. doi: 10.4103/1673-5374.237139.

Saikosaponin a increases interleukin-10 expression and inhibits scar formation after sciatic nerve injury.

J Stroke Cerebrovasc Dis. 2018 Aug 14. pii: S1052-3057(18)30399-9. doi: 10.1016/j.jstrokecerebrovasdis.2018.07.030. [Epub ahead of print]

The Effects of Mouse Recombinant Resistin on mRNA Expression of Proinflammatory and Anti-Inflammatory Cytokines and Heat Shock Protein-70 in Experimental Stroke Model.

Neurosci Lett. 2018 Sep 14;683:181-184. doi: 10.1016/j.neulet.2018.07.027. Epub 2018 Jul 29.

Association of Parkinson's disease-related pain with plasma interleukin-1, interleukin-6, interleukin-10, and tumour necrosis factor-α.

Abstract

OBJECTIVE: To study the association between Parkinson's disease (PD)-related pain and plasma interleukin (IL)-1, IL-6, IL-10, and tumour necrosis factor (TNF)-α levels.

METHODS: Sixty-seven participants were enrolled. Plasma inflammatory cytokine levels of IL-1, IL-6, IL-10, and TNF-α were measured with enzyme-linked immunosorbent assay. We additionally administered the third part of the Unified Parkinson's Disease Rating Scale (UPDRS III) and Hoehn and Yahr (H-Y) scale stage and recorded the course of the disease, the type and location of the pain, and the use of drugs.

Neuropsychopharmacology. 2018 Jul 16. doi: 10.1038/s41386-018-0154-1. [Epub ahead of print]

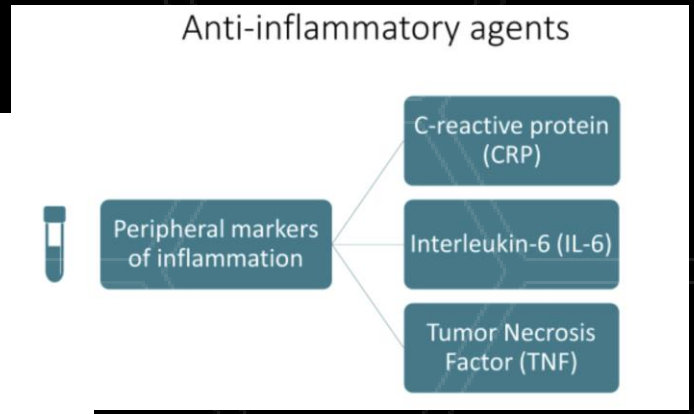
Resolution of inflammation-induced depression requires T lymphocytes and endogenous brain interleukin-10 signaling.

OXYMED

IL13

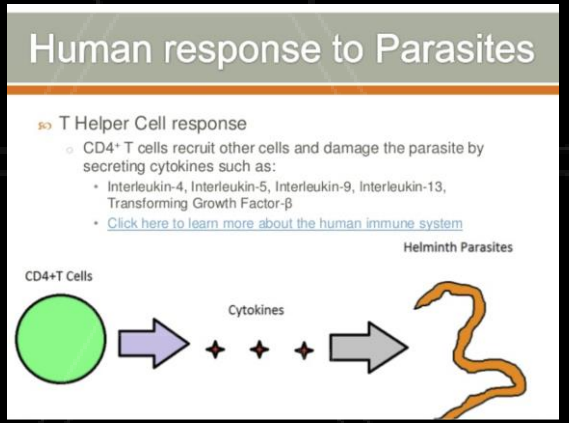
Reoxygenation of Hypoxic Glioblastoma Multiforme Cells Potentiates the Killing Effect of an Interleukin-13-Based Cytotoxin

Tie Fu Liu,[1,2] Jiaozhong Cai,[2] Denise M. Gibo,[1] and Waldemar Debinski[1]



- IL13 has **anti-tumour effects** and when combined with HBO enhances the killing effects of Glioblastoma and other cancers.
- **Interleukin-13 receptor-targeted cytotoxin** (IL13-PE38) is highly cytotoxic to human glioblastoma (GBM) cells.

- IL13 is a cytokine found to powerfully inhibits proinflammatory cytokines.
- Elevated IL13 levels in the CSF are reported to have **neuroprotective** effects in multiple neurodegenerative disorders including Multiple Sclerosis.
- **IL13 enhances neuronal integrity and synaptic function in neurodegenerative disorders including MS.**
- IL13 specifically induces physiological changes in parasitized organs that are required to expel the offending organisms or their products. For example, expulsion from the gut of a variety of mouse helminths requires IL13.
- IL-13 induces several changes in the gut that create an environment **hostile to the parasite**, including enhanced contractions and glycoprotein hyper-secretion from gut epithelial cells, that ultimately lead to detachment of the organism from the gut wall and their removal.



PLoS One. 2018 Apr 5;13(4):e0193565. doi: 10.1371/journal.pone.0193565. eCollection 2018.

IL-13 receptors as possible therapeutic targets in diffuse intrinsic pontine glioma.

OXYMED

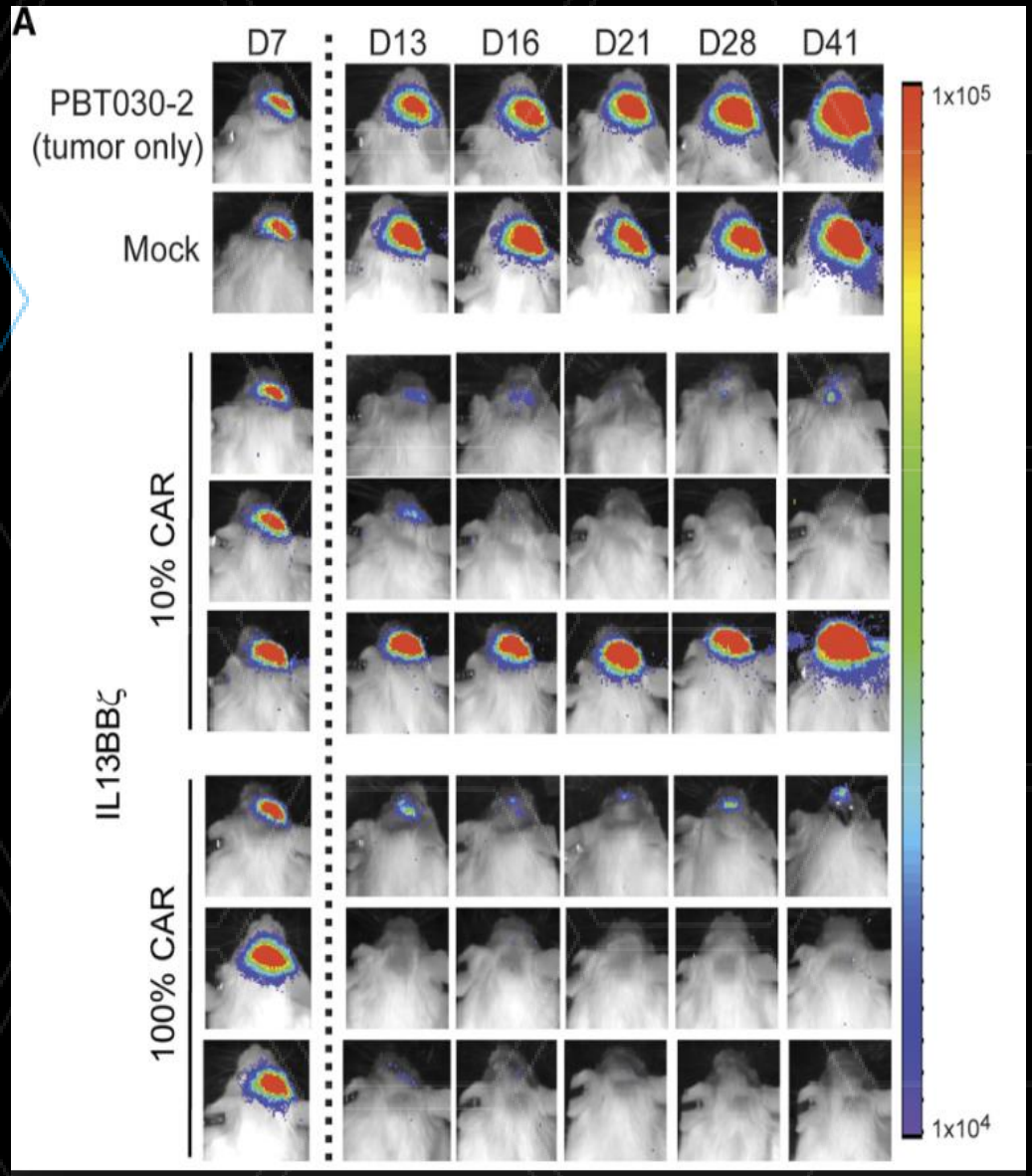
IL13 - T Cell Immunotherapy

ORIGINAL ARTICLE | VOLUME 26, ISSUE 1, P31-44, JANUARY 03, 2018

Optimization of IL13Rα2-Targeted Chimeric Antigen Receptor T Cells for Improved Anti-tumor Efficacy against Glioblastoma

- IL13 based T cell immunotherapy is emerging as a powerful strategy to treat cancer and may improve outcomes for patients with glioblastoma (GBM).
- **Chimeric Antigen Receptor** (CAR) T cell immunotherapy targeting IL-13 receptor α2 (IL13Rα2) for the treatment of GBM. The general premise of CAR-T cells is to artificially generate T-cells targeted to markers found on cancer cells. **Scientists can remove T-cells from a person, genetically alter them, and put them back into the patient to attack the cancer cells.**

- Intracranial delivery of CAR T cells elicits superior anti-tumor efficacy as compared to intravenous administration, with intraventricular infusions exhibiting possible benefit over intracranial tumor infusions.



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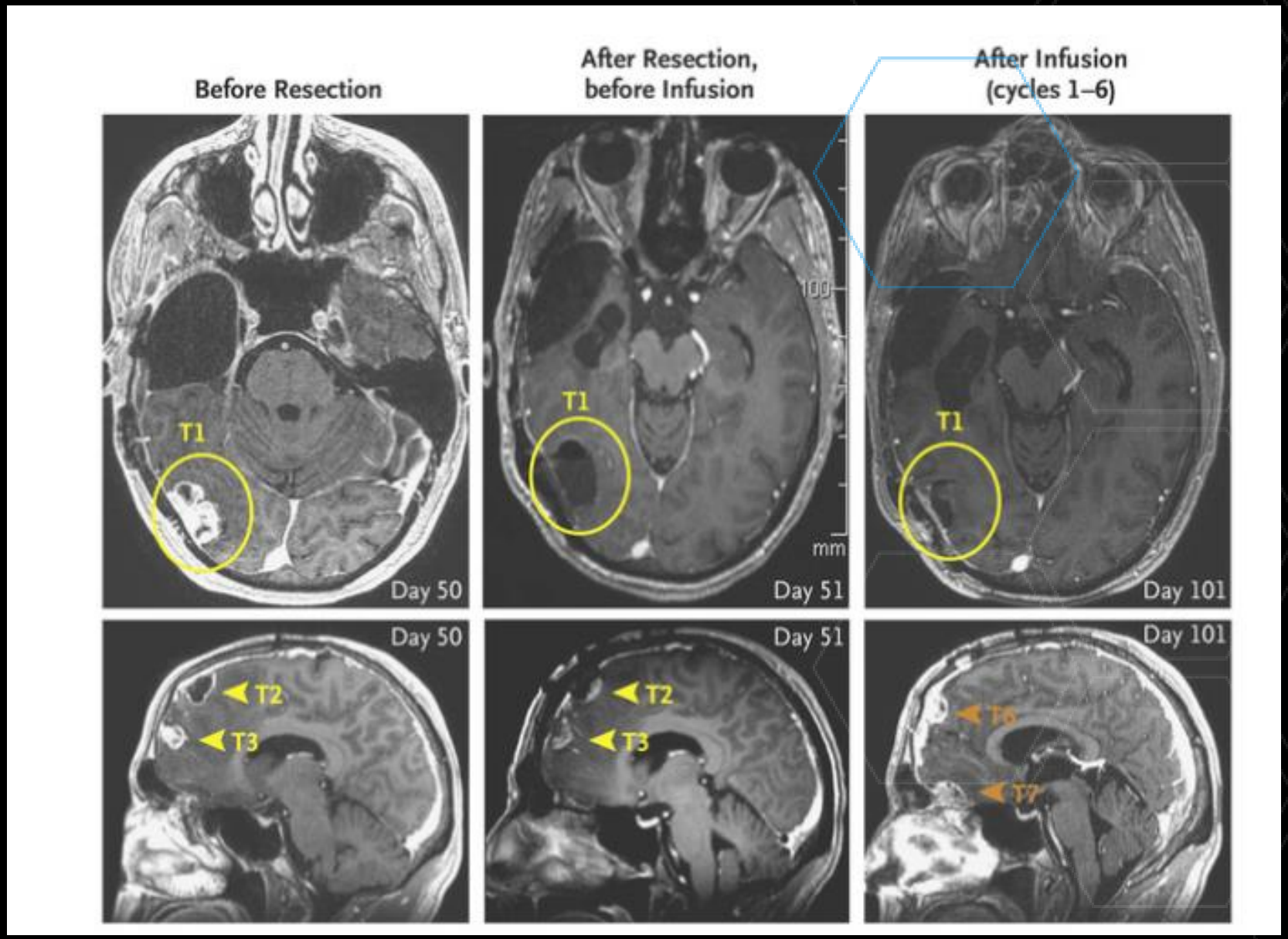
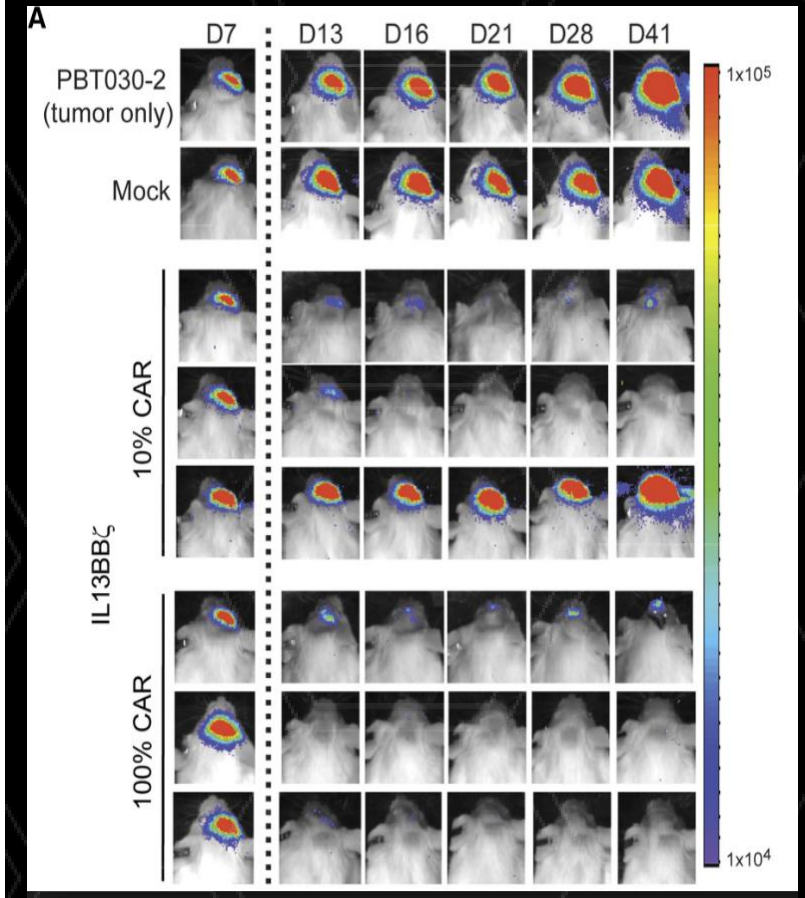
IL13CAR - Glioblastoma





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Brain Derived Neurotrophic Factor (BDNF)

Int J Geriatr Psychiatry. 2018 Aug 29. doi: 10.1002/gps.4962. [Epub ahead of print]

Role of physical activity in ameliorating neuropsychiatric symptoms in Alzheimer disease: A narrative review.

J Stroke Cerebrovasc Dis. 2018 Aug 24. pii: S1052-3057(18)30356-2. doi: 10.1016/j.jstrokecerebrovasdis.2018.06.032. [Epub ahead of print]

Brain-Derived Neurotrophic Factor Levels are Lower in Chronic Stroke Patients: A Relation with Manganese-dependent Superoxide Dismutase ALA16VAL Single Nucleotide Polymorphism through Tumor Necrosis Factor-α and Caspases Pathways.

Med Sci Monit. 2018 Aug 26;24:5943-5950. doi: 10.12659/MSM.909449.

Diagnostic Significance of Serum Levels of Nerve Growth Factor and Brain Derived Neurotrophic Factor in Diabetic Peripheral Neuropathy.

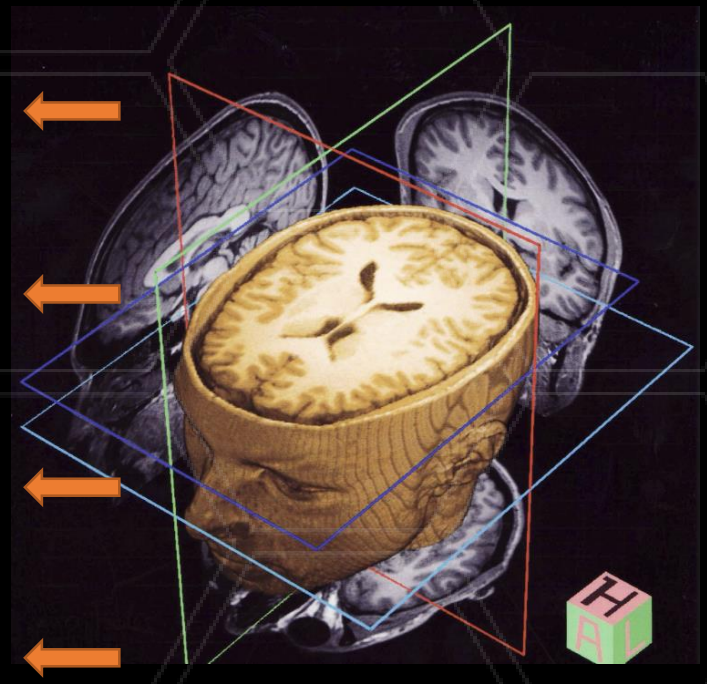
Psychiatry Res. 2018 Jul 26;268:478-483. doi: 10.1016/j.psychres.2018.07.013. [Epub ahead of print]

Altered levels of brain-derived neurotrophic factor, proBDNF and tissue plasminogen activator in children with posttraumatic stress disorder.

Zh Nevrol Psikhiatr Im S S Korsakova. 2018;118(5. Vyp. 2):51-56. doi: 10.17116/jnevro20181185251.

[A neuroprotective approach to optimizing treatment and correction activities in children with autism spectrum disorders].

RESULTS AND CONCLUSION: There was a decrease in NGF, BDNF, Hsp27, Hsp70 in the total group and in boys with severe ASD. These changes reflect the deterioration of neuroprotective processes in the brain in children with ASD that demands further research and development of treatment procedures.



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Cerebrolysin (BDNF)

- Cerebrolysin – brain derived neurotrophic factor (BDNF), glial derived neurotrophic factor (GDNF), nerve growth factor (NGF), ciliary neurotrophic factor (CNTF) and other peptide fragments.
- Improves cognitive performance and global function in neurodegenerative disorders with 'increased daily quality living'. CL activates cerebral mechanisms related to attention and memory processes.
- CL improvements reported in mild to moderate progressive neurodegenerative disease - multiple sclerosis, Parkinson's disease, Alzheimer's disease, dementia, acute and chronic Stroke, acute traumatic brain injury, childhood autism (89%) and cerebral palsy.
- Attenuates motor neuron damage in spinal cord with significant motor recovery. reducing chronic nerve cell inflammation in both acute and chronic neurodegenerative diseases.
- Neuroprotective and neurorestorative properties, demonstrates 'anti-aging' with benefits 'improving cognition, memory function, brain metabolism with capacity.
- Reduces brain Aβ deposition, and tau-related neuropathology. Modulates neuroinflammation, attenuating microglia activation and IL-1β release, reducing the elevated serum levels of TNF-α and TNF receptor-1 in AD patients.
- Neurotrophic-like actions on neuronal survival and outgrowth, increases circulating IGF-1 and BDNF levels in humans. Enhances the supply of glucose to the brain and ameliorates the slowing of brain bioelectrical activity.
- Promotes neural plasticity, neurogenesis and neuronal survival protecting from apoptosis and degeneration.

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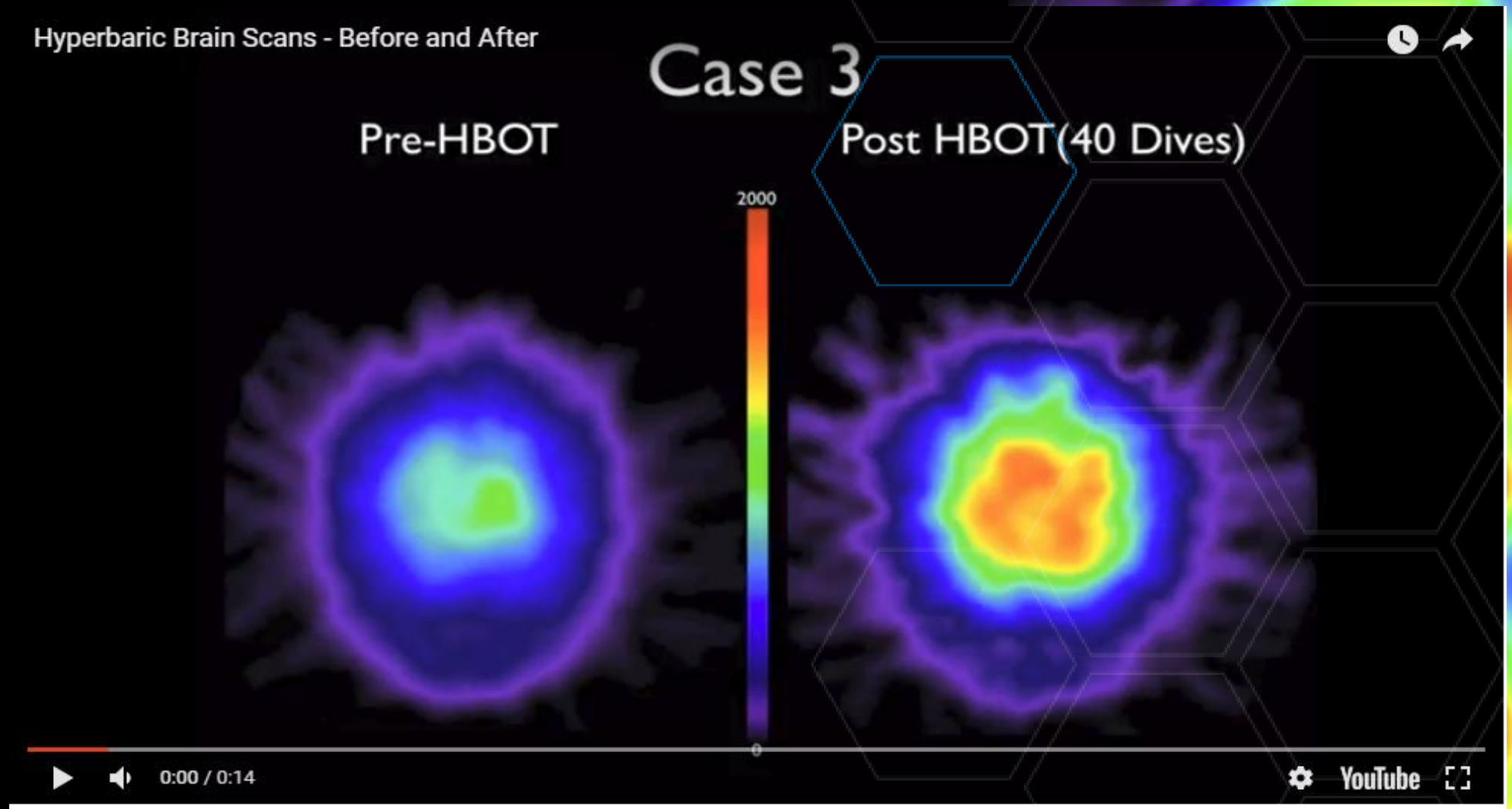
OXYGENE - The Power Within



"A picture is worth a 1000 words. These are side by side pre and post HBOT metabolic SPECT scans showing the improvement over time in the brain function of the individual with 40 hyperbaric therapy treatments" Dr. Ted Fogarty, MD IHMF President & Dr. Paul Harch, MD IHMA Executive Board

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N = 1 Hyperbaric Oxygen impacts the Cellular landscape

- Hyperbaric Oxygen is breathing 100 per cent oxygen at pressures greater than normal. HBOT increases dissolved oxygen into the into the damaged regions of the body. HBO increases blood plasma by 10-15 fold (1000-1500 per cent). The normal blood plasma carry only 1-2 per cent oxygen with red blood cells carrying approximately 98 per cent oxygen.
- *"Hyperbaric Oxygen Therapy creates a 'fertile neurovascular platform' for emerging stem cell, immunotherapies and nanotechnology techniques. The impact and success of these and future procedures are dependent on the integrity of the underlying supporting neurovascular bed."* (Hooper 2005).
- HBO acts as a 'catalyst' promoting immune modulation. HBO results in increased blood flow by fostering the formation of **existing and new capillary dynamics** (neovascularization) activating damaged and dormant nerve cells ('penumbra state'). HBO accelerates neuroplasticity.
- Approximately 20 to 30 percent of the body's consumption of Oxygen occurs within three to five per cent of the body mass – the brain and spinal cord. These structures are extremely sensitive to Oxygen deficiency and the use of HBO.
- Increased Oxygenation accelerates the rate of healing, stabilization and repair through numerous immune modulating effects, providing upregulation of anti-inflammatory cytokines, including: Granulocyte Macrophage Colony Stimulating Factor (GM-CSF), Interleukin-3 (IL3), Interleukin-4 (IL4), Interleukin10 (IL10), Interleukin-13 (IL13), Interleukin-21 (IL21), Brain Derived Neural Growth Factors (BDNF, GDNF), Vascular Growth Factors (VEGF), TGFβ1 Signaling and IGF1.
- In chronic injuries, the microenvironment is in a constant smoldering "cytokine storm" state. Pro-inflammatory cytokines are important in mobilizing the reparative and regenerative responses when 'attacked', but chronic over-expression leads to immune confusion and autoimmune degradation. Over-expressions of pro-inflammatory cytokines can affect synaptic strength and synaptic plasticity, and excess contributes to maladaptive plasticity and chronic pain syndromes.

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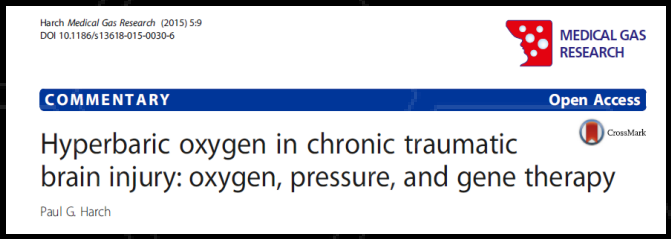
Hyperbaric Oxygen ‘Epigenetic Therapy’ (Harch 2015)

- In essence, **hyperbaric therapy physicians are playing a symphony with patients’ gene expression**, the music of which is determined by the **various pressures and amounts of hyperoxia** to which the patient is exposed.
- Tissue growth requires replication of DNA. **HBOT is a DNA signaling agent.**
- A single HBOT, at the pressure used for diabetic foot wounds and radiation wounds up or downregulated the expression of **8,101 genes** (nearly 50%) of the known 19-20,000 protein-coding genes in the human genome.
- Clusters of neuronal genes are affected by **‘different pressures’ and ‘different amounts of hyperoxia’.**
- **Upregulated genes** are primarily **growth and repair hormones and anti-inflammatory genes.**
- **Downregulated genes** are the **pro-inflammatory and apoptotic genes.**
- HBO **'expands the therapeutic window’** reducing continuing neurovascular deterioration. HBO upregulates the patient's own **target specific Stem Cells** (with an 8-fold or 800 percent increase in circulating CD34+).
- HBO enhances **Mitochondrial respiration.**
- HBO proliferates Granulocyte Macrophage Colony Stimulating Factor (GM-CSF), Interleukin-3 (IL3), Interleukin-4 (IL4), Interleukin-10 (IL10), Interleukin-13 (IL13), Interleukin-21 (IL21), Brain Derived Neural Growth Factors (BDNF, GDNF), Vascular Growth Factors (VEGF), TGFβ Signalling, IGF1.
- HBO reduces Telomere degeneration.
- HBO down regulates toxic intra and extra cellular inflammatory Cytokines (IL1, 2, 6, 7, 8, 17), Tumour Necrosis Factor Alpha (TNFα), GlycA, S100B.
- HBO inhibits opportunistic infections (MRSA, viral, bacterial, parasitic), cell sepsis and more.



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Stem Cell Mobilization (2005)

Am J Physiol Heart Circ Physiol. 2006 Apr;290(4):H1378-86. Epub 2005 Nov 18.

Stem cell mobilization by hyperbaric oxygen.

Thom SR[1], Bhopale VM, Velazquez OC, Goldstein LJ, Thom LH, Buerk DG.

Author information

Abstract

We hypothesized that exposure to hyperbaric oxygen (HBO(2)) would mobilize stem/progenitor cells from the bone marrow by a nitric oxide (*NO) -dependent mechanism. The population of CD34(+) cells in the peripheral circulation of humans doubled in response to a single exposure to 2.0 atmospheres absolute (ATA) O(2) for 2 h. Over a course of 20 treatments, circulating CD34(+) cells increased eightfold, although the overall circulating white cell count was not significantly increased. The number of colony-forming cells (CFCs) increased from 16 +/- 2 to 26 +/- 3 CFCs/100,000 monocytes plated. Elevations in CFCs were entirely due to the CD34(+) subpopulation, but increased cell growth only occurred in samples obtained immediately posttreatment. A high proportion of progeny cells express receptors for vascular endothelial growth factor-2 and for stromal-derived growth factor. In mice, HBO(2) increased circulating stem cell factor by 50%, increased the number of circulating cells expressing stem cell antigen-1 and CD34 by 3.4-fold, and doubled the number of CFCs. Bone marrow *NO concentration increased by 1,008 +/- 255 nM in association with HBO(2). Stem cell mobilization did not occur in knockout mice lacking genes for endothelial *NO synthase. Moreover, pretreatment of wild-type mice with a *NO synthase inhibitor prevented the HBO(2)-induced elevation in stem cell factor and circulating stem cells. We conclude that HBO(2) mobilizes stem/progenitor cells by stimulating *NO synthesis.

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HBOT Erectile Dysfunction

Int J Impot Res. 2018 May 18. doi: 10.1038/s41443-018-0023-9. [Epub ahead of print]

Hyperbaric oxygen can induce angiogenesis and recover erectile function.

Hadanny A1,2,3, Lang E4,5,6, Copel L5,7, Meir O4, Bechor Y4, Fishlev G4,5, Bergan J4,5, Zisman A5,6, Efrati S4,5,8,9.
Author information

Erratum in

- Correction: Hyperbaric oxygen can induce angiogenesis and recover erectile function. [Int J Impot Res. 2018]

Abstract

Erectile dysfunction (ED) is caused by microvascular or macrovascular insufficiency in the majority of patients. Recent studies have shown that hyperbaric oxygen therapy (HBOT) can induce angiogenesis in different body organs. The effect of HBOT on the non-surgery-related ED has not been investigated yet. The aim of the current study was to evaluate the effects of HBOT on sexual function and penile vascular bed in non-surgical ED patients. A prospective analysis of patients suffering from chronic ED treated with 40 daily HBOT sessions (80-hours HBO 2 ATA 100% O2). Clinical efficacy was assessed using the International Index of Erectile Function questionnaire (IIEF) and a global efficacy question (GEQ). The effect on the penile vascular bed was evaluated by perfusion MRI.

Thirty men (mean age of 59.2 ± 1.4) suffering from ED for 4.2 ± 0.6 years completed the protocol. HBOT significantly improved all IIEF domains by 15-88% ($p < 0.01$). Erectile function improved by 88% ($p < 0.0001$) and 80% of the patients reported positive outcome according to the GEQ. Angiogenesis was indicated by perfusion MRI that showed a significant increase by 153.3 ± 43.2% of K-trans values in the corpous cavernous ($p < 0.0001$).

- HBOT can induce penile angiogenesis and improve erectile function in men suffering from EcD. HBOT reverses the basic common pathophysiology, atherosclerosis and decreased penile perfusion, responsible for most cases of ED.

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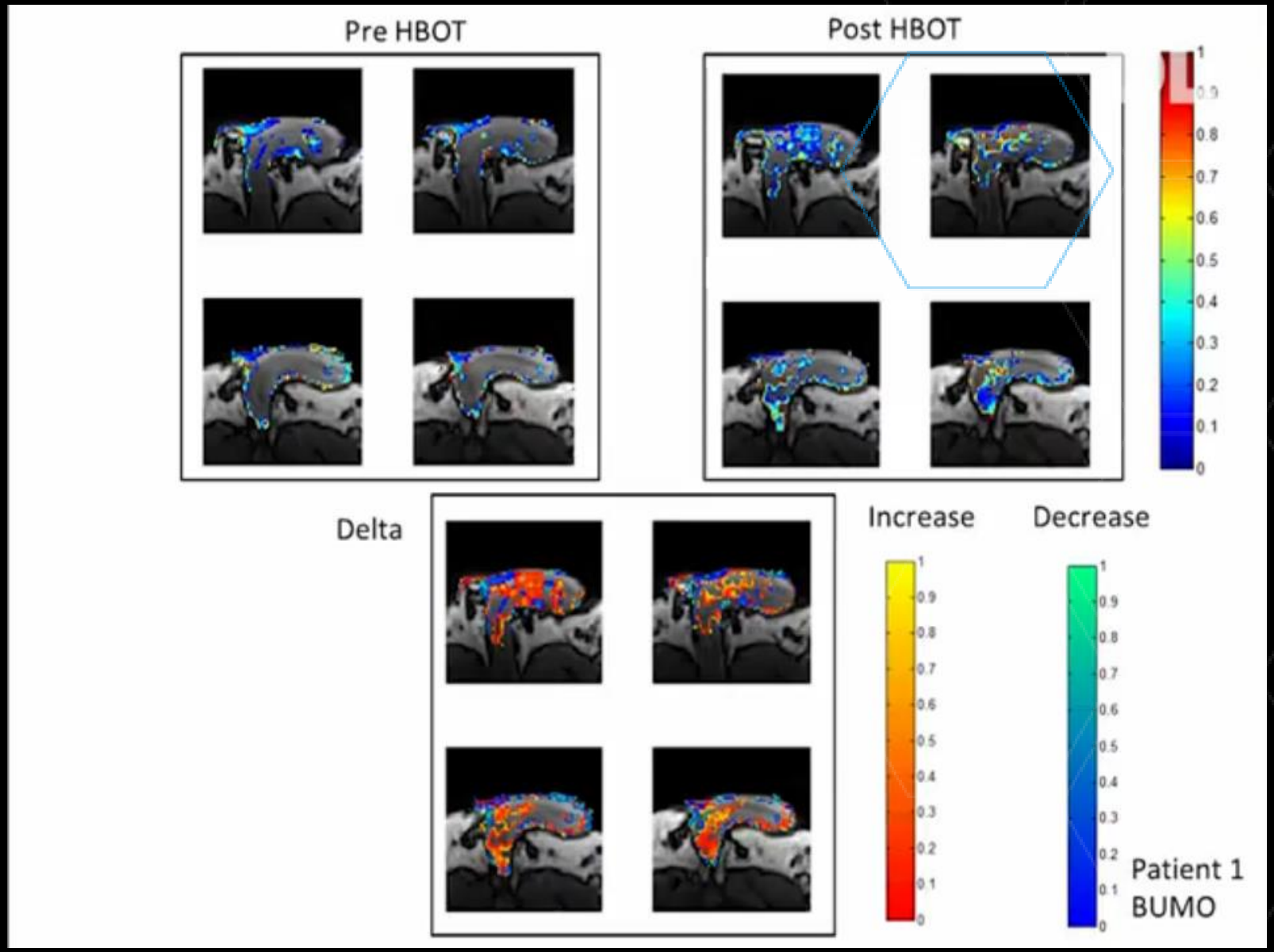
Blood flow changes



The Future? HBOT & Nobel Gases – Argon, Xenon

REVIEW

The role of argon in stroke

Xiang Li#, Zhu-Wei Zhang#, Zhong Wang*, Jin-Quan Li*, Gang Chen
Department of Neurosurgery & Brain and Nerve Research Laboratory, The First Affiliated Hospital of Soochow University, Suzhou, Jiangsu Province, China.

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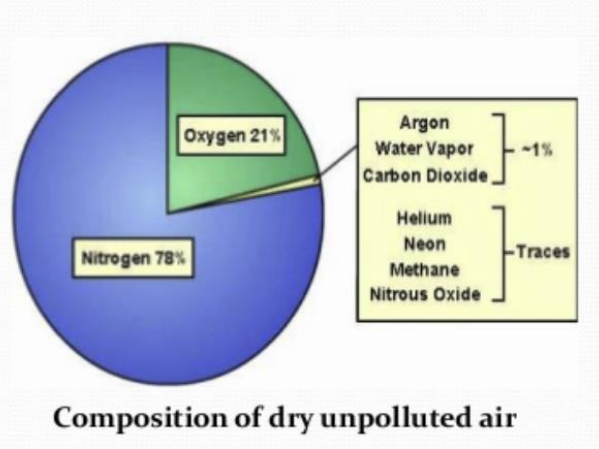
orcid: 0000-0002-6317-2980 (Jin-Quan Li)

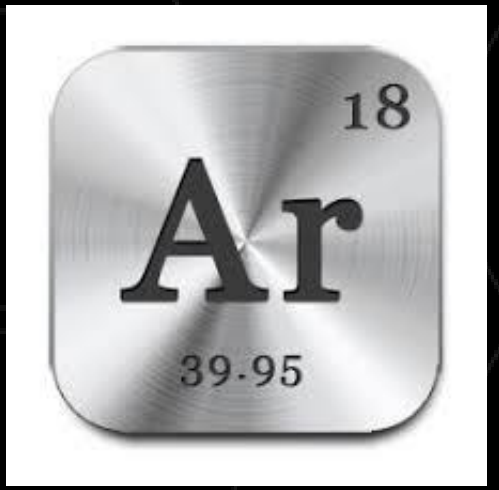
Abstract

Stroke, also known as "cerebrovascular accident", is an acute cerebrovascular disease that is caused by a sudden rupture of blood vessels in the brain or obstruction of the blood supply by blockage of blood vessels, thus including hemorrhagic and ischemic strokes. The incidence of ischemic stroke is higher than that of hemorrhagic stroke, and accounts for 80% of the total number of strokes. However, the mortality rate of hemorrhagic stroke is relatively high. Internal carotid artery and vertebral artery occlusion and stenosis can cause ischemic stroke, and especially males over 40 years of age are at a high risk of morbidity. According to the survey, stroke in urban and rural areas has become the first cause of death in China. It is also the leading cause of disability in Chinese adults. In a word, stroke is characterized by high morbidity, high mortality and high disability rates. Studies have shown that many noble gases have the neuroprotective effects. For example, xenon has been extensively studied in various animal models of neurological injury including stroke, hypoxic-ischemic encephalopathy. Compared to xenon, Argon, as a noble gas, is abundant, cheap and widely applicable, and has been also demonstrated to be neuroprotective in many research studies. In a variety of models, ranging from oxygen-glucose deprivation in cell culture to complex models of mid-cerebral artery occlusion, subarachnoid hemorrhage or retinal ischemia-reperfusion injury in animals. Argon administration after individual injury demonstrated favorable effects, particularly increased cell survival and even improved neuronal function. Therefore the neuroprotective effects of argon may be of possible clinical use for opening a potential therapeutic window in stroke. It is important to illuminate the mechanisms of argon in nerve function and to explore the best use of this gas in stroke treatment.

Key words: argon; stroke; experimental research; underlying mechanism; clinical research; stroke treatment; neuroprotective effects; therapeutic implications



Composition of dry unpolluted air



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HBOT Neuroprotection

A Dual Role for Hyperbaric Oxygen in Stroke Neuroprotection: Preconditioning of the Brain and Stem Cells

Grant M. Liska, Trenton Lippert, Eleonora Russo, Norton Nieves, and Cesar V. Borlongan[1]

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The publisher's final edited version of this article is available at Cond Med

Abstract

Go to:

Stroke continues to be an extremely prevalent disease and poses a great challenge in developing safe and effective therapeutic options. Hyperbaric oxygen therapy (HBOT) has demonstrated significant pre-clinical effectiveness for the treatment of acute ischemic stroke, and limited potential in treating chronic neurological deficits. Reported benefits include reductions in oxidative stress, inflammation, neural apoptosis, and improved physiological metrics such as edema and oxygen perfusion, all of which contribute to improved functional recovery. This pre-clinical evidence has failed to translate into an effective evidence-based therapy, however, due in large part to significant inconsistencies in treatment protocols and design of clinical studies. While the medical community works to standardize clinical protocols in an effort to advance HBOT for acute stroke, pre-clinical investigations continue to probe novel applications of HBOT in an effort to optimize stroke neuroprotection. One such promising strategy is HBOT preconditioning. Based upon the premise of mild oxidative stress priming the brain for tolerating the full-blown oxidative stress inherent in stroke, HBOT preconditioning has displayed extensive efficacy. Here, we first review the pre-clinical and clinical evidence supporting HBOT delivery following ischemic stroke and then discuss the scientific basis for HBOT preconditioning as a neuroprotective strategy. Finally, we propose the innovative concept of stem cell preconditioning, in tandem with brain preconditioning, as a promising regenerative pathway for maximizing the application of HBOT for ischemic stroke treatment.

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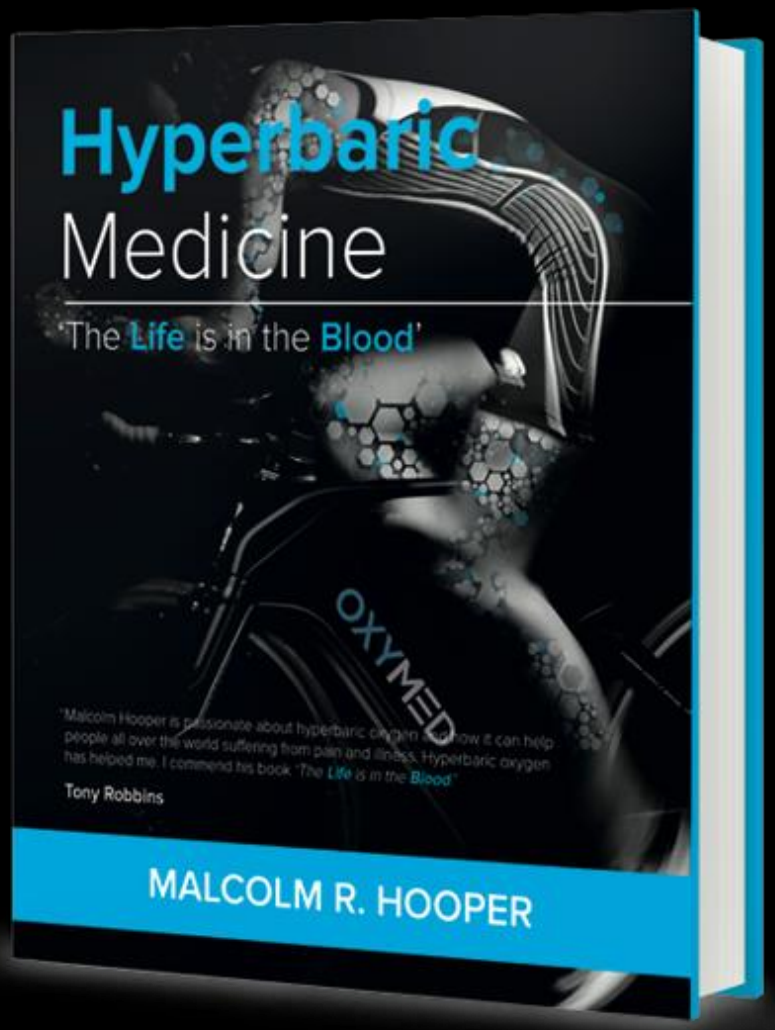
Malcolm R. Hooper



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Thankyou

"The purpose of my life is to be 'complete', to live in God's Grace and Blessing, to lift the 'Spirit of Humanity' for myself and others. I am passionate with the vision that people of all nations will gain the opportunity to access the abundant benefits of Oxygenation – the cornerstone of HealthCare in the modern era". Malcolm R. Hooper

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