Malcolm R. Hooper

OXYMED Australia

Tony Robbins

Cytokine Profiles May 2018

The Final Frontier – the Power within

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Unleash the Power within

And keep the Power going

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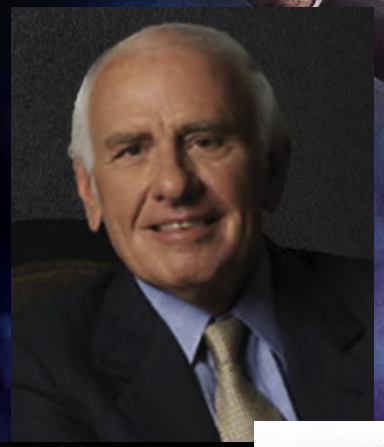
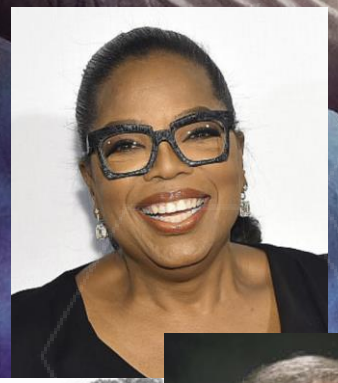
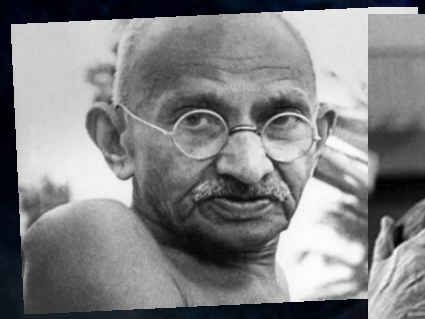
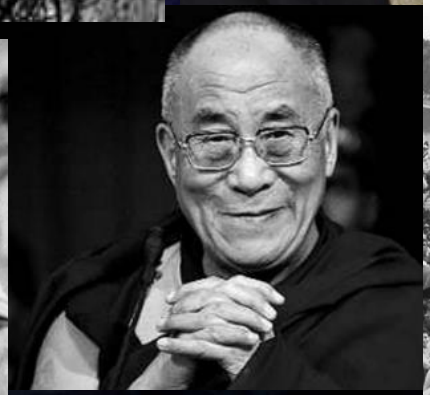
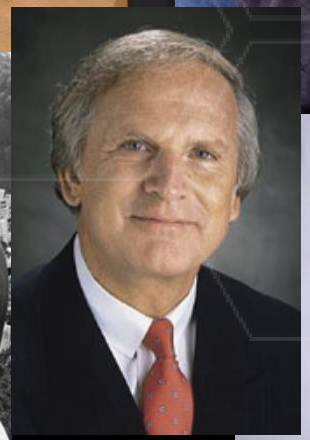
Who influences the influencer?











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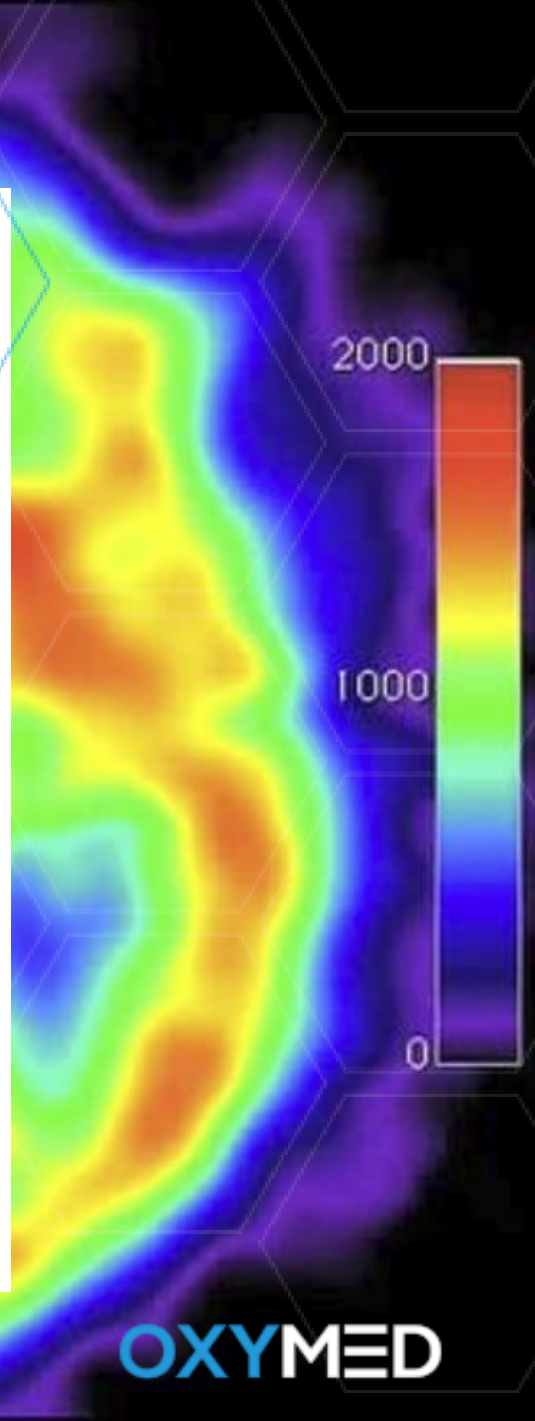
TechKnowledge Abundance

The frontier within requires the thinking of tomorrow

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** (1,000-1500 per cent). The normal blood plasma carry only 1-2 per cent oxygen with red blood cells carrying approximately 98 per cent oxygen.
- 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 (referred to as the 'penumbra state'). HBOT 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 significantly 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), TGFβ Signaling and IGF1.
- *"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).
- 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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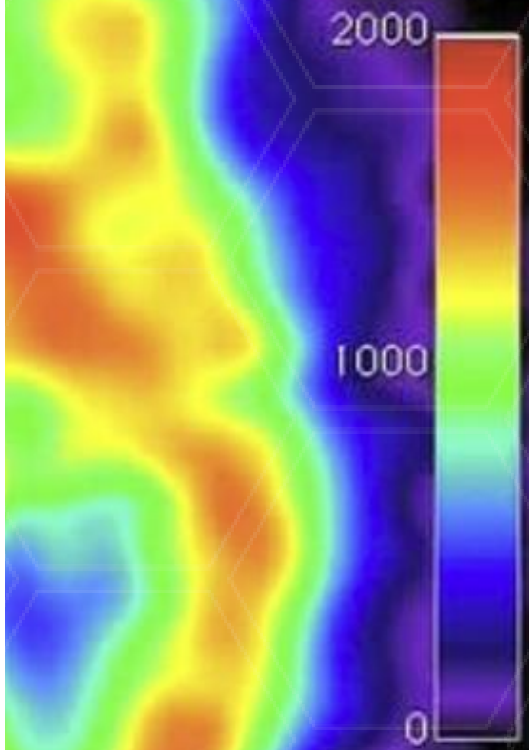
Hyperbaric Oxygen Epigenetic Therapy

Summary of Benefits:

HBO effects Traumatic Brain Injury: Oxygen, Pressure & Gene Therapy (Harch 2015)

- In essence, during **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. The oxygen component of 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 (nearly 50%) of the known 19-20,000 protein-coding genes in the human genome.
- Further work showed 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 and HBOT

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

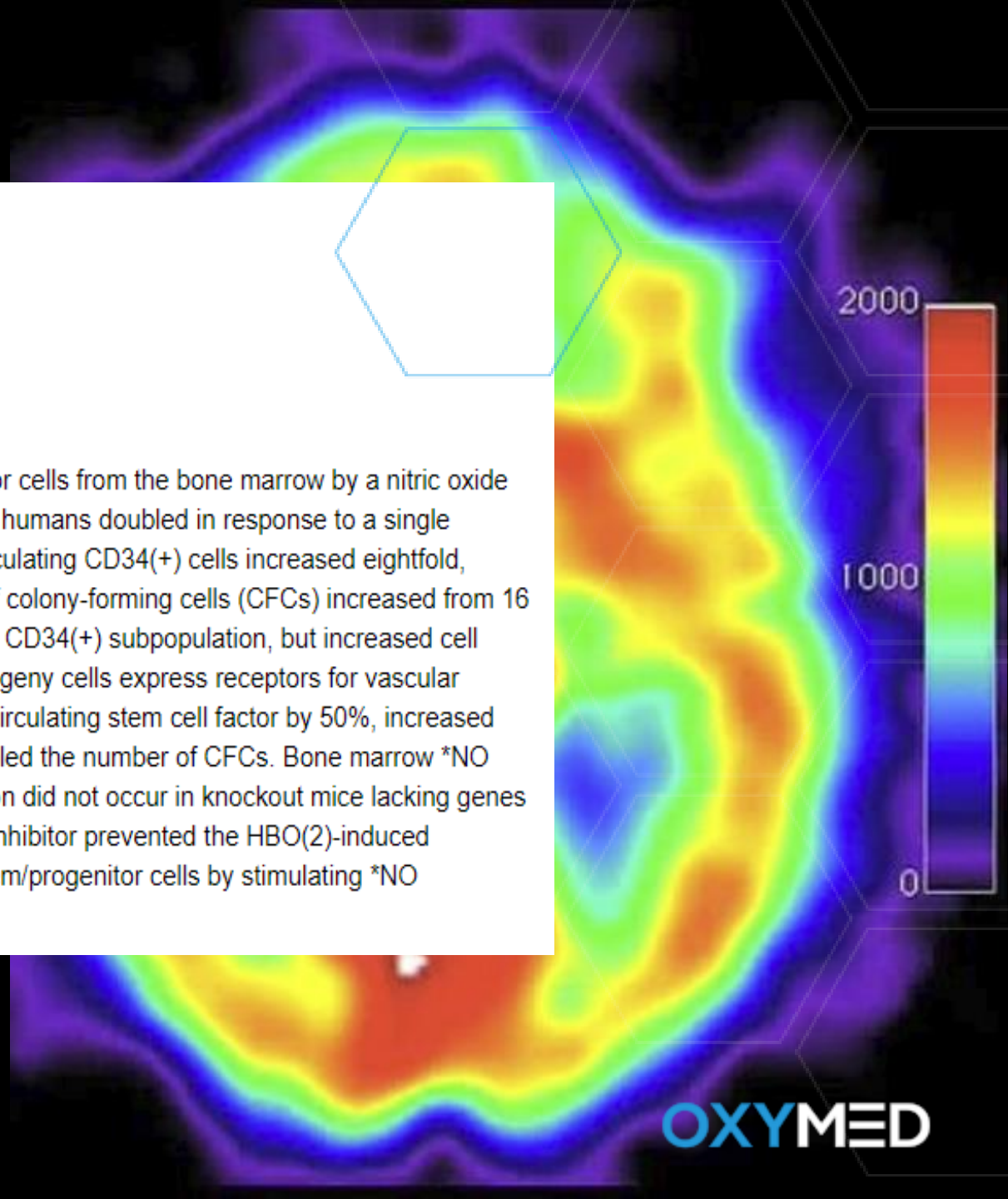
Stem cell mobilization by hyperbaric oxygen.

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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Cytokine Gene Signaling – The Cellular Landscape

TONY ROBBINS **- MALCOLM HOOPER**

NutriPATH
Integrative Pathology Services

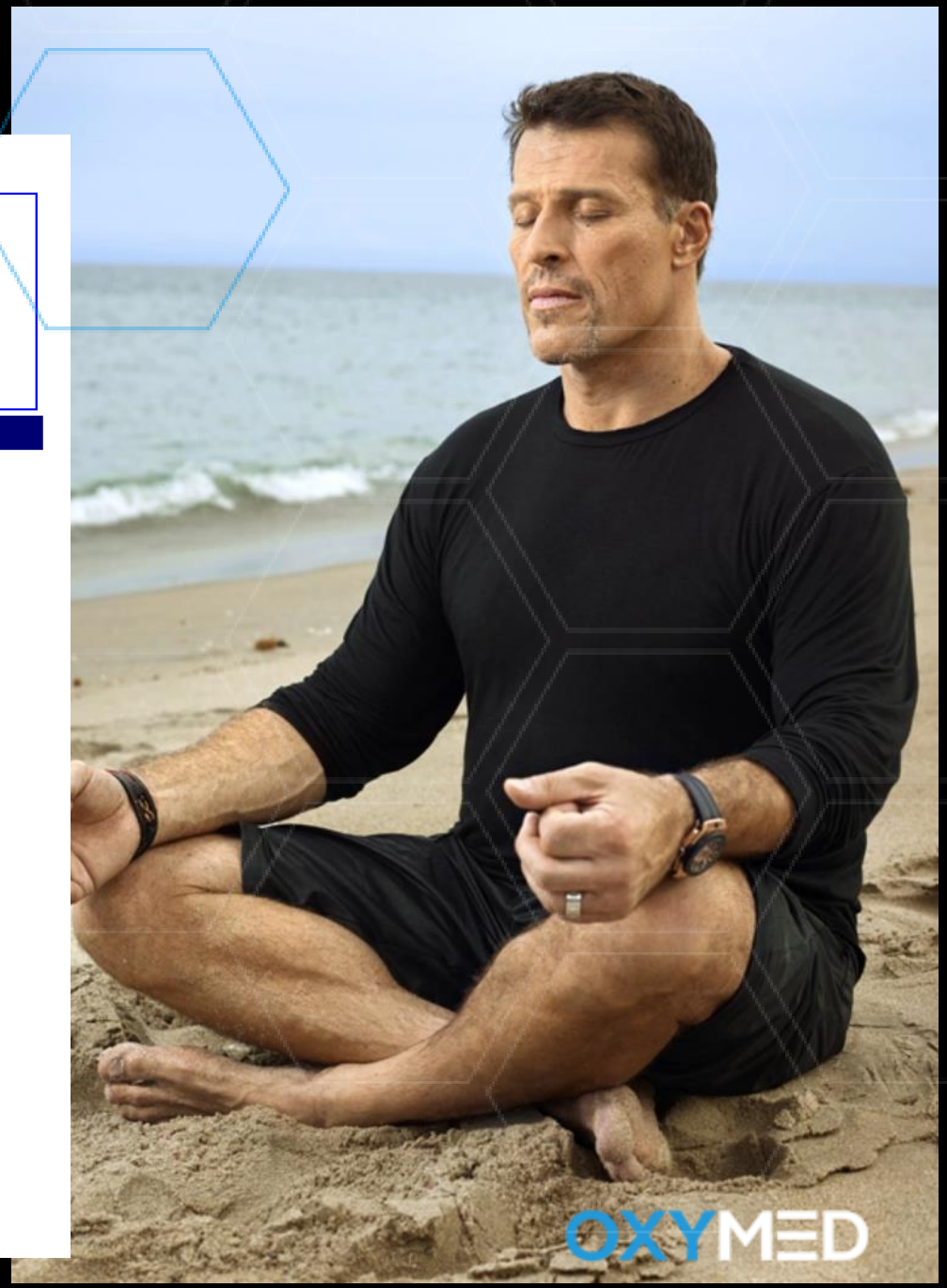
P: 1300 688 522
E: info@nutripath.com.au
A: PO Box 442 Ashburton VIC 3142

Date of Birth : 29-Feb-1960
Sex : M
Collected : 28-May-2018
643 CHAPEL STREET
SOUTH YARRA VIC 3141
Lab id: **3541170** UR#:

OXYMED, 643 CHAPEL STREET
SOUTH YARRA VIC 3141

INTEGRATIVE MEDICINE

BLOOD - SERUM	Result	Range	Units
CYTOKINES,Extensive Panel			
ProInflammatory Cytokines (TH1)			
Interleukin 1	*4.3 *H*	0.0 - 2.8	pg/mL
Interleukin 6	4.3	0.0 - 11.0	pg/mL
Interleukin 7	7.7	0.0 - 16.0	pg/mL
Interleukin 8	*674.0 *H*	0.0 - 28.0	pg/mL
Interleukin 17	12.3	< 13.0	pg/mL
TNFa	*18.60 *H*	0.00 - 13.00	pg/mL
TNFb	134.0	0.0 - 156.0	pg/mL
S100B	*12.0 *L*	60.0 - 100.0	pg/mL
AntiInflammatory Cytokines (TH2)			
GM-CSF	78.8	0.0 - 80.0	pg/mL
Interleukin 2	5.1	0.0 - 10.0	pg/mL
Interleukin 3	1.4	< 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	*7.5 *H*	0.0 - 7.0	pg/mL
Interleukin 12	2.5	0.0 - 14.0	pg/mL
Interleukin 13	*10.1 *H*	0.0 - 6.0	pg/mL
INFg	13.8	0.0 - 28.0	pg/mL
TGFb	38.0	28.0 - 64.0	pg/mL
Brain Derived Neurotrophic Factor BDNF	*72.0 *H*	20.0 - 50.0	ng/mL



Cytokine Gene Expression Testing

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’?

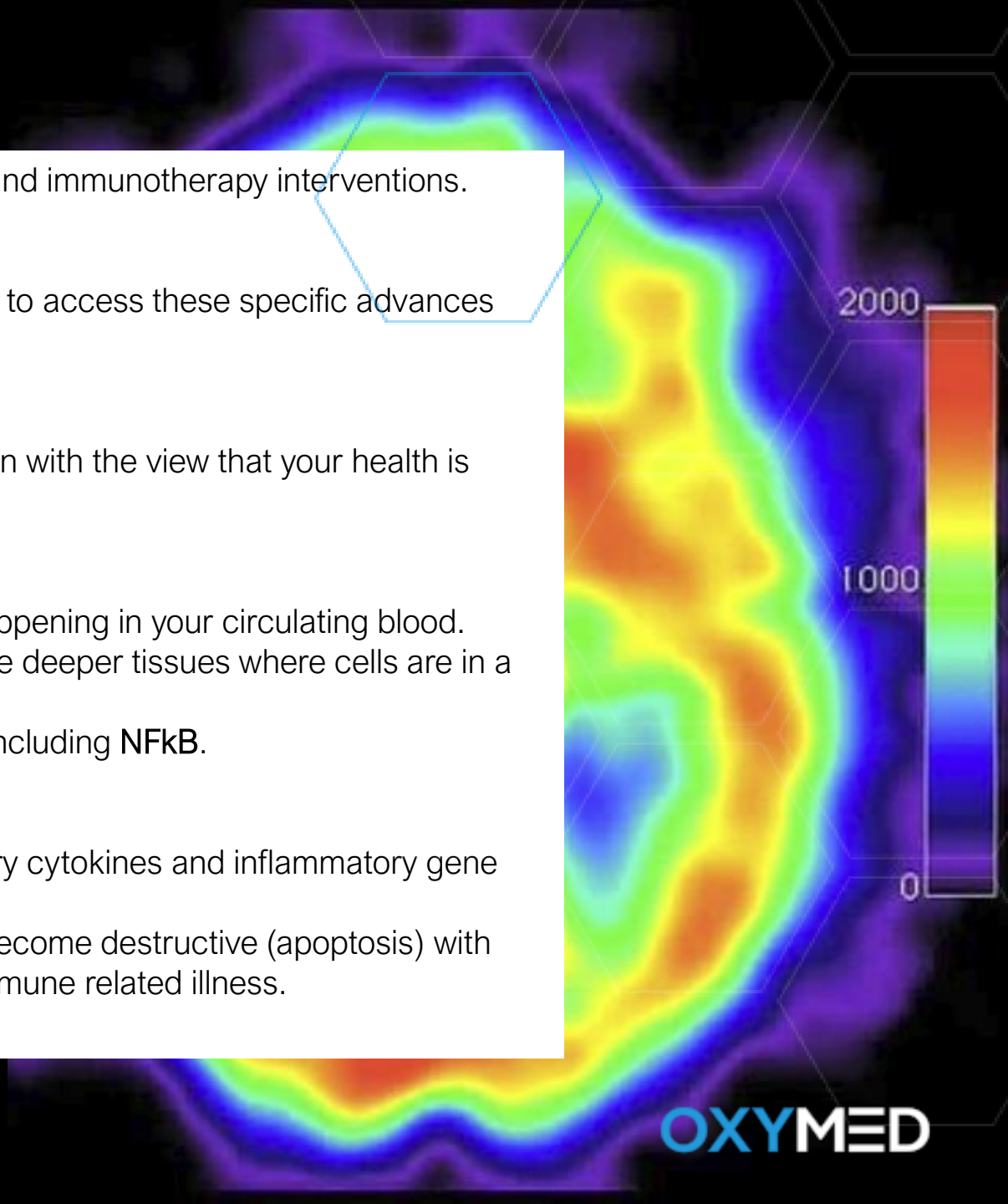
The answer is simple.

- 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.
- There are also a number of additional markers that we are working on including **NFkB**. https://www.oxymed.com.au/inflammaging-nfkb

- 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.



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Pro-inflammatory Cytokines – IL1

Interleukin 1 (IL1)
NOW 4.3 – Previously Normal (0-2.8)
https://www.oxymed.com.au/interleukin-1

- 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.
- Elevations in serum levels and joint fluids (synovial fluids) are detected in rheumatoid arthritis.

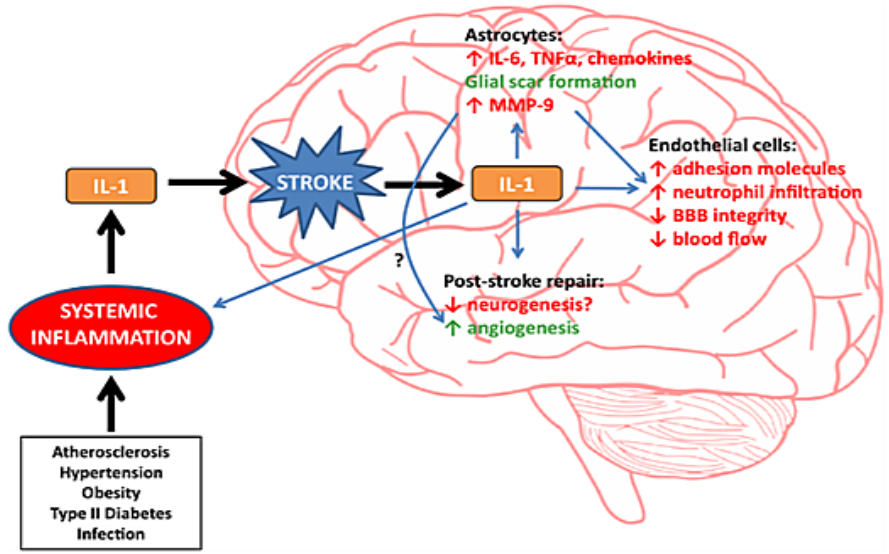

Figure. Mechanisms of interleukin-1 (IL-1) action in stroke. Stroke-related comorbidities and risk factors are associated with a raised systemic inflammatory profile, mediated in part by IL-1. Post-stroke increases in IL-1 in the brain mediate detrimental (indicated in red) inflammatory processes in 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.

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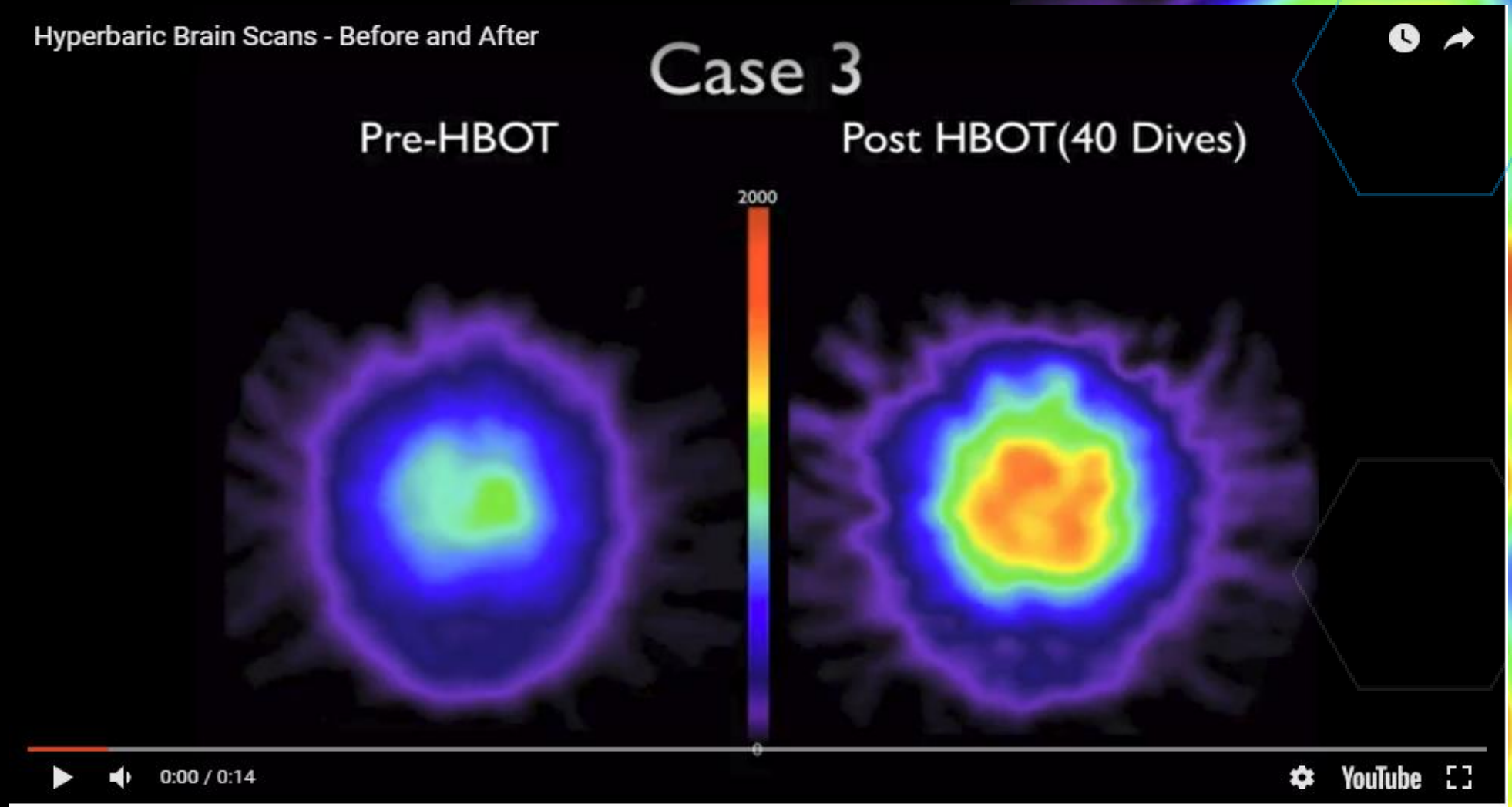
OXYGENE - The Power Within



"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

2000
1000
0

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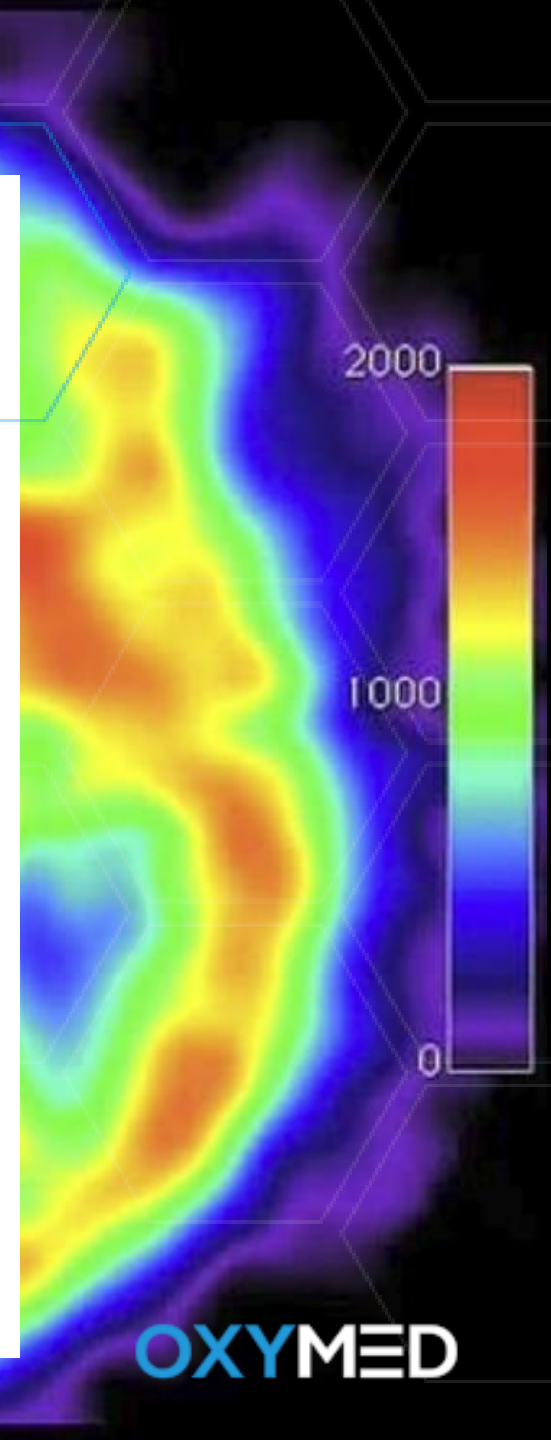
Pro-inflammatory - IL6, IL7

Interleukin 6 (IL6)
Normal
https://www.oxymed.com.au/interleukin-6

- 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).

Interleukin 7 (IL7)
Normal
https://www.oxymed.com.au/interleukin-7 An important marker in cancer activity.



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Pro-inflammatory – IL8

Interleukin 8 (IL8)
NOW 674 – Previously 164.7 - Very High (normal reference range 0-28).
https://www.oxymed.com.au/interleukin-8

- IL8 is a significant inflammatory marker, and certainly in combination with elevated IL1, IL6, TNFa, S100B.
- IL8 associated with circulatory (blood vessel) inflammation including cerebrovascular and cardiovascular disorders. It is associated with neurodegenerative and autoimmune related disorders including cancers etc.

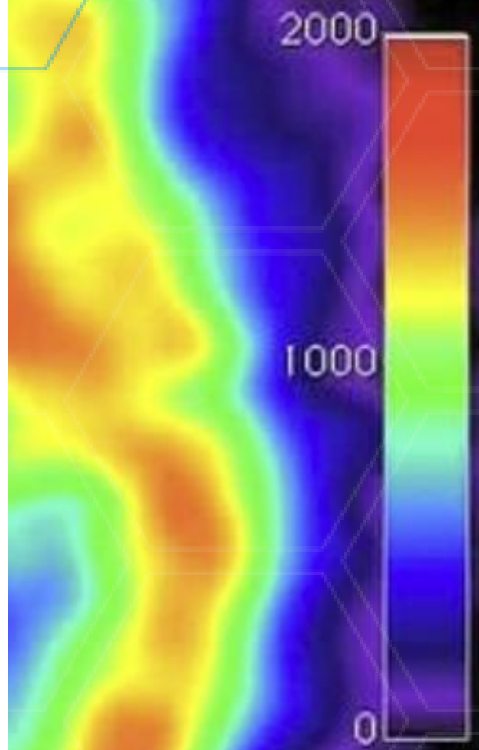
Chemokine Interleukin-8 (IL-8) in Alzheimer's and Other Neurodegenerative Diseases

- Neuroinflammation is a critical component in the pathogenesis of neurodegenerative diseases. Evidence suggests that activated microglia serve as a primary source for a host of inflammatory mediators, leading to chronic neurotoxicity in the inflamed brain. Mobilization of chemokine factors is a response to changes in brain homeostatic conditions leading to localized accumulation of reactive microglia at target sites. In particular, levels of the chemokine IL8 are significantly elevated in neurodegenerative disease.

IL8 & Cardiovascular Disease

- IL8 is associated with the inflammatory basis of atherosclerosis, and a potential mediator of the biochemical pathways towards lesion formation. IL8 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- in the establishment of the inflammatory micro-environment of the insulted vascular wall.

- 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.



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Pro-inflammatory – IL8

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

EUROPEAN SOCIETY OF CARDIOLOGY®

Review

Interleukin 8 and cardiovascular disease

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

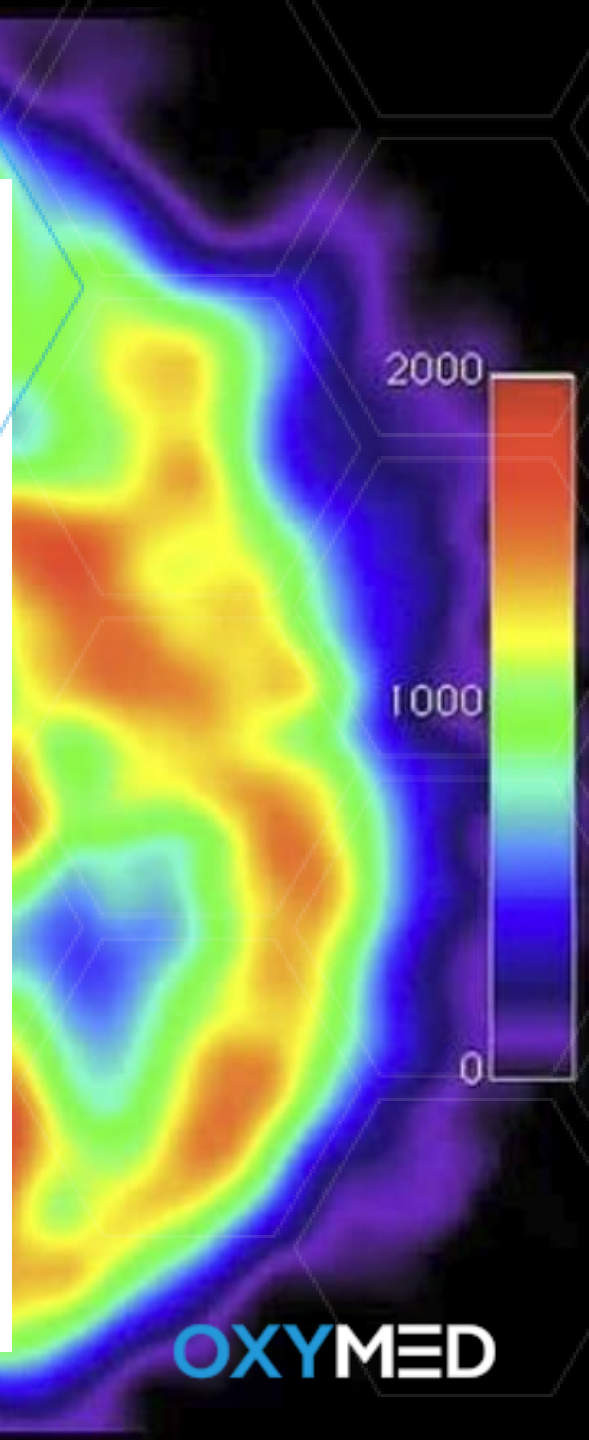
Laboratory of Clinical Virology, Faculty of Medicine, University of Crete, 71409 Heraklion, Crete, Greece

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.



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Pro-inflammatory – IL8

Journal of Alzheimer's Disease & Parkinsonism

McLarnon, J Alzheimers Dis Parkinsonism 2016, 6:5
DOI: 10.4172/2161-0460.1000273

Mini Review Open Access

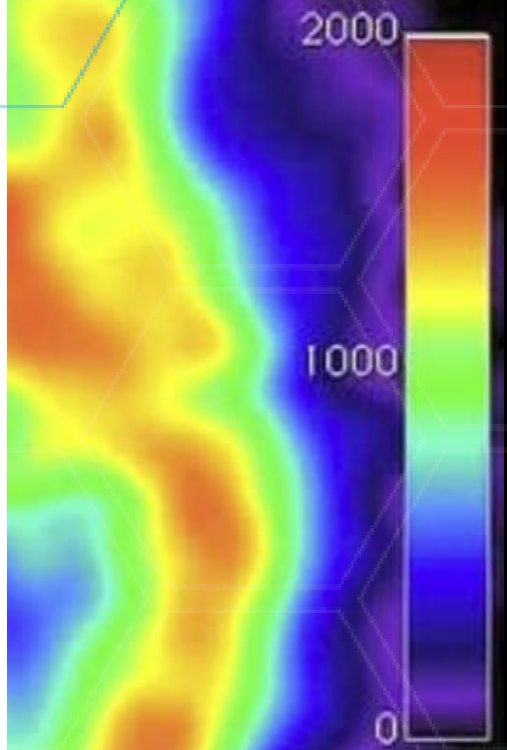
Chemokine Interleukin-8 (IL-8) in Alzheimer's and Other Neurodegenerative Diseases

James G McLarnon*

The University of British Columbia, Vancouver British Columbia, V6T1W3, Canada

Abstract

Interleukin -8 (IL-8), a member of the CXC chemokine family, is well-documented as an important chemotactic signaling factor for recruitment of neutrophils to sites of infection and damage. However in neurodegenerative disease such as Alzheimer's disease (AD), it is the resident macrophage cells, microglia, which are primary responding cells to brain insult such as deposition of amyloid β peptide. IL-8 exhibits an autocrine interaction with microglia by inducing a recruitment of the cells to specific sites of inflammation and subsequent increased production of the chemokine from activated cells. This positive feedback process thus has the capacity to amplify and sustain inflammatory response and brain neuroinflammation. The net result is that a localized and enhanced inflammatory response is induced by accumulating activated microglia at sites of inflammation serving to exacerbate inflammatory reactivity in AD brain. Importantly, under certain conditions the chemotaxis and subsequent activation of microglia may be deleterious to bystander cells including neurons. This review summarizes work from selected studies concerning the involvement and contributions of IL-8 mobilization from activated microglia to brain neuroinflammation as documented in the neurodegenerative diseases Alzheimer's disease (AD), Huntington's disease (HD), amyotrophic lateral sclerosis (ALS) and Parkinson's disease (PD).



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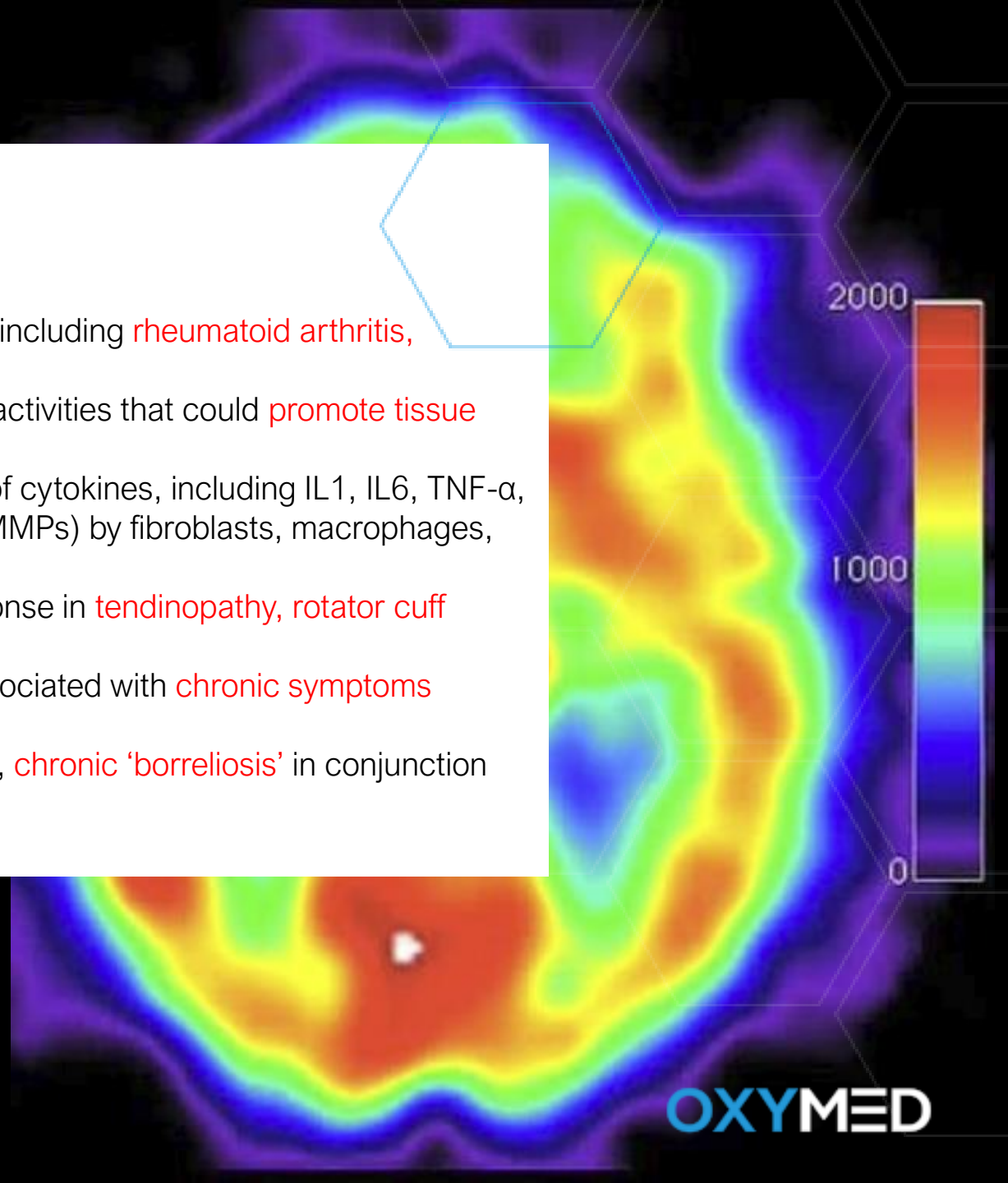
Pro-inflammatory – IL17

Interleukin 17 (IL17)
Normal (<13).
https://www.oxymed.com.au/interleukin-17 - IL17

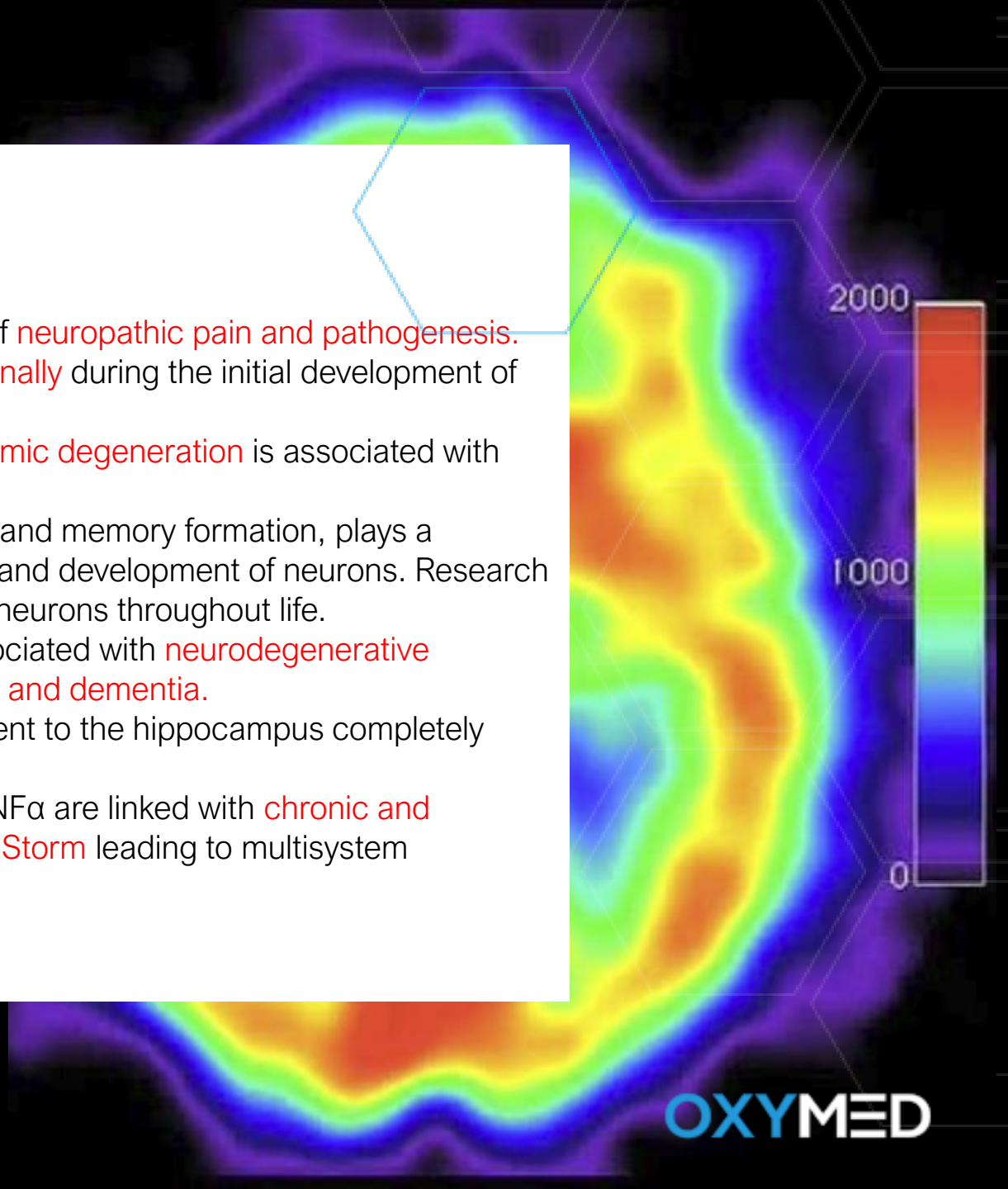
- High levels of IL17 are associated with chronic inflammatory diseases including rheumatoid arthritis, psoriasis and multiple sclerosis.
- Elevation of IL17 also associated with IL17A exerts various biological activities that could promote tissue destruction and degeneration during inflammation.
- In particular, it induces the production, often in a synergistic manner, of cytokines, including IL1, IL6, TNF-α, chemokines, inducible NO synthase, and matrix metalloproteinases (MMPs) by fibroblasts, macrophages, and endothelial cells.
- Experimental models provide evidence for an early inflammatory response in tendinopathy, rotator cuff injuries and repetitive strain injuries.
- 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.



Pro-inflammatory – TNFa

Tumour Necrosis Factor alpha (TNFa)
NOW 18.60 – Marginal (0-13)
https://www.oxymed.com.au/tumour-necrosis-factor-alpha

- Proinflammatory tumor necrosis factor-alpha (TNF) is a key mediator of neuropathic pain and pathogenesis.
- TNF is elevated at sites of nerve injury, in the spinal cord, and supraspinally during the initial development of pain.
- Chronic neuropathic pain associated with crush injury and spinal ischemic degeneration is associated with elevated levels of TNFa, IL1, IL6, IL8.
- The hippocampus part of the brain most notable for its role in learning and memory formation, plays a fundamental role in pain sensation. Neurogenesis refers to the growth and development of neurons. Research has shown that the human hippocampus retains its ability to generate neurons throughout life.
- Elevated levels of TNF in the hippocampal region leads to atrophy associated with neurodegenerative disorders and mental health issues ie depression, psychosis, addiction and dementia.
- Animal studies demonstrate that infusion of an anti -TNFα agent adjacent to the hippocampus completely alleviated pain. Refer to Etanercept.
- 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!



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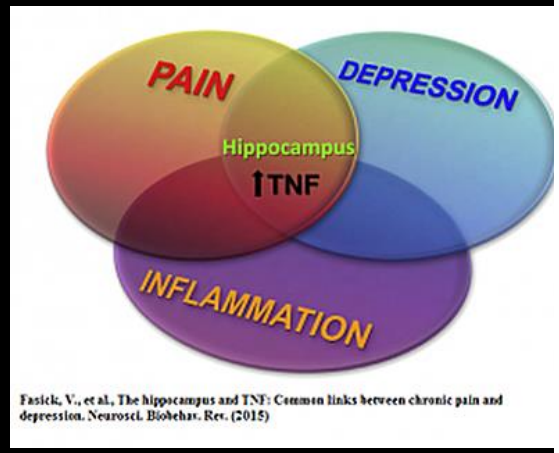
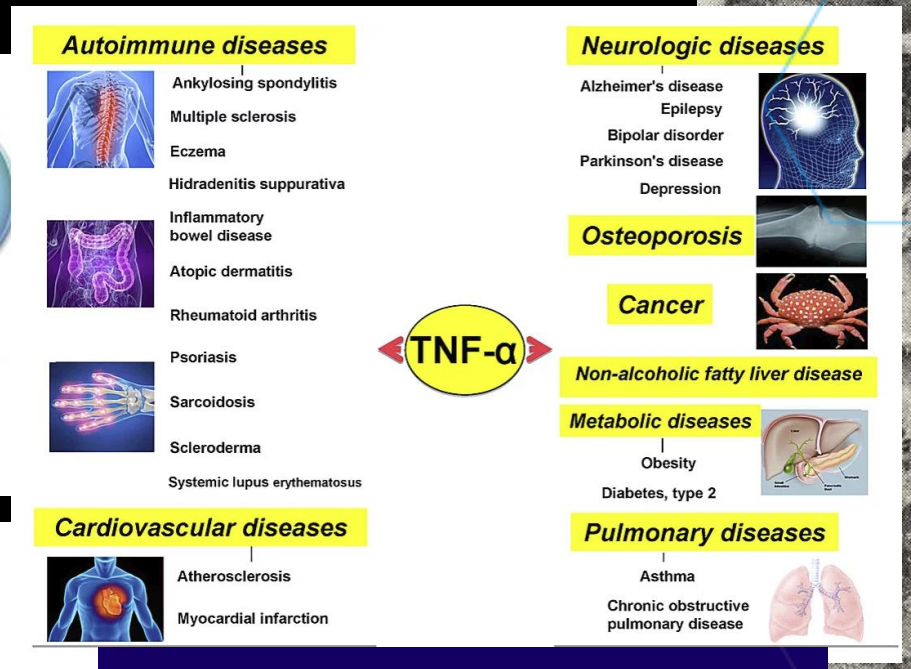
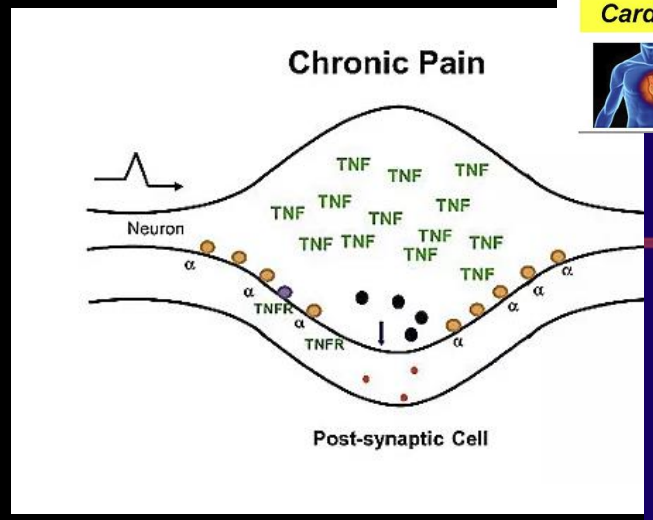
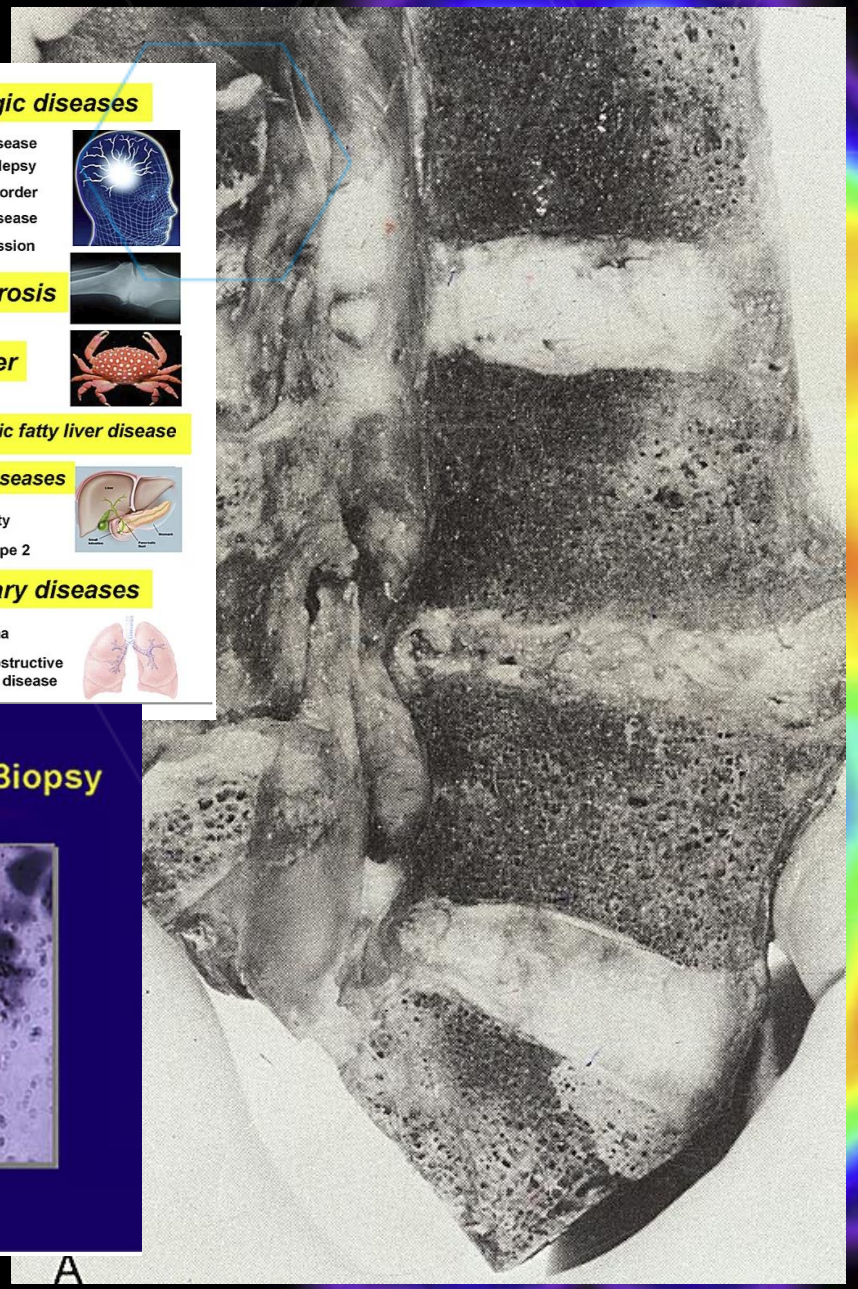
Pro-inflammatory TNFa

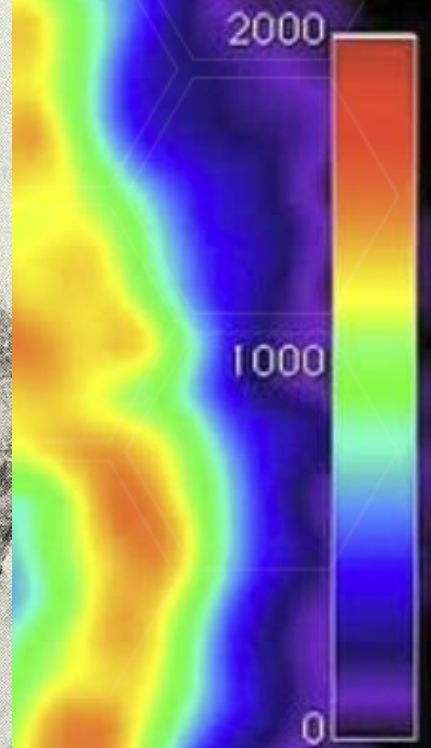


Fasick, V., et al., The hippocampus and TNF: Common links between chronic pain and depression. Neurosci. Biobehav. Rev. (2015)









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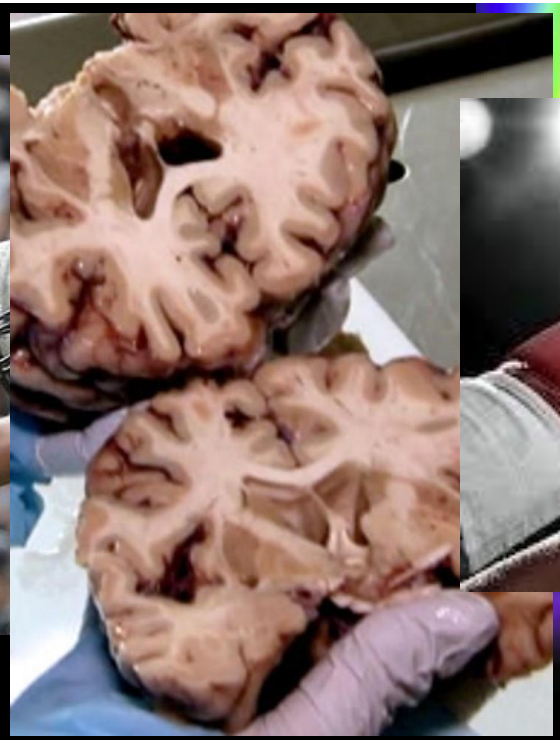
Pro-inflammatory – S100B

S100B
12 – **Low (normal)** (60-100)
https://www.oxymed.com.au/s100b

- S100B is a very comprehensive marker associated with mental health issues, brain disorders but also a marker of other systemic issues.
- S100B is elevated with Traumatic Brain Injury (TBI), Post Traumatic Stress Disorders (PTSD), shock blast injury, concussions syndromes, blunt head injury and progressive neurodegeneration disorders.



OXYMED

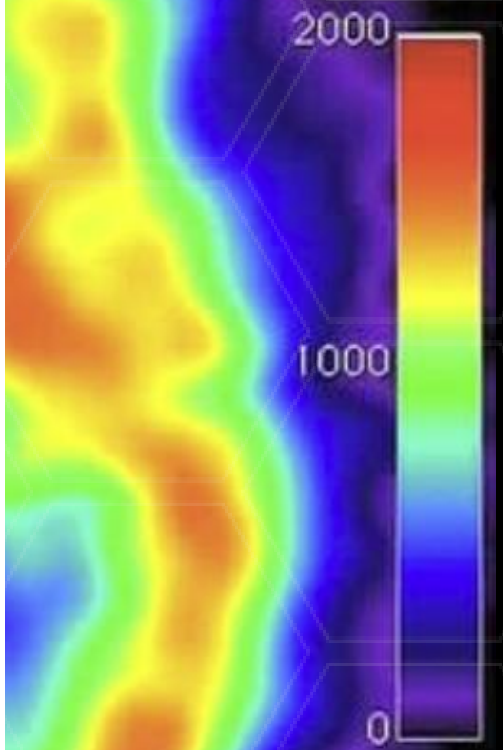
Anti-inflammatory Cytokine Expressions

Granulocyte Macrophage Colony Stimulating Factor (GM-CSF)
Normal (0-80).
https://www.oxymed.com.au/gm-csf

- Regulator of innate immune modulation. GM-CSF is typically a “good-guy” and part of the family of anti-inflammatory gene expressions and function.
- GM-CSF are “growth factors” and specifically make the bone marrow produce blood cells, activating production and mobilisation of stem cells from the bone marrow into the blood circulation. Stem cells are the cells in the bone marrow from which red blood cells, white cells and platelets develop.
- GM-CSF makes the body produce white blood cells to reduce the risk of infection due to immunosuppression after types of cancer treatments.
- GM-CSF attenuated inflammation in the CNS in a mouse model of ALS and delayed the progression of the neurodegenerative disease.
- Chronic elevation of GM-CSF and other adaptive cytokines are observed post vaccines. This is an important finding as the vaccine can cause the child’s immune response to cascade into a **Cytokine Storm** triggering other autoimmune pro-inflammatory reactions including elevation of IL1, IL8, S100B and lowered BDNF.

Sargramostim (GM-CSF) stimulates the production, maturation and activation of (WBC).

- Neutrophils are the most abundant WBC and are the first to respond to infection. Macrophages also capture and digest foreign invaders but are longer acting than neutrophils. Dendritic cells continuously scan their environment and alert other cells when they find something foreign such as an infection.
- Sargramostim can accelerate the recovery of white blood cells (neutropenia) occurring during chemotherapy. Sargramostim is used to stimulate stem cells prior to harvesting for peripheral stem cell transplant, and stimulate recovery of bone marrow cells after bone marrow transplantation.



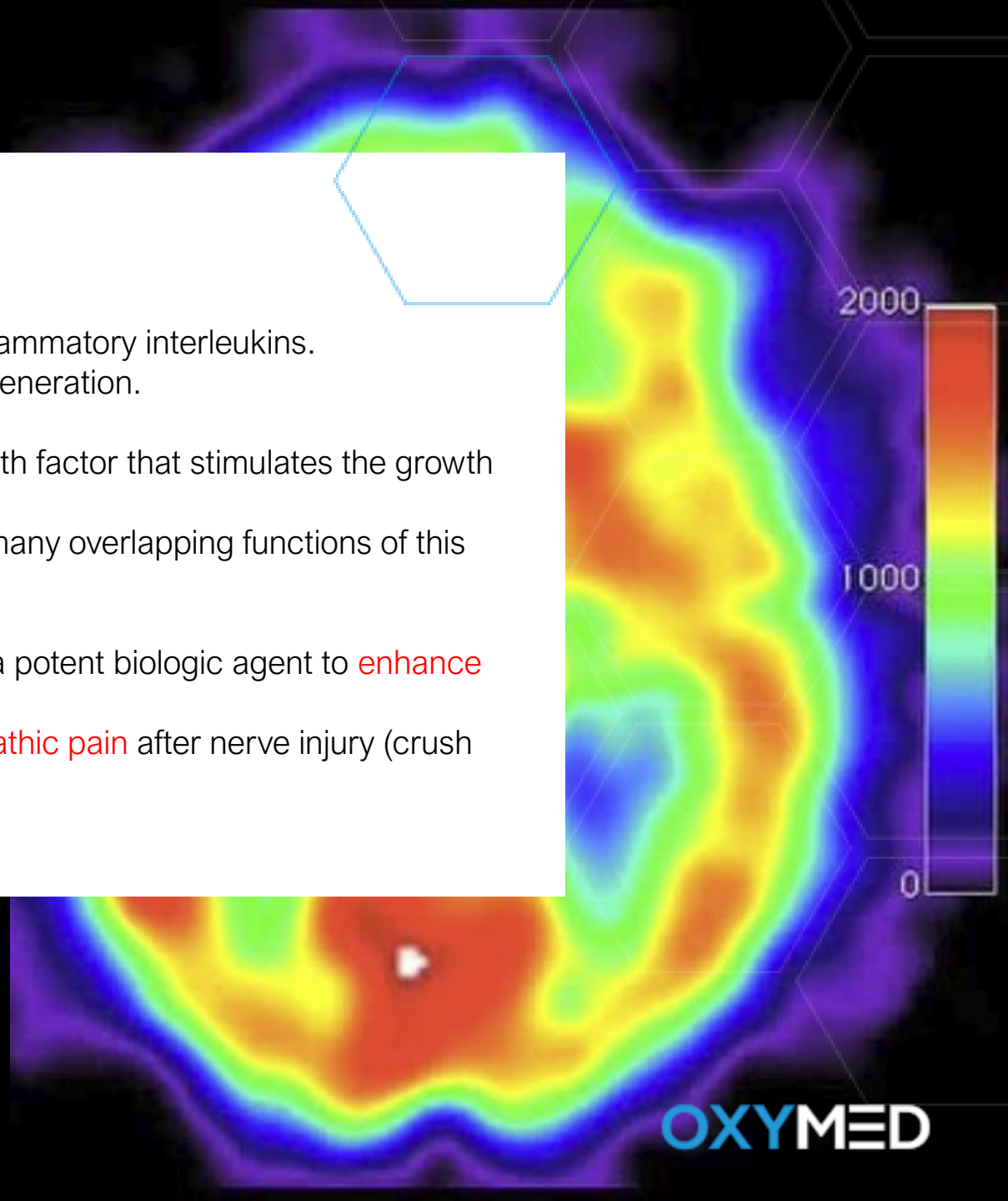
OXYMED

Anti-inflammatory IL4

Interleukin 4 (IL4)
Normal (0-19)
https://www.oxymed.com.au/interleukin-4

- IL4 is a potent anti-inflammatory cytokine combining with other anti inflammatory interleukins.
- IL4 promotes survival, growth, and differentiation adding repair and regeneration.
- In macrophages, IL4 can inhibit the production of TNF, IL1, and IL6.
- IL4 is an immune-stimulating molecule and a potent lymphoid cell growth factor that stimulates the growth and survivability of certain B cells and T cells.
- The interleukin 4 receptor also binds to IL13, which may contribute to many overlapping functions of this cytokine and IL13.

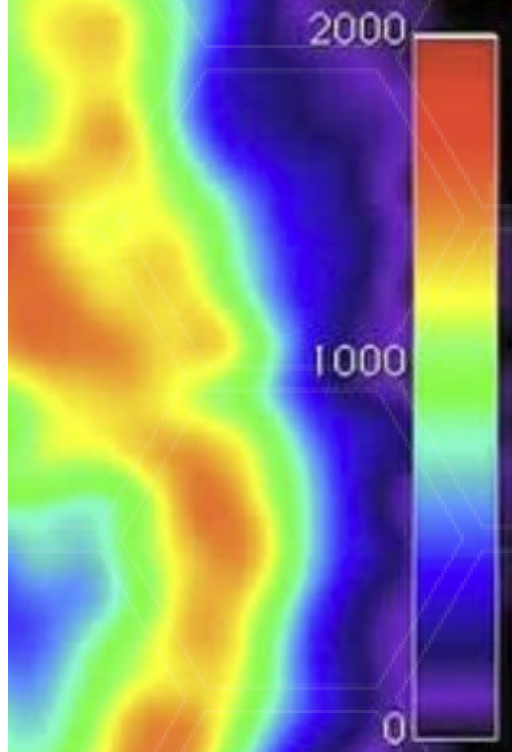
- IL4 has striking antitumor activities with the possibility that IL4 may be a potent biologic agent to enhance immune elimination of certain tumor cells.
- IL4 ameliorates 'non-resolving neuro-inflammation' that causes neuropathic pain after nerve injury (crush injury), failed surgery, complex regional pain syndromes, disc prolapse.
- It is closely related and has functions similar to Interleukin 13.



Anti-inflammatory IL10

Interleukin 10 (IL10)
7.5 - Slight Elevation (0-7)
https://www.oxymed.com.au/interleukin-10

- IL10 is a potent anti-inflammatory TH2 cytokine that has a critical role in limiting the immune response to pathogens to prevent host damage.
- IL10 is a strong inhibitor of inflammation.
- IL10 elevates in response to 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 of IL10 in suppressing rejections of grafts after organ transplantations. IL10 is capable of inhibiting synthesis of pro-inflammatory cytokines. It also displays a potent ability to suppress the antigen-presentation capacity of antigen presenting cells. However, it is also stimulatory towards certain T cells and mast cells and stimulates B cell maturation and antibody production.
- Knockout studies suggested the function of IL10 as an essential immunoregulator in the intestinal tract. Patients with Crohn's disease react favourably towards treatment with bacteria producing recombinant IL10, showing the importance of IL10 for counteracting excessive immunity in the human body.
- The anti-inflammatory cytokine IL10 is capable to induce a resistance of the brain cells to ischemia-evoked damages in in vivo and in vitro models of the ischemic insults in rats. This protective effect in cultured hippocampal cells is developed rapidly after application of IL10 and strongly associated with the IL10 elicited elimination of [Ca(2+)] in response to ischemia.
- Thus, our results provide the evidence that anti-inflammatory cytokine IL10, in addition to an activation of the canonical signalling pathways, is capable to exert the rapid neuroprotective effects through transcription-independent modulation of ischemia-induced intracellular Ca(2+) responses in the brain cells.



OXYMED

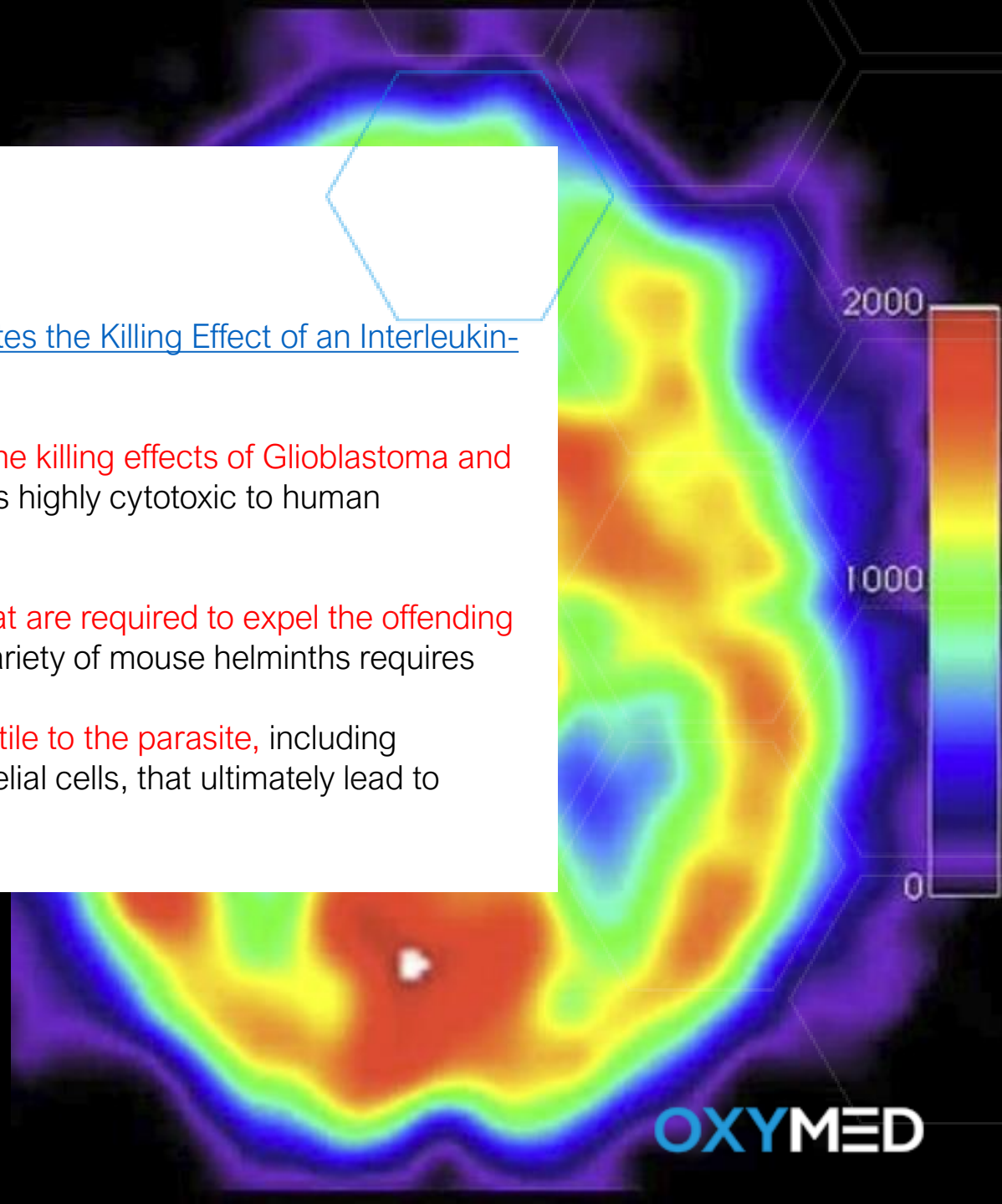
Anti-inflammatory IL13

Interleukin 13 (IL13)
10.1 – Slight Elevation (0-6)
https://www.oxymed.com.au/interleukin-13

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

- 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.

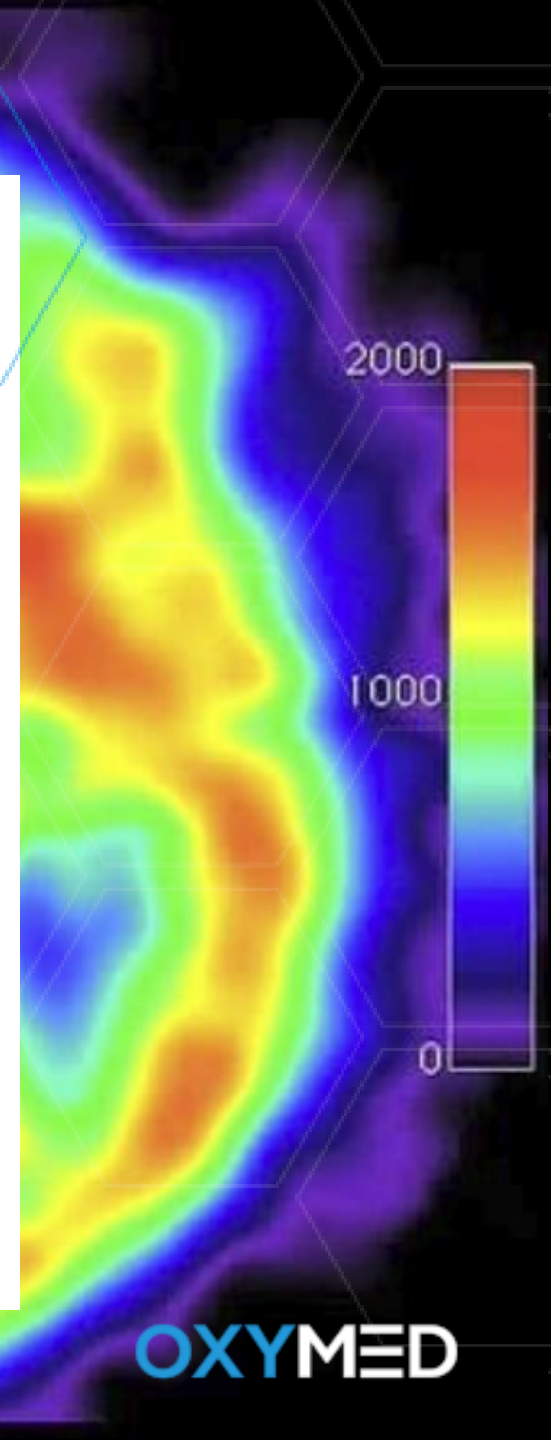
- IL13 specifically induces physiological changes in parasitized organs that are required to expel the offending organisms or their products. For example, expulsion from the gut of a variety of mouse helminths requires IL13 secreted by Th2 cells.
- IL13 induces several changes in the gut that create 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.



Brain Derived Neurotrophic Factor (BDNF)

Brain Derived Neurotrophic Factor (BDNF)
72.0 Elevated (20-50)
https://www.oxymed.com.au/cerebrolysin

- BDNF is a neural growth factor gene.
- Chronic elevation of pro-inflammatory cytokines supresses BDNF functions.
- BDNF is a member of the neurotrophin family of growth factors found in the brain and the periphery.
- BDNF acts on the central nervous system and the peripheral nervous system, helping to support neurogenesis - the survival of existing neurons, and encourage the growth and differentiation of new neurons and synapses.
- In the brain, BDNF is active in the hippocampus, cortex, and basal forebrain—areas vital to learning, memory, and higher thinking. It is also expressed in the retina, motor neurons, the kidneys, saliva, and the prostate.
- BDNF itself is important for long-term memory. Although the vast majority of neurons in the mammalian brain are formed prenatally, parts of the adult brain retain the ability to grow new neurons from neural stem cells.
- Neurotrophins are proteins that help to stimulate and control neurogenesis, BDNF being one of the most active. Mice born without the ability to make BDNF suffer developmental defects in the brain and sensory nervous system, and usually die soon after birth, suggesting that BDNF plays an important role in normal neural development.
- Intensive physical exercise have been shown to markedly (threefold) increase BDNF synthesis in the human brain, a phenomenon which is partly responsible for exercise-induced neurogenesis and improvements in cognitive function.
- Gastrodin, N-Acetyl Cysteine, Niacin (B3) appear to upregulate BDNF.



OXYMED

Inflammaging and Anti-inflammaging

Arch Immunol Ther Exp (Warsz).
2016 Apr;64(2):111-26. doi: 10.1007/s00005-015-0377-3. Epub 2015 Dec 12.

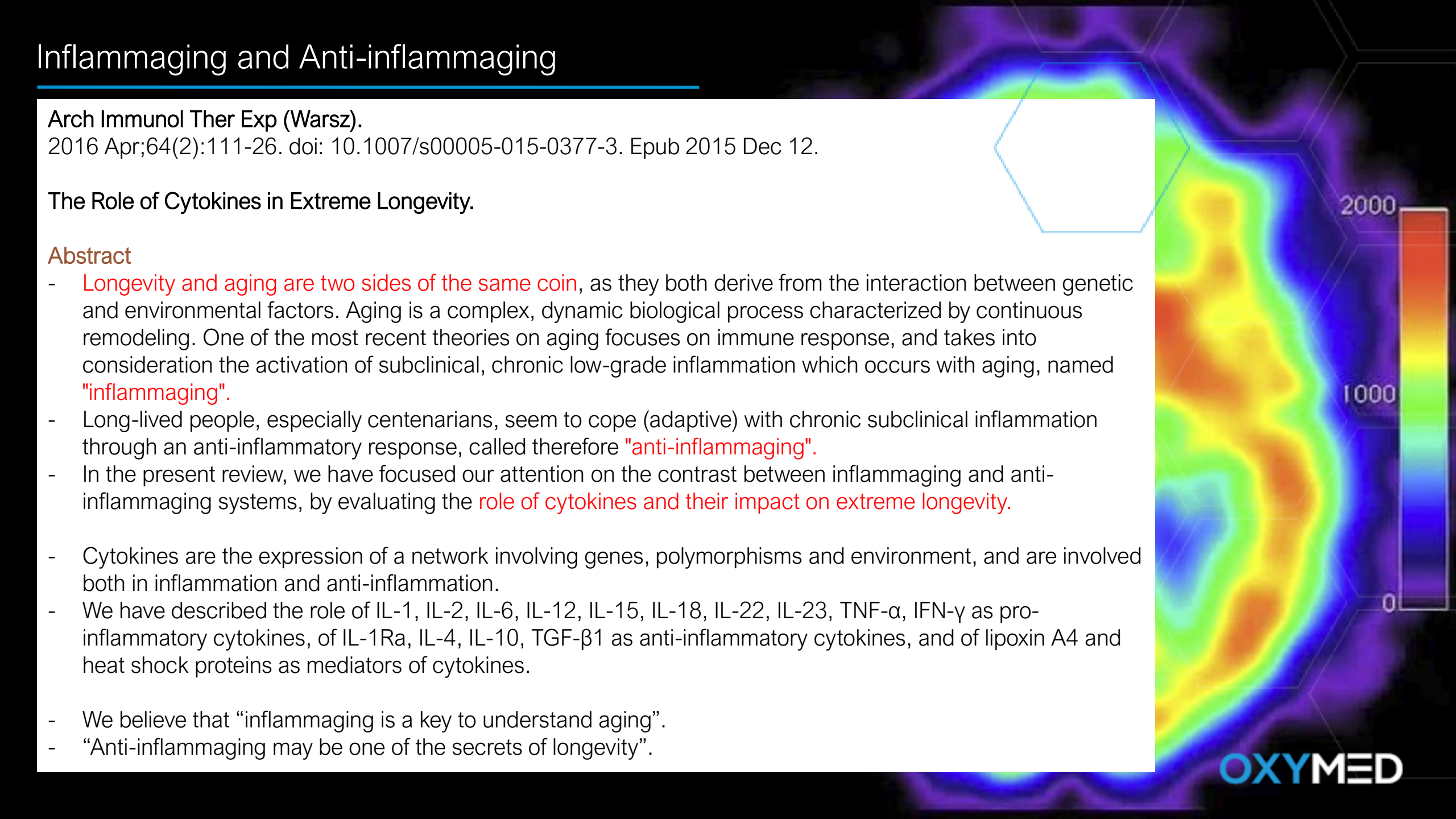
The Role of Cytokines in Extreme Longevity.

Abstract

- Longevity and aging are two sides of the same coin, as they both derive from the interaction between genetic and environmental factors. Aging is a complex, dynamic biological process characterized by continuous remodeling. One of the most recent theories on aging focuses on immune response, and takes into consideration the activation of subclinical, chronic low-grade inflammation which occurs with aging, named "inflammaging".
- Long-lived people, especially centenarians, seem to cope (adaptive) with chronic subclinical inflammation through an anti-inflammatory response, called therefore "anti-inflammaging".
- In the present review, we have focused our attention on the contrast between inflammaging and anti-inflammaging systems, by evaluating the role of cytokines and their impact on extreme longevity.

- Cytokines are the expression of a network involving genes, polymorphisms and environment, and are involved both in inflammation and anti-inflammation.
- We have described the role of IL-1, IL-2, IL-6, IL-12, IL-15, IL-18, IL-22, IL-23, TNF-α, IFN-γ as pro-inflammatory cytokines, of IL-1Ra, IL-4, IL-10, TGF-β1 as anti-inflammatory cytokines, and of lipoxin A4 and heat shock proteins as mediators of cytokines.

- We believe that “inflammaging is a key to understand aging”.
- “Anti-inflammaging may be one of the secrets of longevity”.



OXYMED

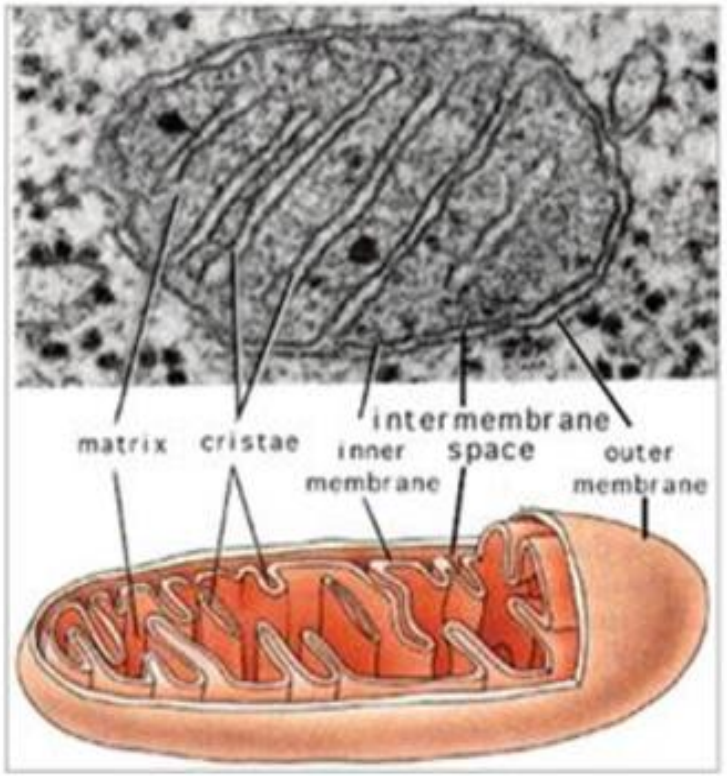
Hypoxic Mitochondrial Dysregulation

Cancer as a metabolic disease: implications for novel therapeutics

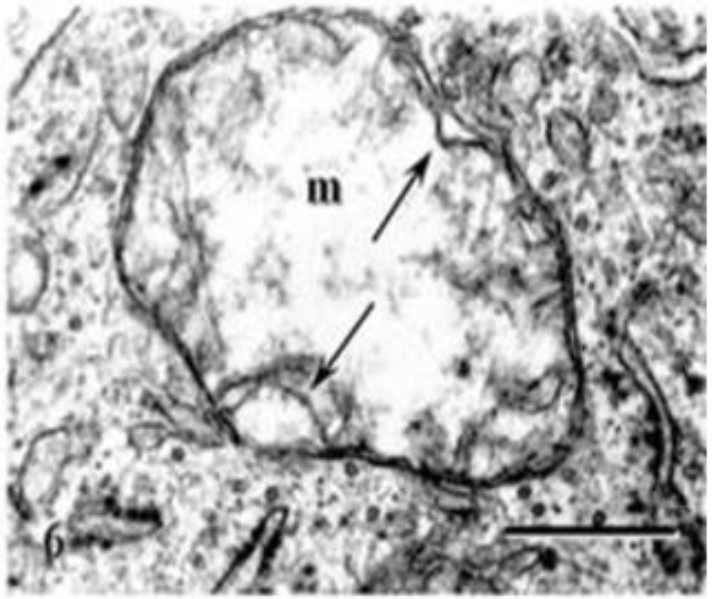
Thomas N. Seyfried ✉, Roberto E. Flores, Angela M. Poff, Dominic P. D'Agostino

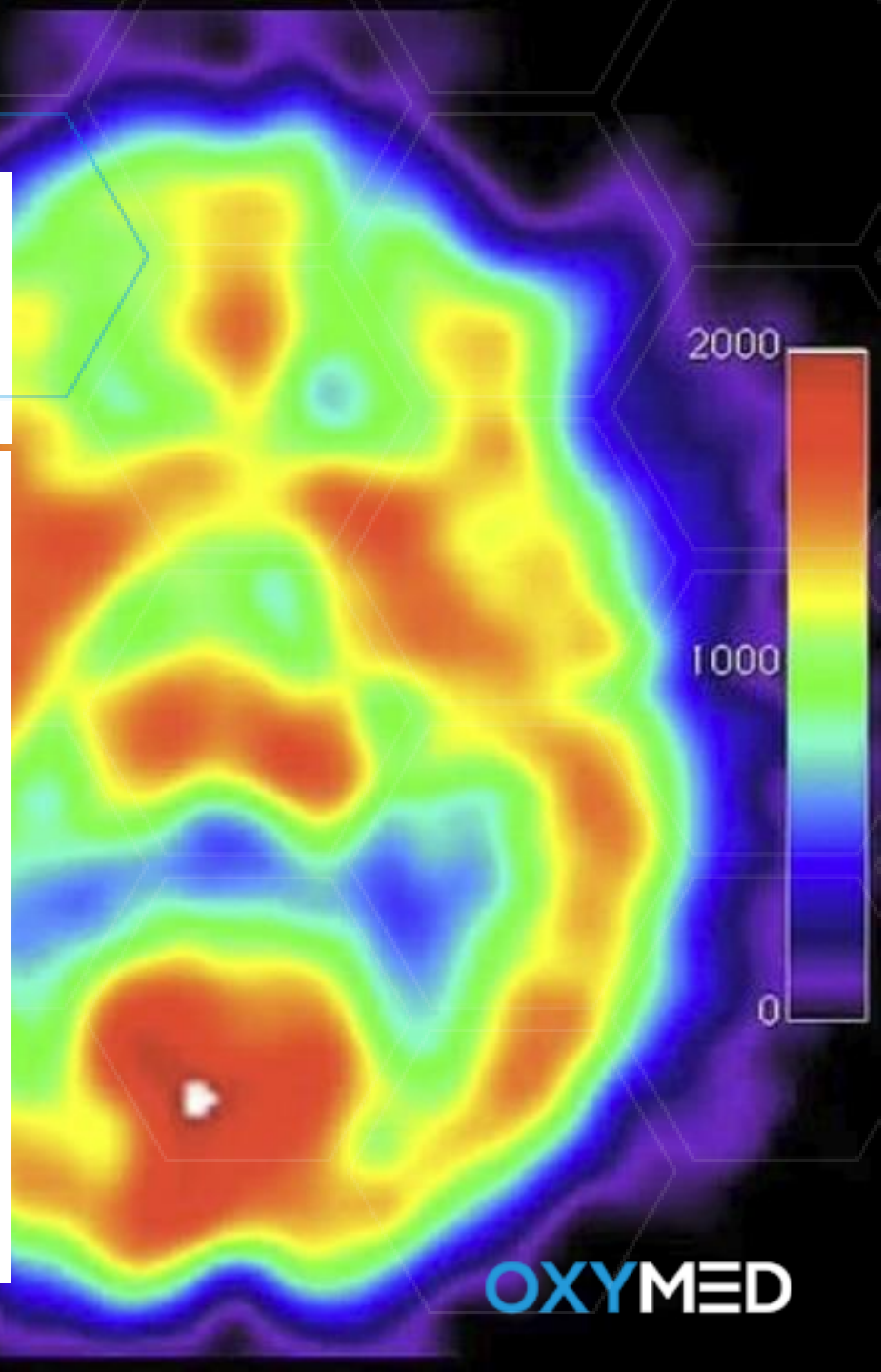
Carcinogenesis, Volume 35, Issue 3, 1 March 2014, Pages 515–527,

Normal Mitochondria



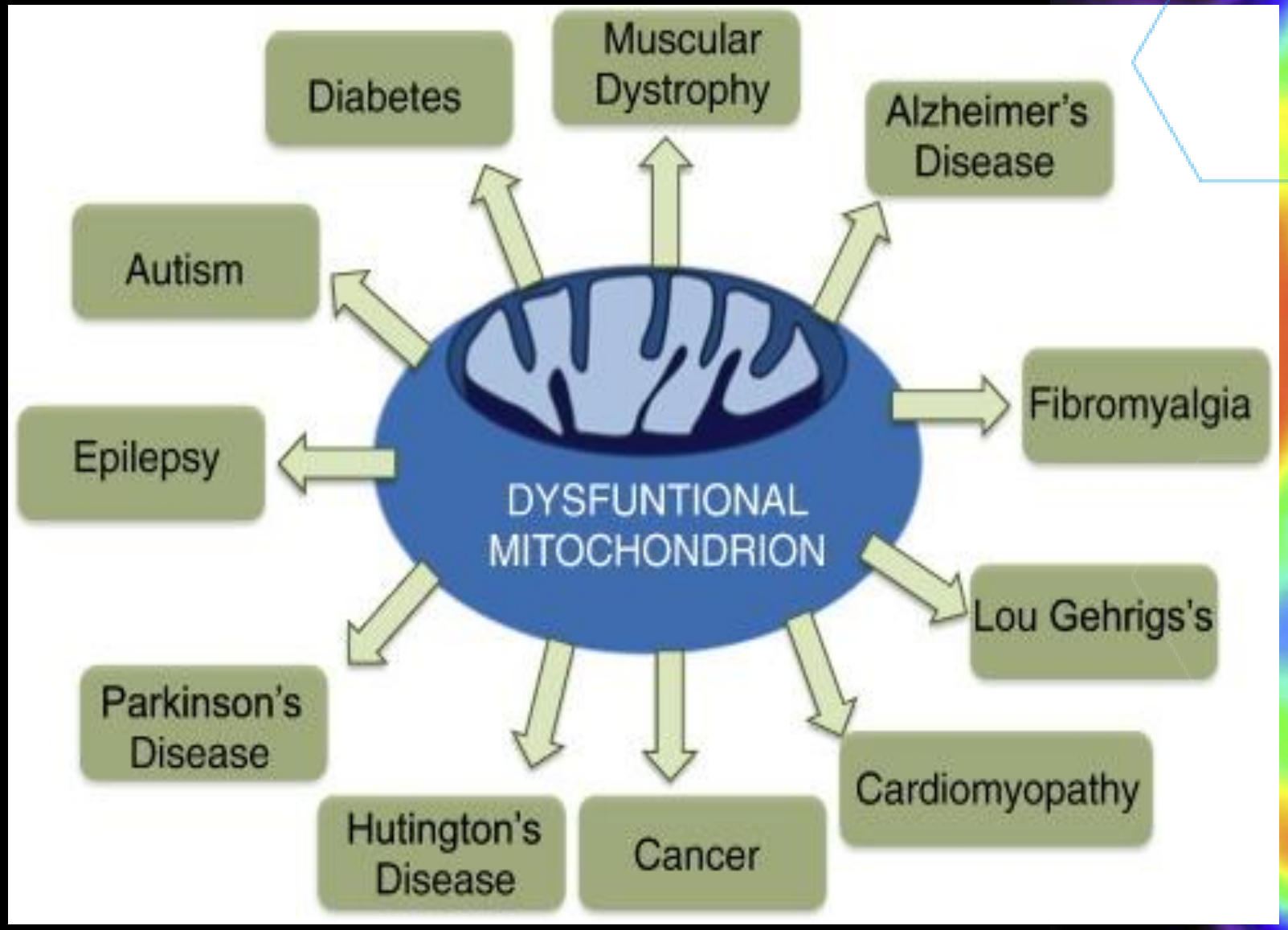
GBM Mitochondria

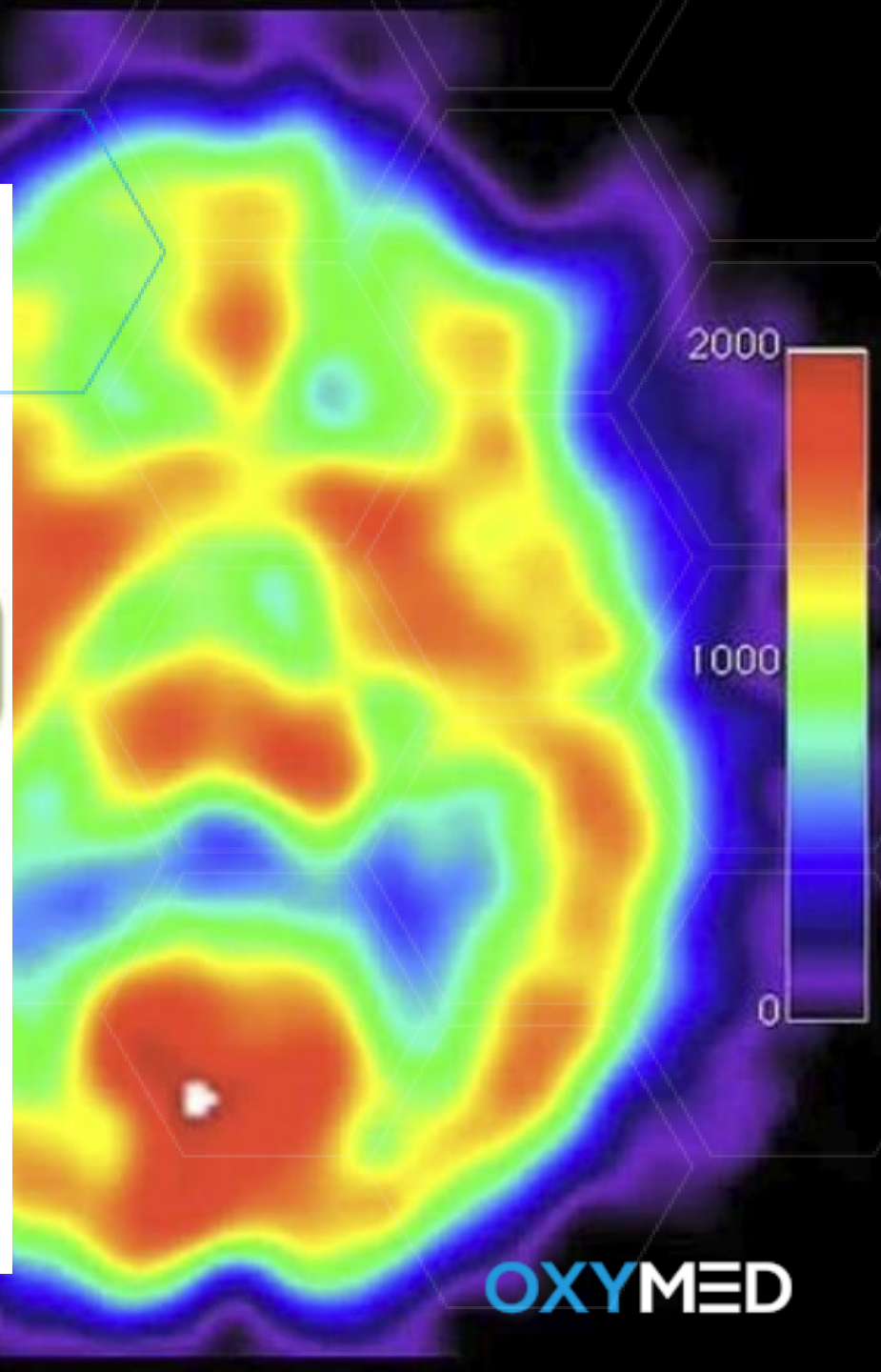




OXYMED

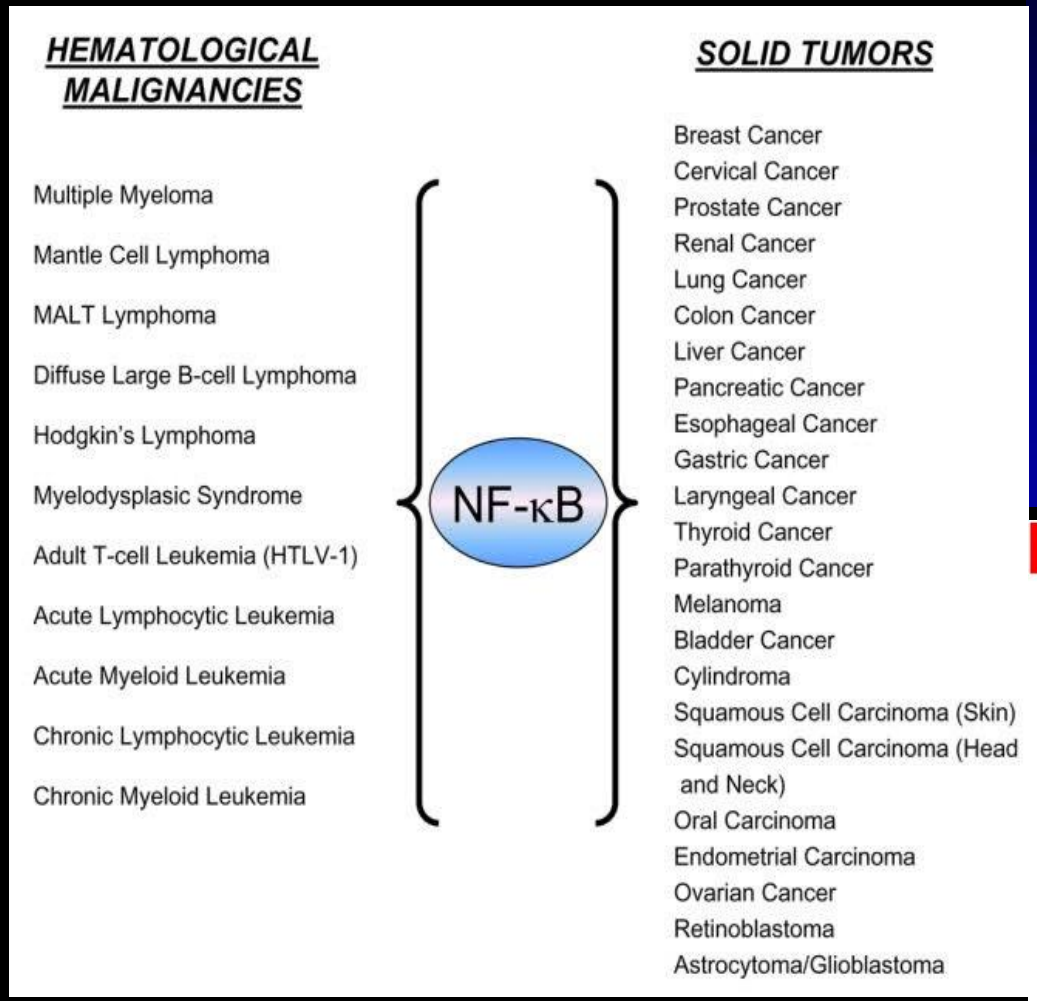
Mitochondrial Dysregulation

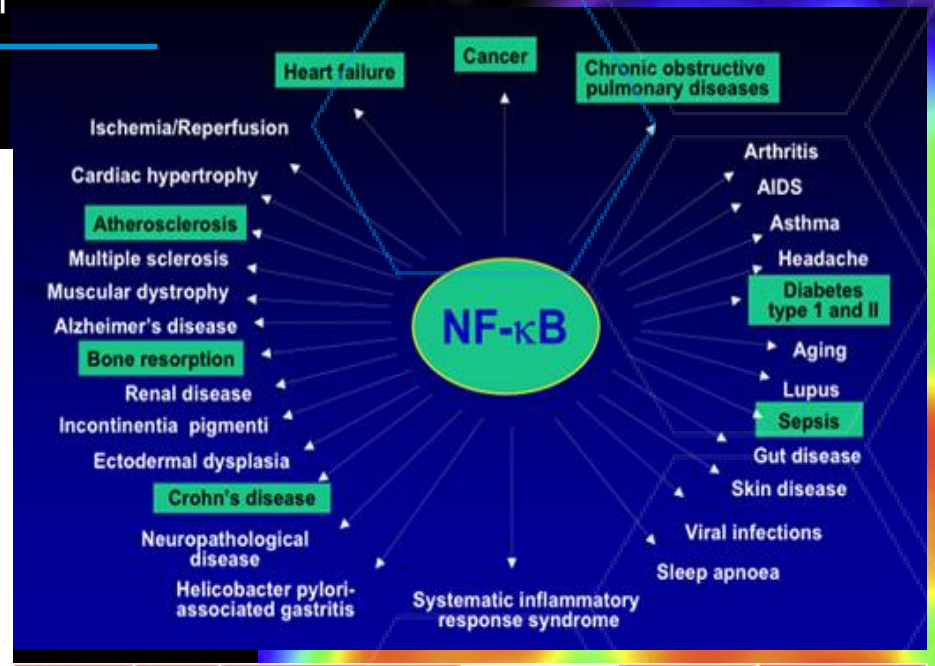


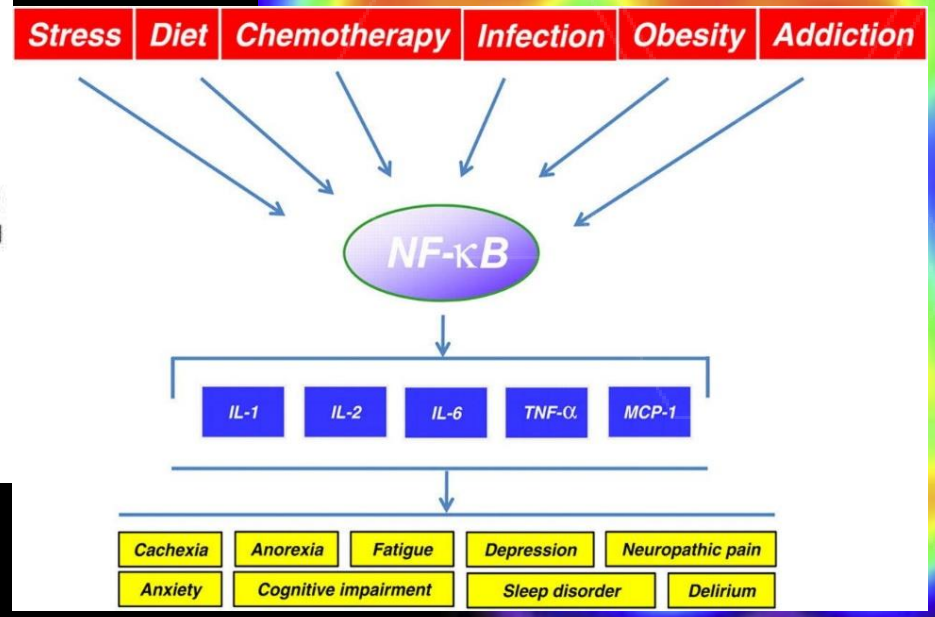


OXYMED

NfKB – Master Regulator of Inflammation







OXYMED

The Perfect - Cytokine Storm

Role of Cytokines and Cytokine Inhibitors in Chronic Inflammation

TNFα
IL-1

IFNγ
GM-CSF
IL-8 and other chemokines

IL-15
IL-16
IL-17
IL-18

TGFβ
IL-6

IL-1RA
sIL-1R1

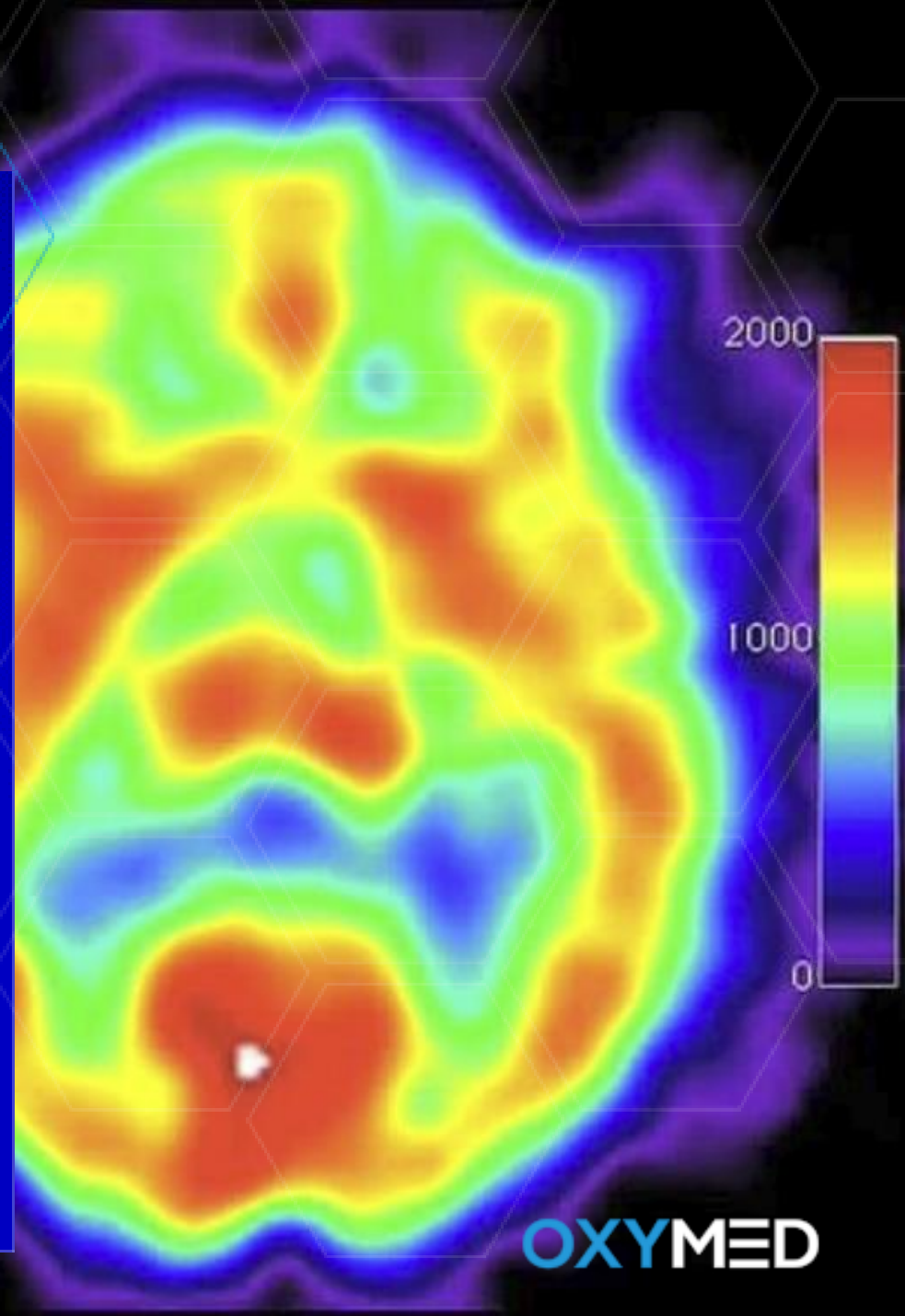
sTNF-R
Monoclonal antibody to TNF

IL-4
IL-10
IL-11
IL-13
IL-18BP

Pro-inflammatory

Anti-inflammatory

Arend. *Arthritis Rheum* 2001.



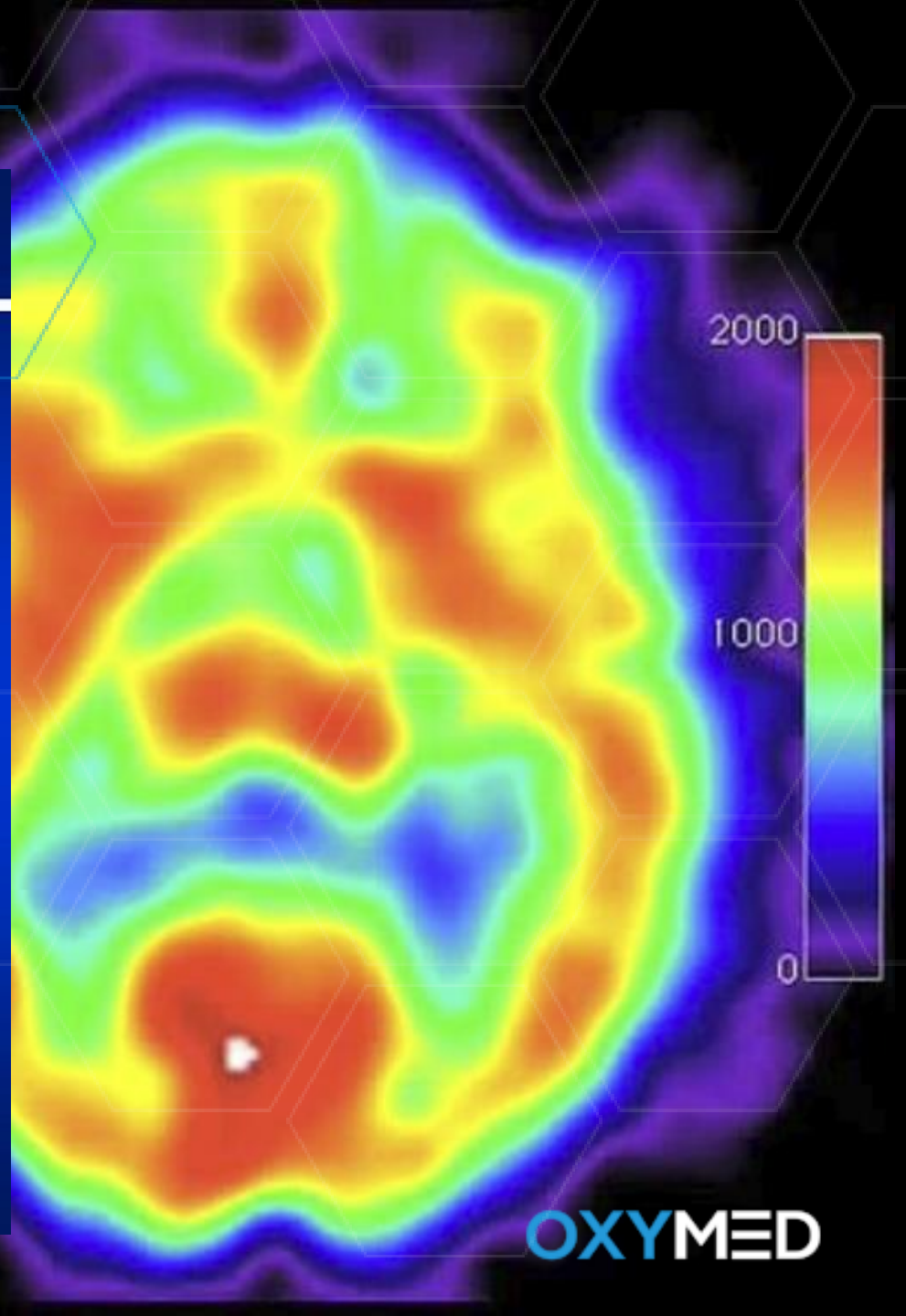
OXYMED

Small start systemic spread

But Cytokine Storm Masks the Injury Site

Cytokine Storm makes the entire body looks inflamed, injured and infected

CytoSorbents



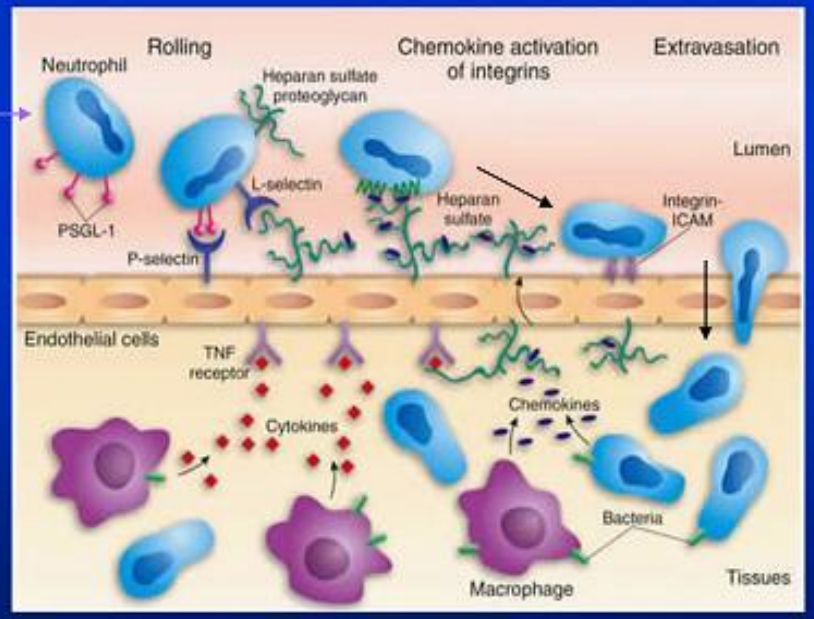
OXYMED

Cytokine Storm equals Immune Confusion

Cytokine Storm Also Causes "Immune Confusion"

An injury or infection usually leads to the local production of cytokines. Normally, these cytokines allow white blood cells to home in on the infection and migrate from the blood stream into the affected area.

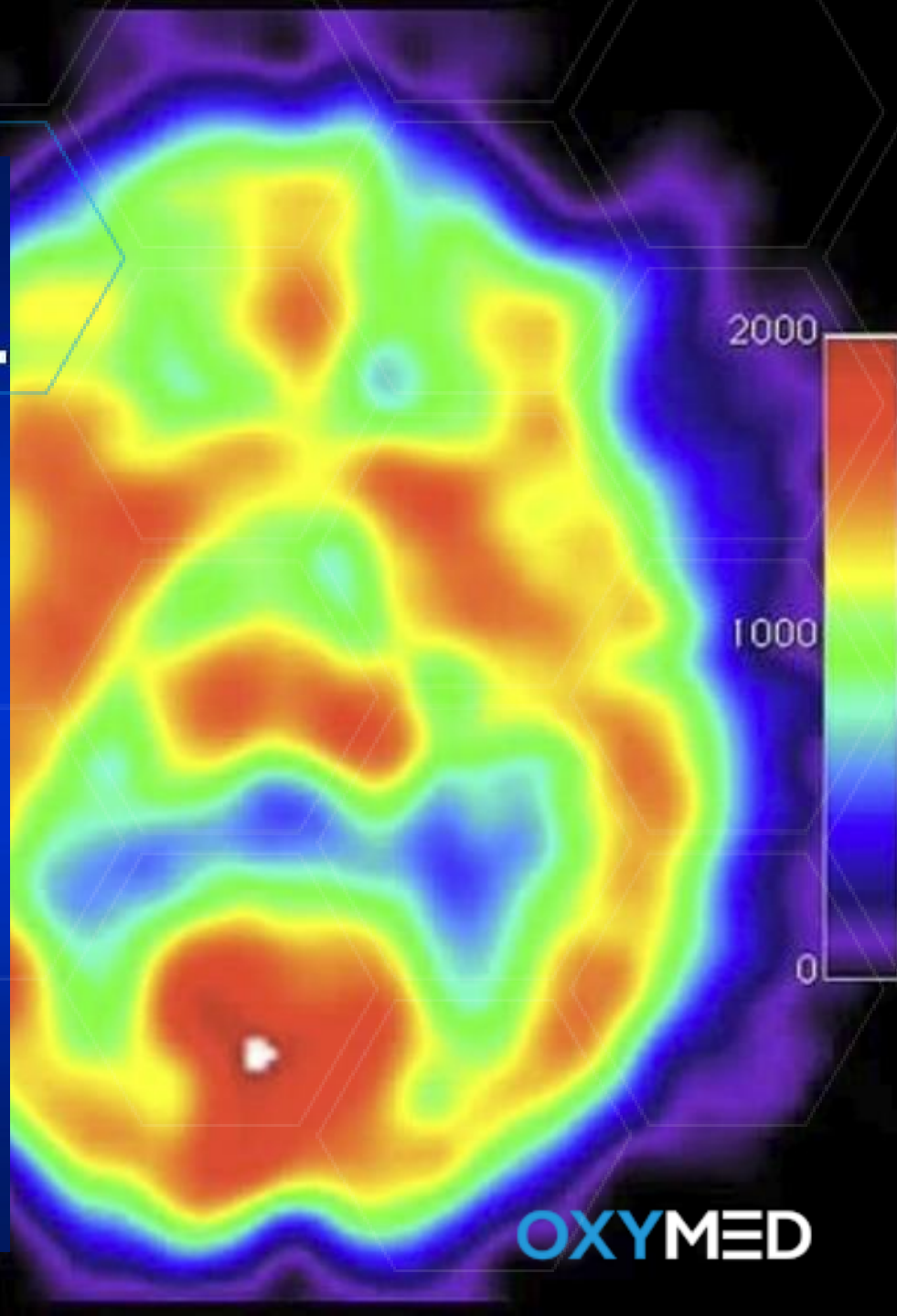
White Blood Cell →



Blood Vessel (blood flow →)

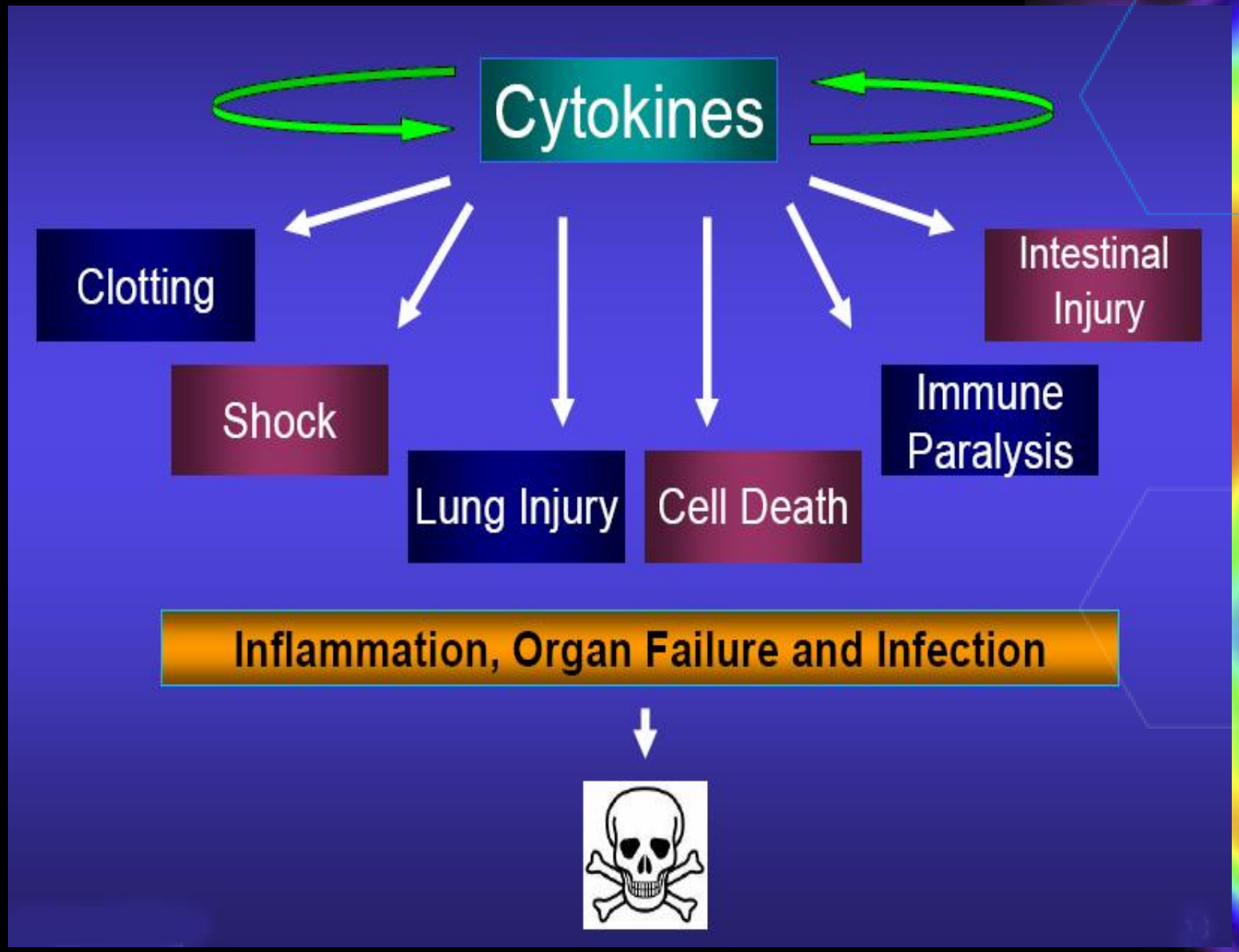
Tissue

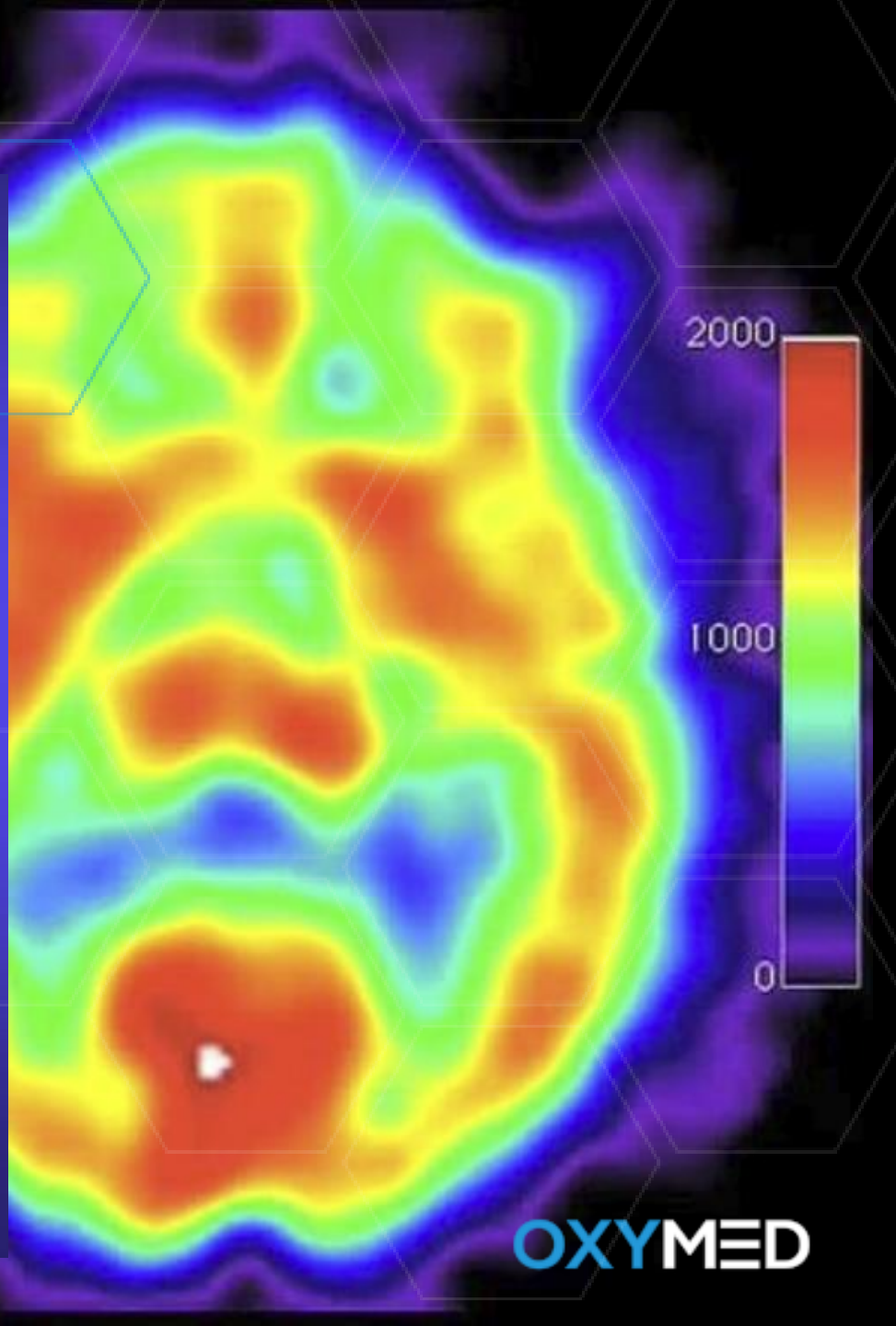
CytoSorbents



OXYMED

Cytokine Storm

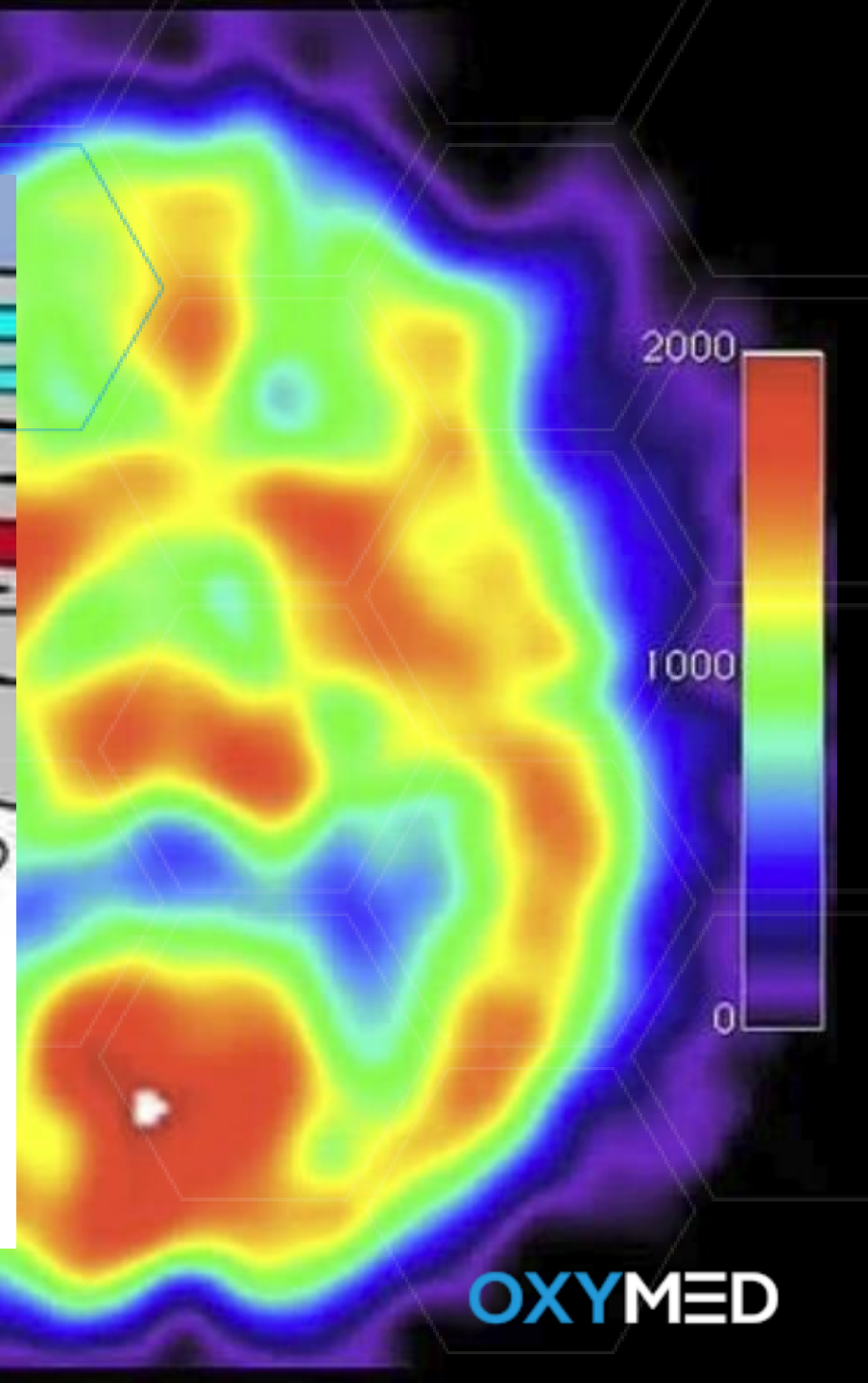




OXYMED

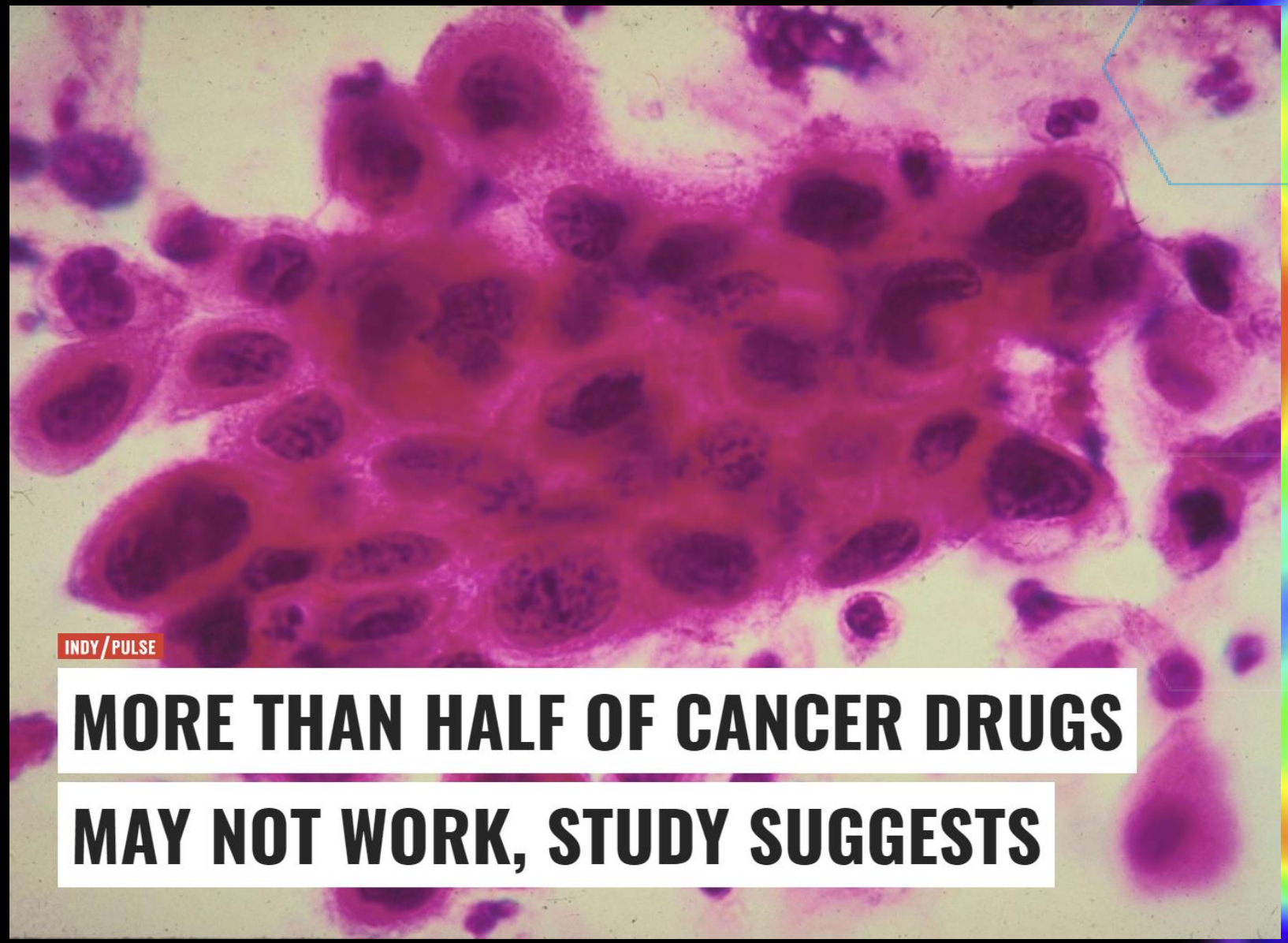
The typical candidate for Hyperbaric Oxygen Therapy

"Nurse, get on the internet, go to SURGERY.COM, scroll down and click on the 'Are you totally lost?' icon."



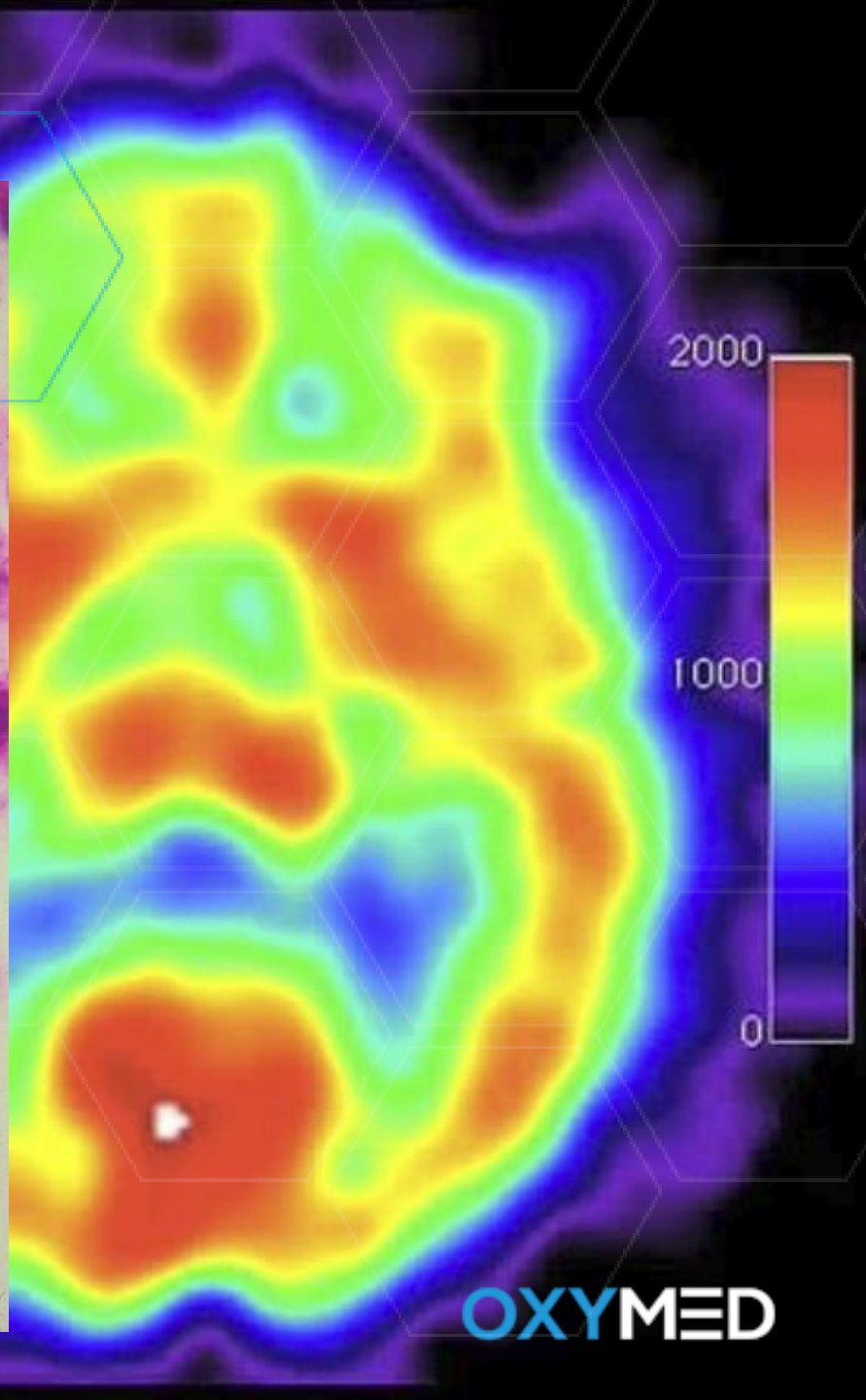
OXYMED

Cancers & Cytokines



INDY/PULSE

MORE THAN HALF OF CANCER DRUGS MAY NOT WORK, STUDY SUGGESTS



OXYMED

BMJ 2009-2013: 57% no evidence

More than half of cancer drugs may be ineffective

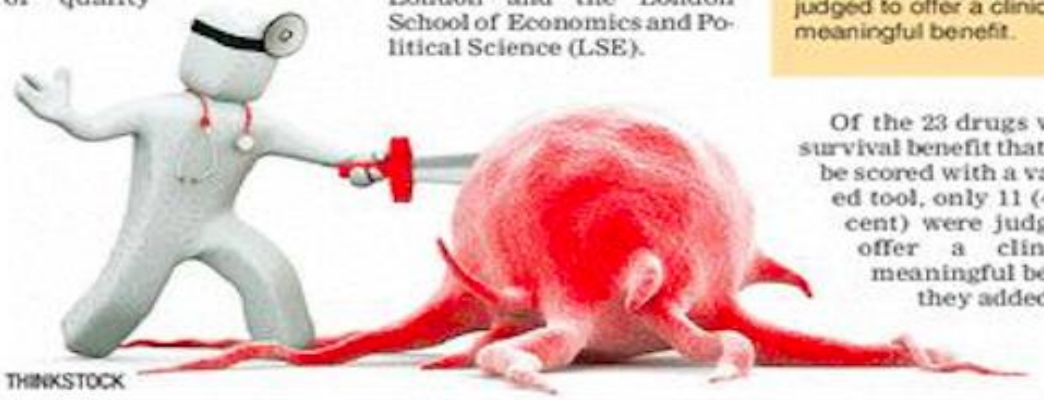
Researchers found certain drugs on the market don't have proven benefits

London: According to a recent study, half of the cancer drugs that have recently arrived on the market have come with little evidence that they boost the survival or wellbeing of patients.

The researchers found that of cancer drugs approved by the European Medicines Agency (EMA) between 2009 and 2013, 57 per cent (39 out of 68) had no supporting evidence of better survival or quality of life when they entered the market.

After an average of five years of follow up, only half of the drugs had shown a survival or quality of life gain in patients over existing treatments or placebo.

For the remaining 33 (49 per cent), uncertainty remains over whether the drugs extend survival or improve quality of life, according to the authors of the study from King's College London and the London School of Economics and Political Science (LSE).



THINKSTOCK

DRUGS OR SCAMS?

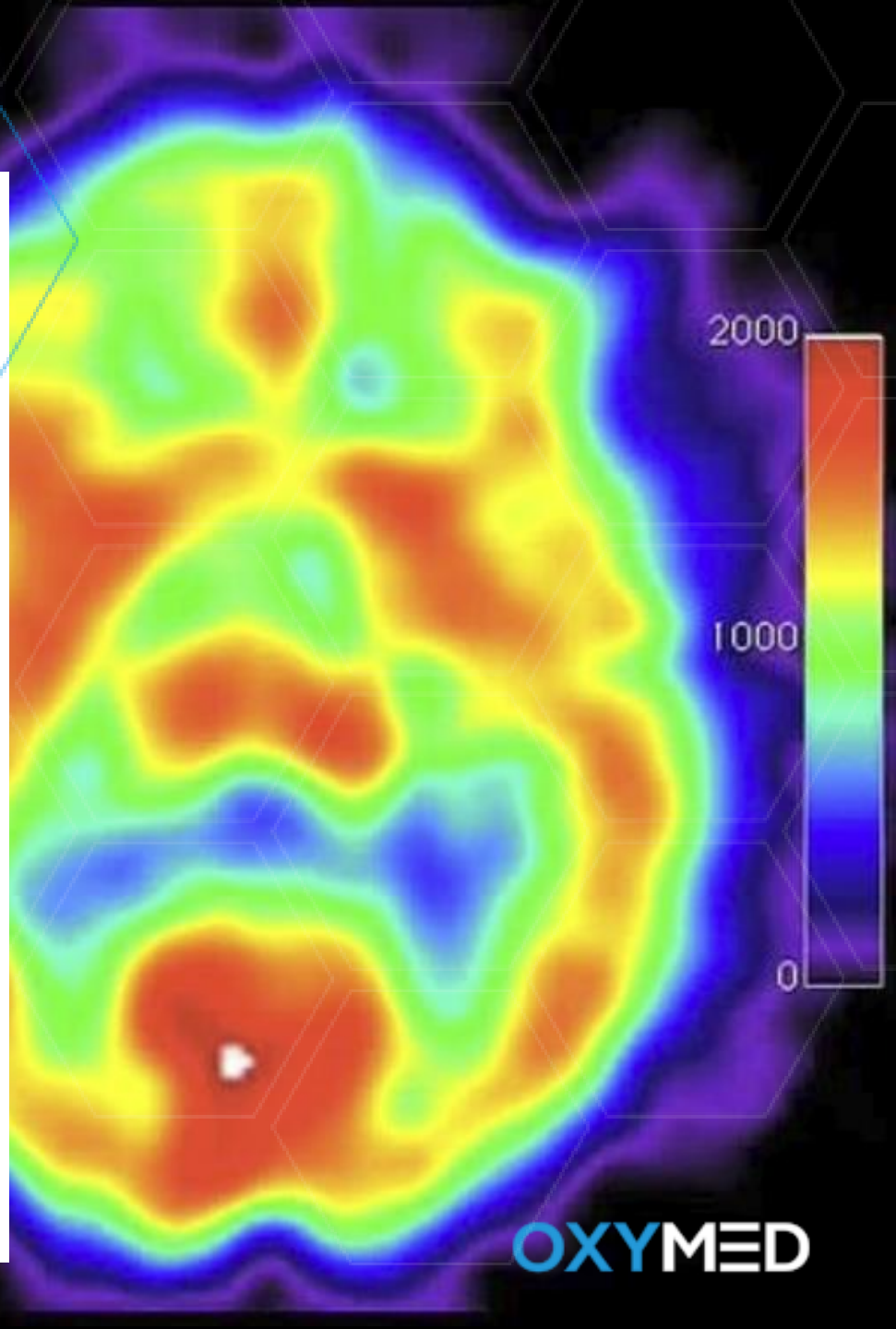
- Of drugs approved by the EMA between 2009 and 2013, 57 per cent had no supporting evidence of better survival.
- **With 49 per cent of the drugs, uncertainty remains over their benefits.**
- Of the 23 drugs with a survival benefit, only 48 per cent were judged to offer a clinically meaningful benefit.

Of the 23 drugs with a survival benefit that could be scored with a validated tool, only 11 (48 per cent) were judged to offer a clinically meaningful benefit, they added.

Author Huseyin Naci, assistant professor in LSE's Department of Health Policy, said, "It is remarkable that so few cancer drugs enter the European market without any clear data on outcomes that matter to patients and their doctors: longer survival and better quality of life. There is a clear need to raise the bar for approving new cancer drugs."

Dr Courtney Davis, a medical and political sociologist in the Department of Global Health and Social Medicine at King's, added, "We evaluated the evidence base for all new drugs entering the market over a five year period and found that the majority came onto the market without clear evidence that they improved patients' survival or quality of life."

The study was published in *The British Medical Journal*. —**Agencies**

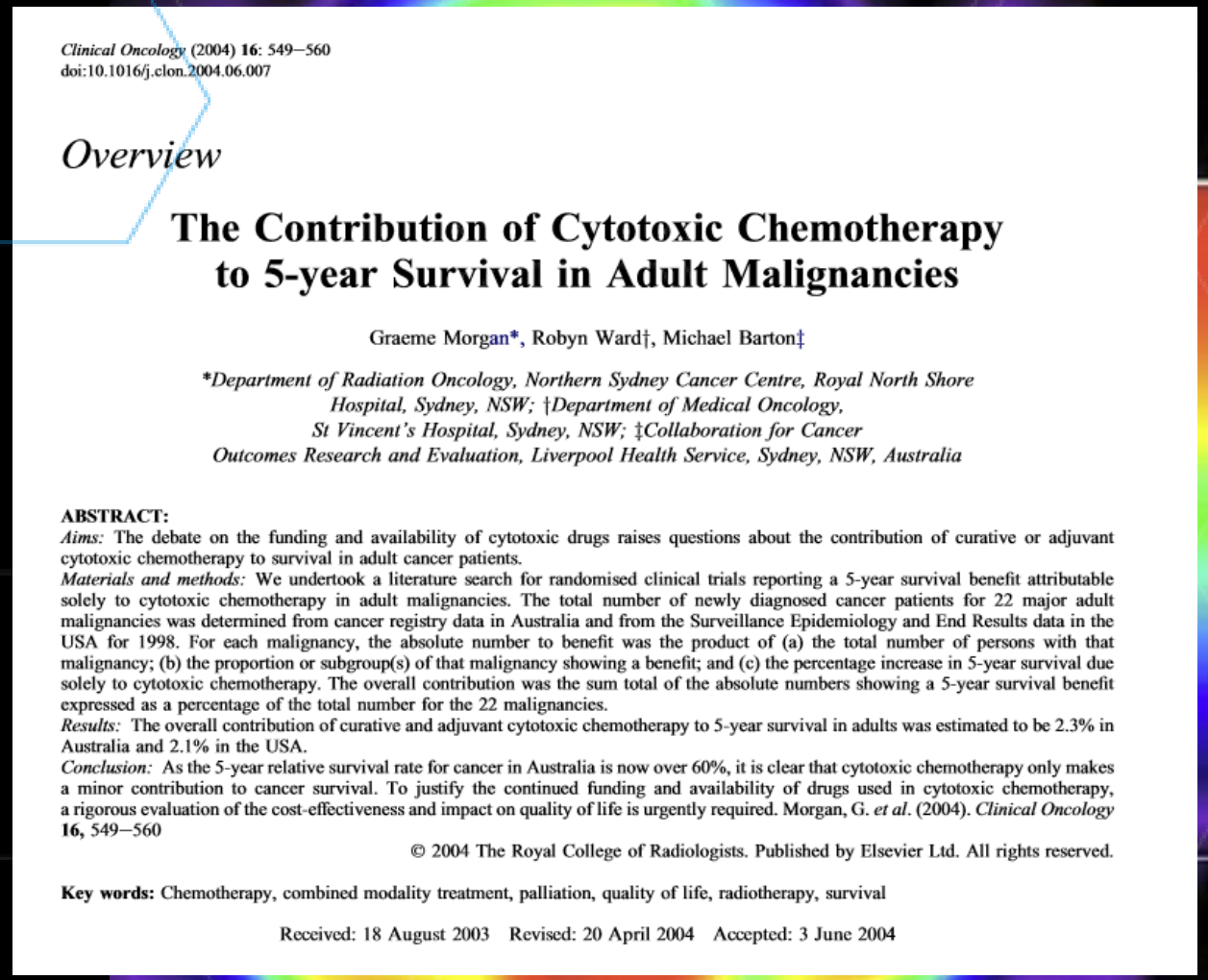


OXYMED

Australia success to a 5-year survival

The Contribution of Cytotoxic Chemotherapy to 5-year Survival in Adult Malignancies

- The overall contribution of curative and adjuvant cytotoxic chemotherapy to 5-year survival in adults was estimated to be 2.3% in Australia and 2.1% in the USA.
- "As the 5-year relative survival rate for cancer in Australia is now over 60%, it is clear that cytotoxic chemotherapy only makes a minor contribution to cancer survival.
- To justify the continued funding and availability of drugs used in cytotoxic chemotherapy, a rigorous evaluation of the cost-effectiveness and impact on quality of life is urgently required.“



Clinical Oncology (2004) **16**: 549–560
doi:10.1016/j.clon.2004.06.007

Overview

The Contribution of Cytotoxic Chemotherapy to 5-year Survival in Adult Malignancies

Graeme Morgan*, Robyn Ward†, Michael Barton‡

**Department of Radiation Oncology, Northern Sydney Cancer Centre, Royal North Shore Hospital, Sydney, NSW; †Department of Medical Oncology, St Vincent's Hospital, Sydney, NSW; ‡Collaboration for Cancer Outcomes Research and Evaluation, Liverpool Health Service, Sydney, NSW, Australia*

ABSTRACT:
Aims: The debate on the funding and availability of cytotoxic drugs raises questions about the contribution of curative or adjuvant cytotoxic chemotherapy to survival in adult cancer patients.
Materials and methods: We undertook a literature search for randomised clinical trials reporting a 5-year survival benefit attributable solely to cytotoxic chemotherapy in adult malignancies. The total number of newly diagnosed cancer patients for 22 major adult malignancies was determined from cancer registry data in Australia and from the Surveillance Epidemiology and End Results data in the USA for 1998. For each malignancy, the absolute number to benefit was the product of (a) the total number of persons with that malignancy; (b) the proportion or subgroup(s) of that malignancy showing a benefit; and (c) the percentage increase in 5-year survival due solely to cytotoxic chemotherapy. The overall contribution was the sum total of the absolute numbers showing a 5-year survival benefit expressed as a percentage of the total number for the 22 malignancies.
Results: The overall contribution of curative and adjuvant cytotoxic chemotherapy to 5-year survival in adults was estimated to be 2.3% in Australia and 2.1% in the USA.
Conclusion: As the 5-year relative survival rate for cancer in Australia is now over 60%, it is clear that cytotoxic chemotherapy only makes a minor contribution to cancer survival. To justify the continued funding and availability of drugs used in cytotoxic chemotherapy, a rigorous evaluation of the cost-effectiveness and impact on quality of life is urgently required. Morgan, G. *et al*. (2004). *Clinical Oncology* **16,** 549–560

Key words: Chemotherapy, combined modality treatment, palliation, quality of life, radiotherapy, survival

Received: 18 August 2003 Revised: 20 April 2004 Accepted: 3 June 2004

OXYMED

Cancer is a metabolic disease driven by hypoxia

Carcinogenesis vol.35 no.3 pp.515–527, 2014
doi:10.1093/carcin/bgt480
Advance Access publication December 16, 2013

REVIEW

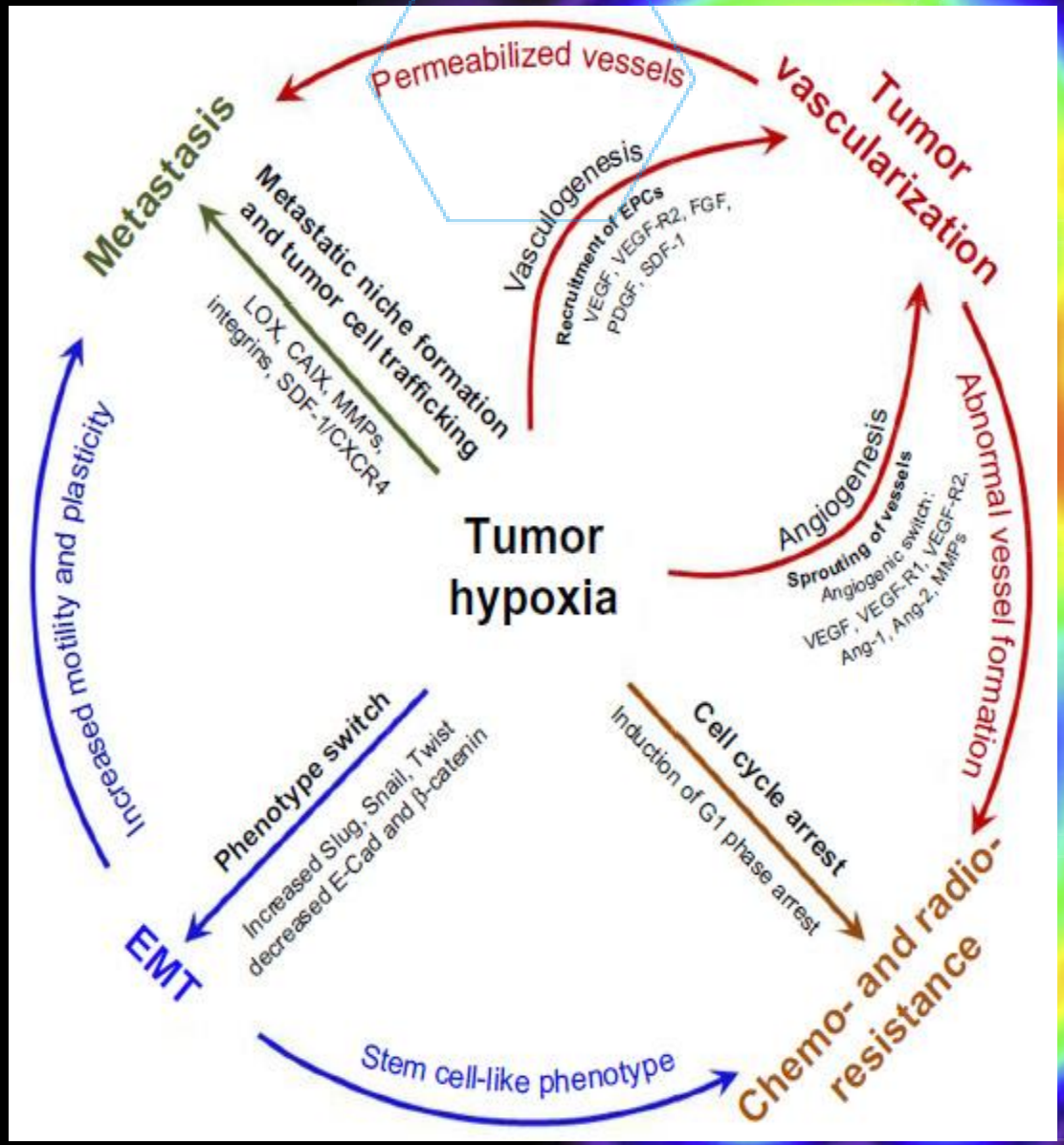
Cancer as a metabolic disease: implications

Thomas N.Seyfried*, Roberto E.Flores, Angela M.Poff[1] and Dominic P.D'Agostino[1]

Biology Department, Boston College, Chestnut Hill, MA 02467, USA and [1]Department of Molecular Pharmacology and Physiology, University of South Florida, Tampa, FL 33612, USA

*To whom correspondence should be addressed. Tel: +1 617 552 3563; Fax: +1 617 552 2011;
Email: thomas.seyfried@bc.edu

Emerging evidence indicates that cancer is primarily a metabolic disease involving disturbances in energy production through respiration and fermentation. The genomic instability observed in tumor cells and all other recognized hallmarks of cancer are considered downstream epiphenomena of the initial disturbance of cellular energy metabolism. The disturbances in tumor cell energy metabolism can be linked to abnormalities in the structure and function of the mitochondria. When viewed as a mitochondrial metabolic disease, the evolutionary theory of Lamarck can better explain cancer progression than can the evolutionary theory of Darwin. Cancer growth and progression can be managed following a whole body transition from fermentable metabolites, primarily glucose and glutamine, to respiratory metabolites, primarily ketone bodies. As each individual is a unique metabolic entity, personalization of metabolic therapy as a broad-based cancer treatment strategy will require fine-tuning to match the therapy to an individual's unique physiology.



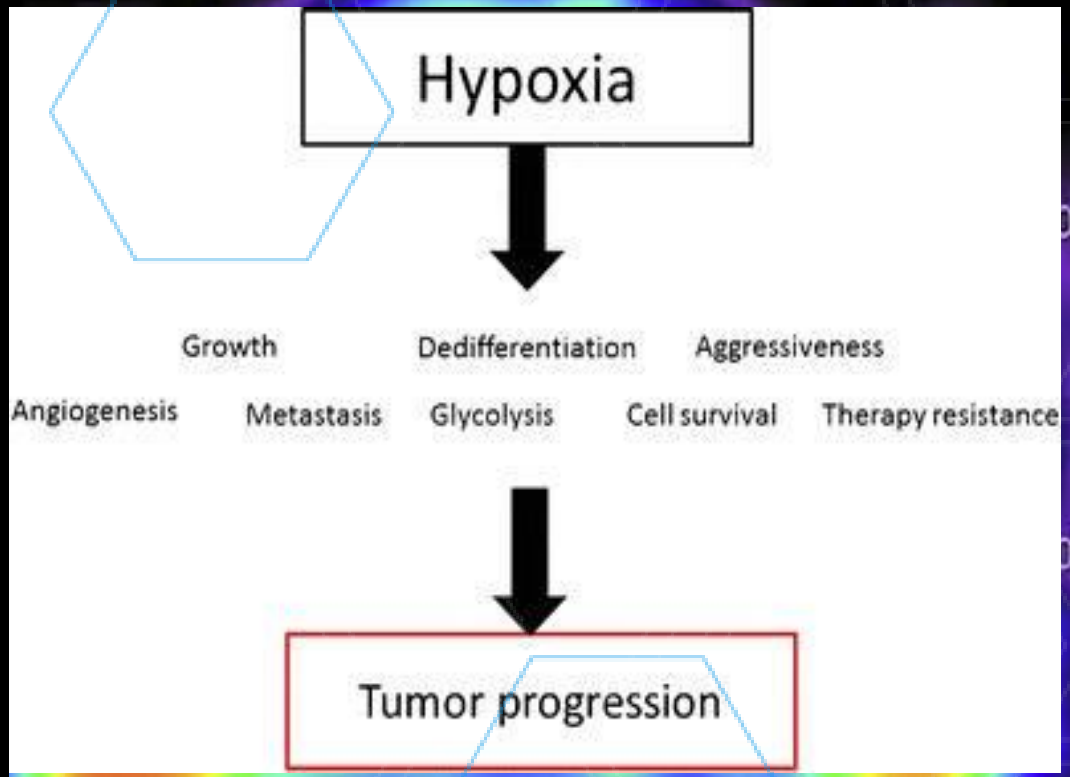
OXYMED

Hypoxia – Feldmeier 2003

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

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

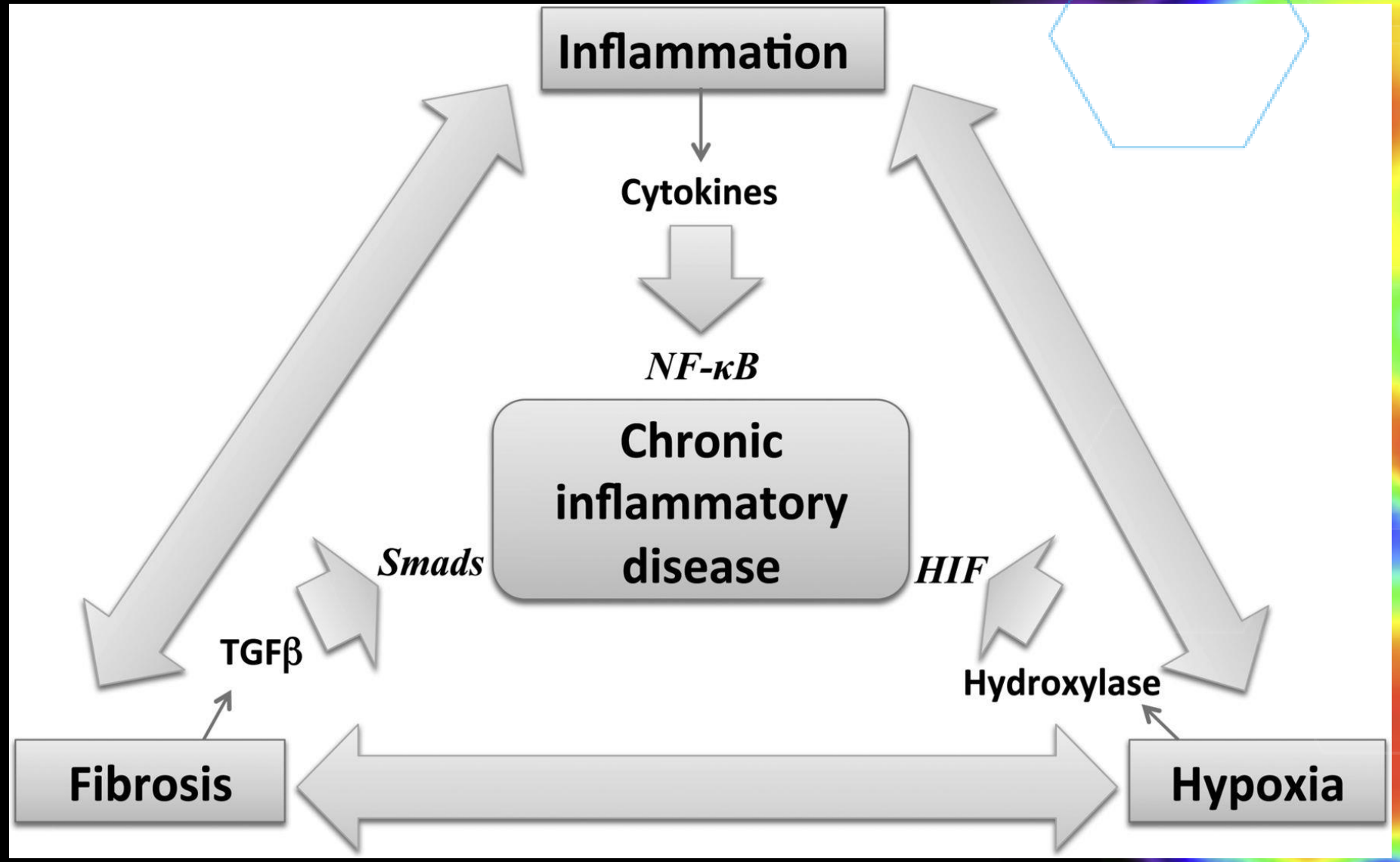
[1]Radiation Oncology Department, Medical College of Ohio, Toledo, OH, USA; [2]Department of Radiation Oncology and Nuclear Medicine, Diakonieskrankenhaus Rotenberg, Germany; [3]Department of Radiation Oncology, Heinrich Heine University, Duesseldorf, Germany; [4]Department of Radiation Oncology, VU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

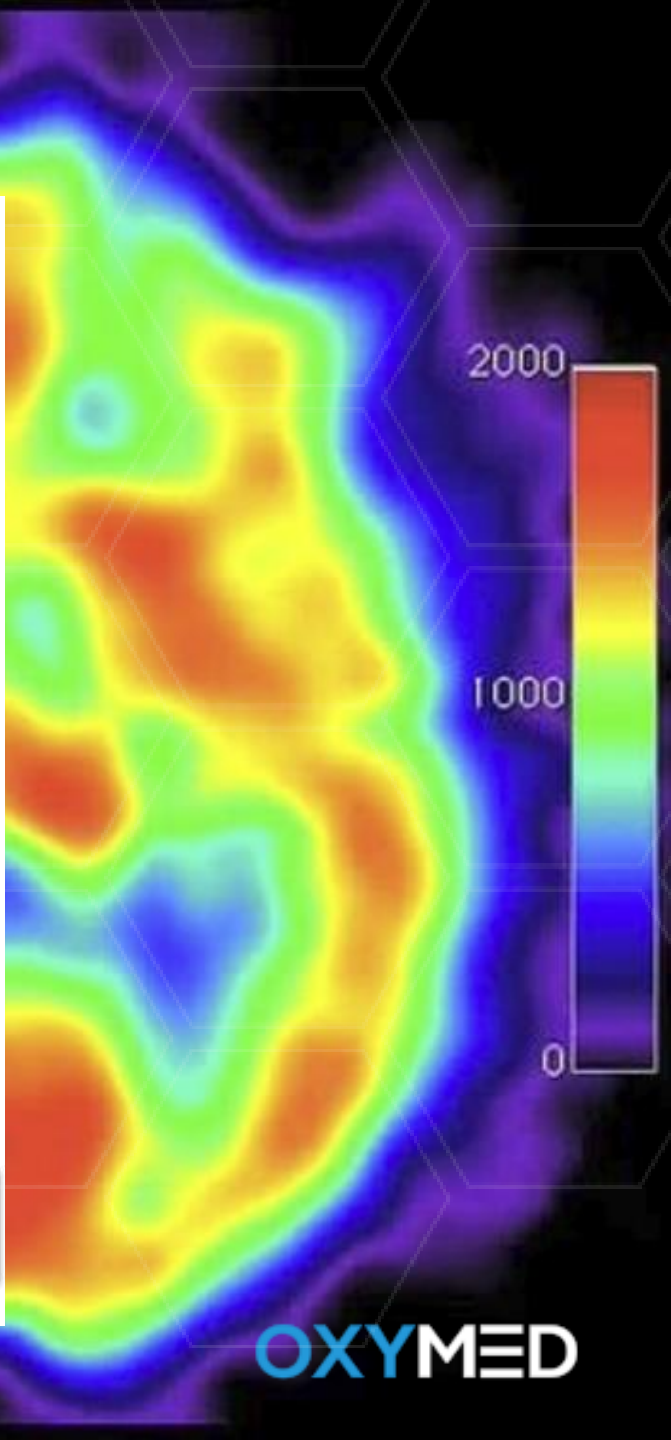


iii. Interleukin-8 release is increased by hypoxia[68] This has been demonstrated in human glioblastoma cells in culture. IL-8 has been shown to have angiogenic properties in this model. The work of Shi and associates[69] confirms an increase in IL-8 by hypoxia and acidosis and suggest this contributes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

OXYMED

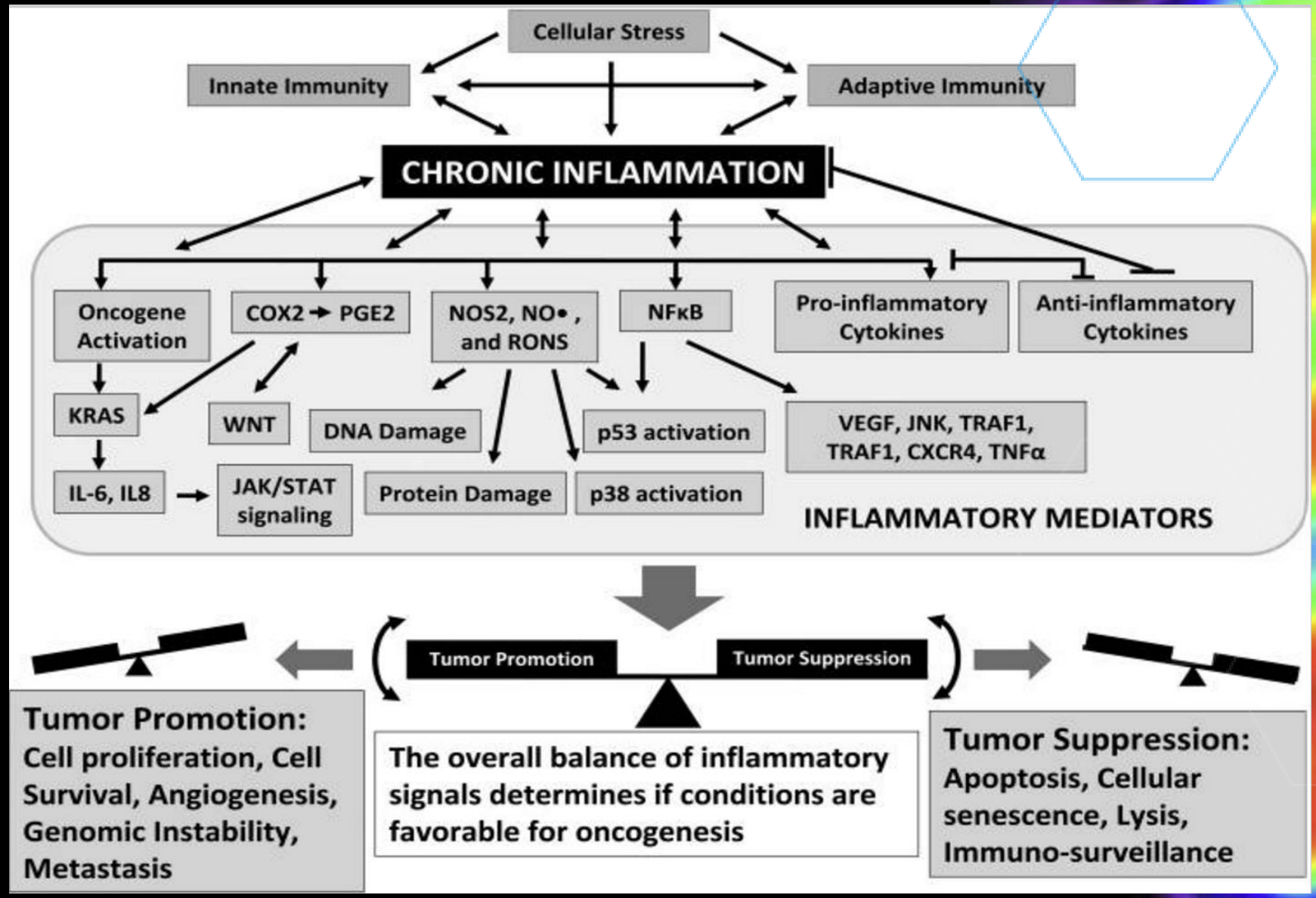
Chronic inflammatory disease

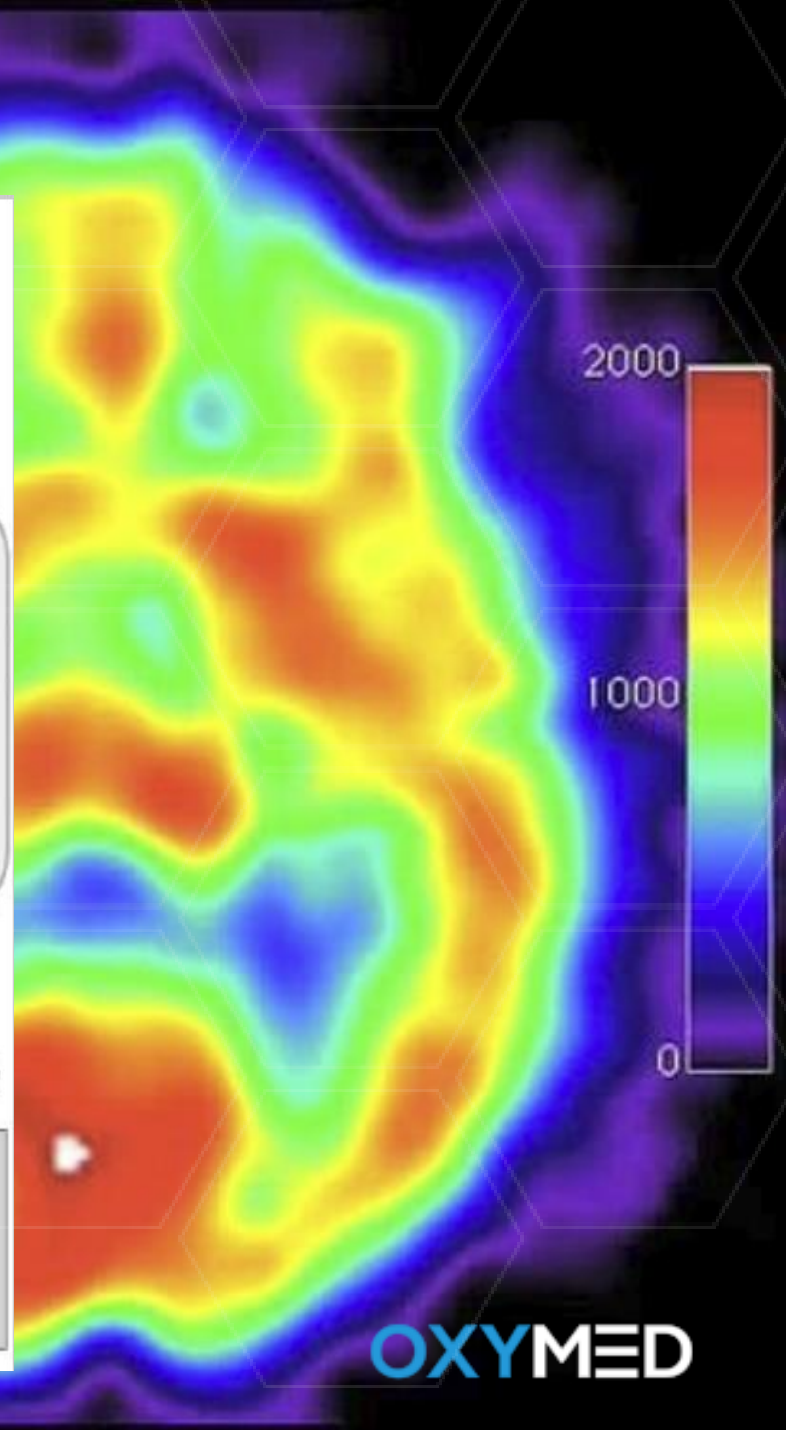




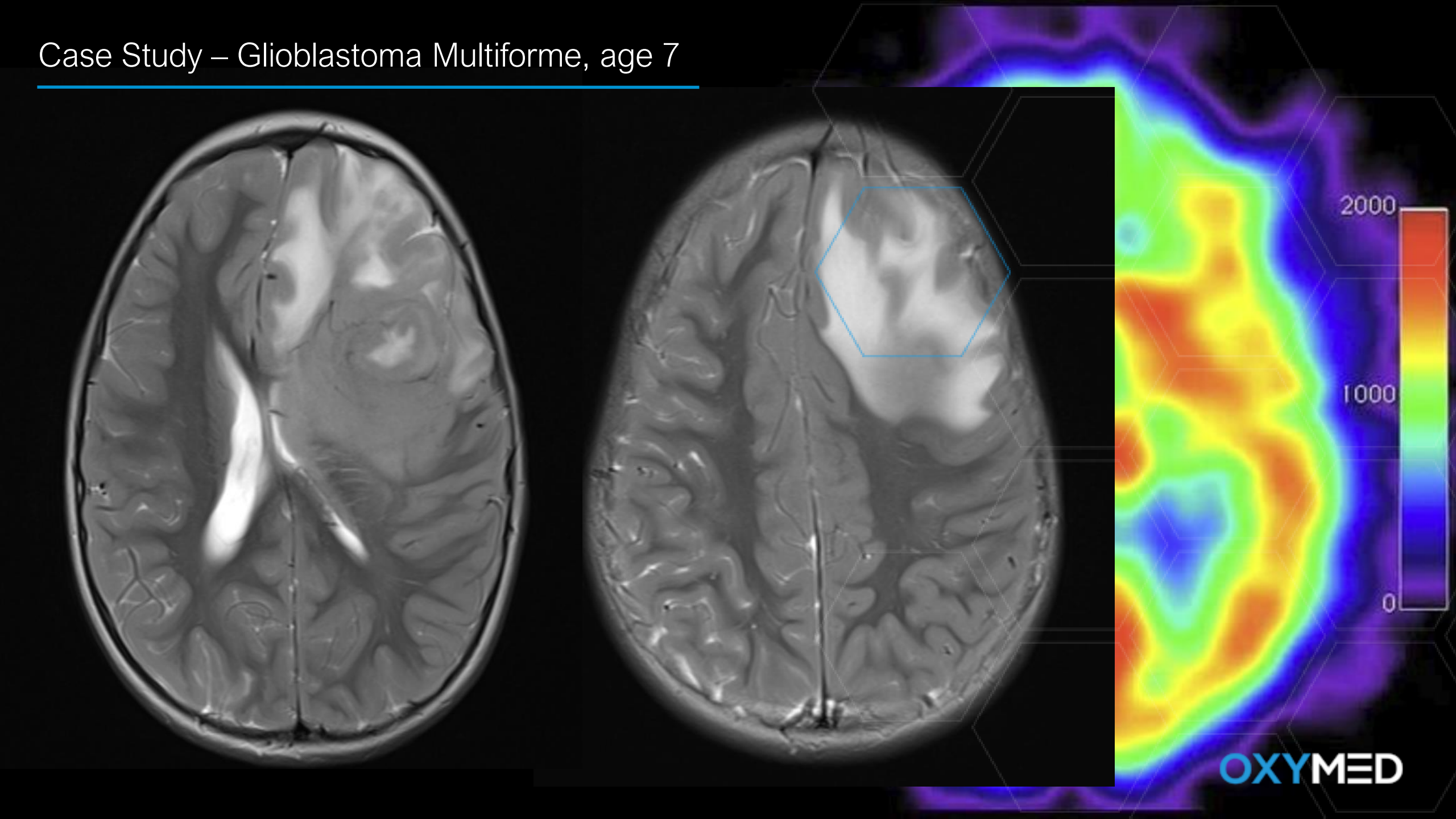
OXYMED

Cancer - Cytokine balance



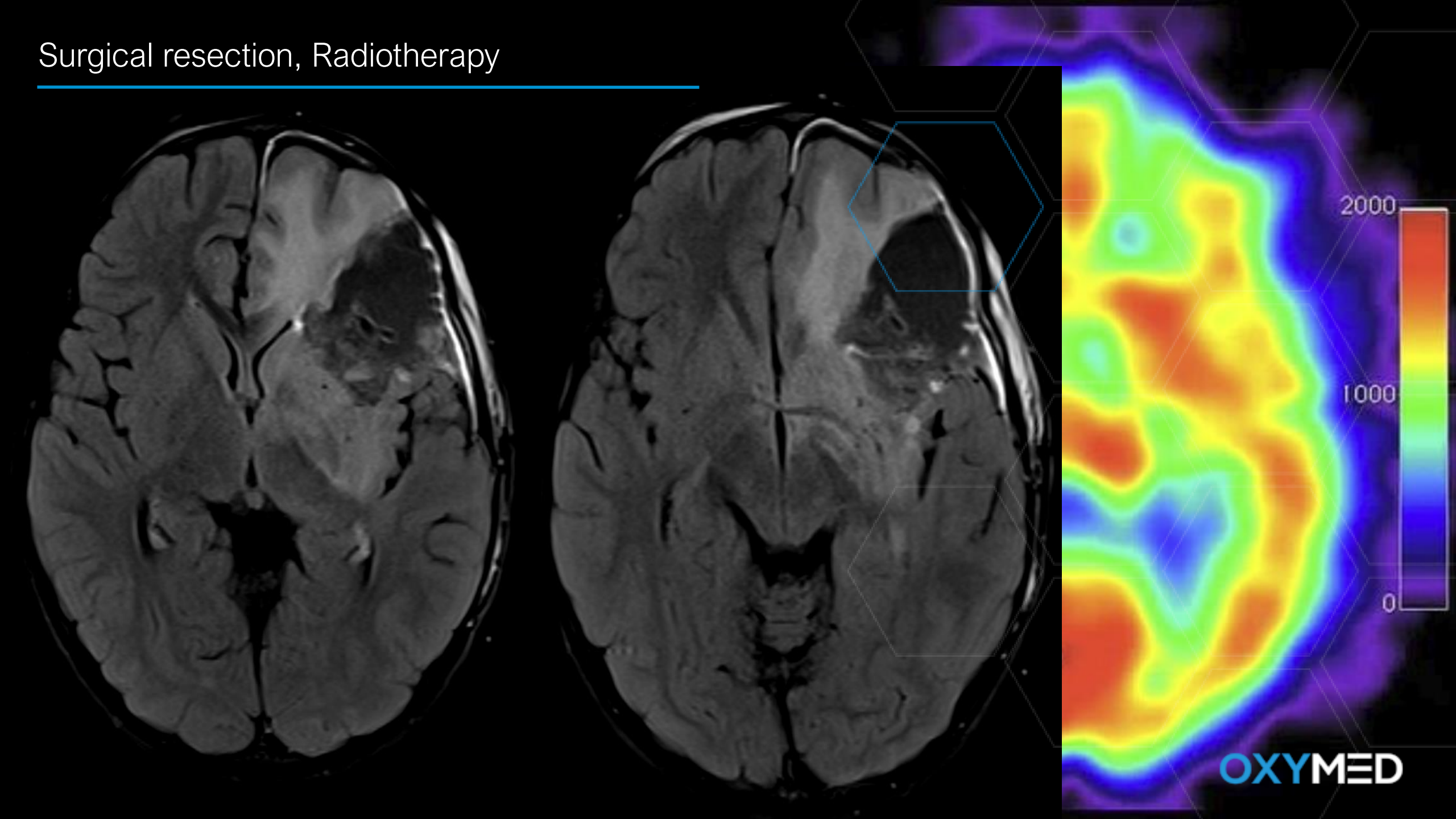


Case Study – Glioblastoma Multiforme, age 7



OXYMED

Surgical resection, Radiotherapy



Cytokine Profile – before HBOT

OXYMED HEALTH

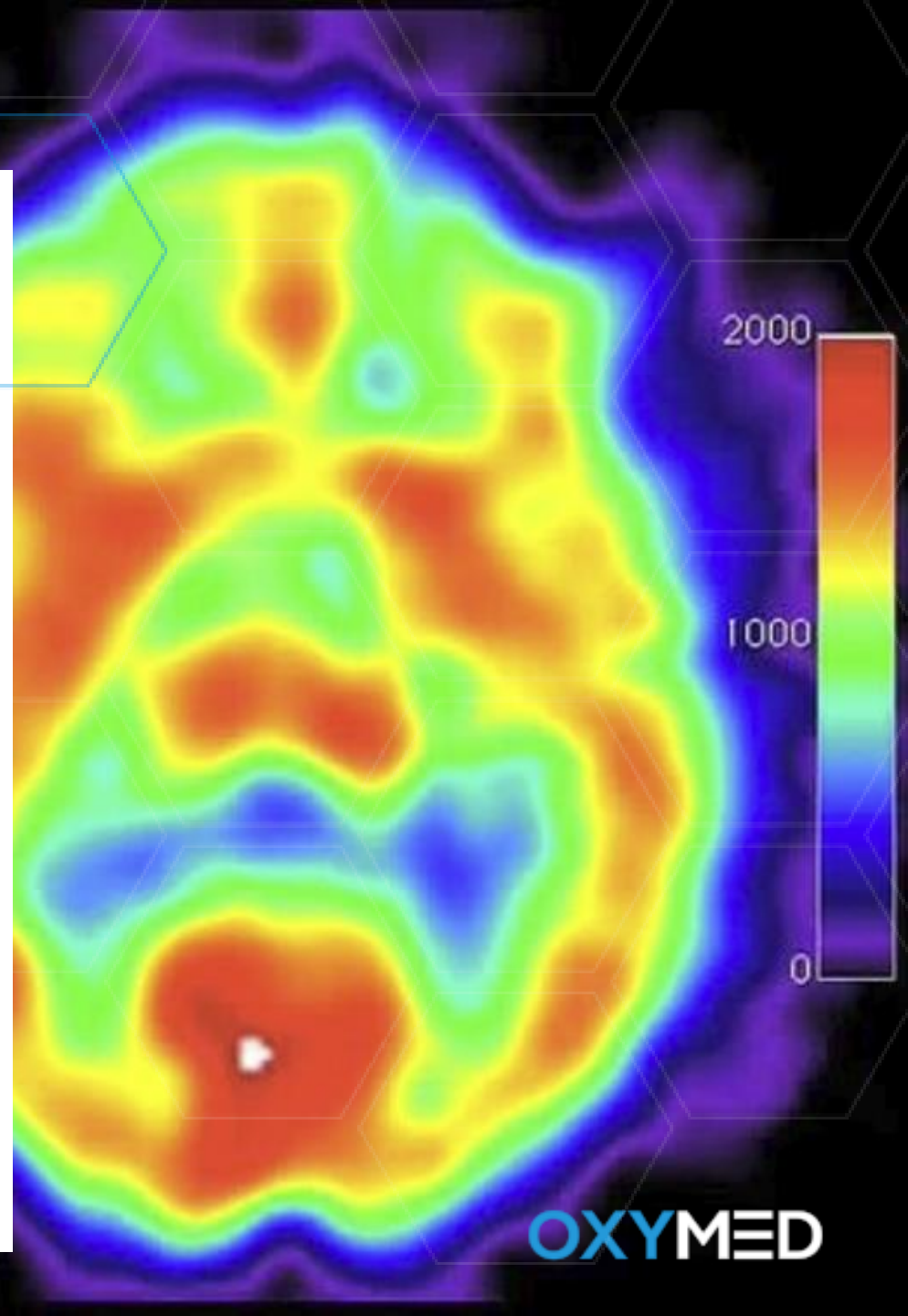
Date of Birth : 21-Oct-2009
Sex : M
Collected : 01-Mar-2017

Lab id: UR#:

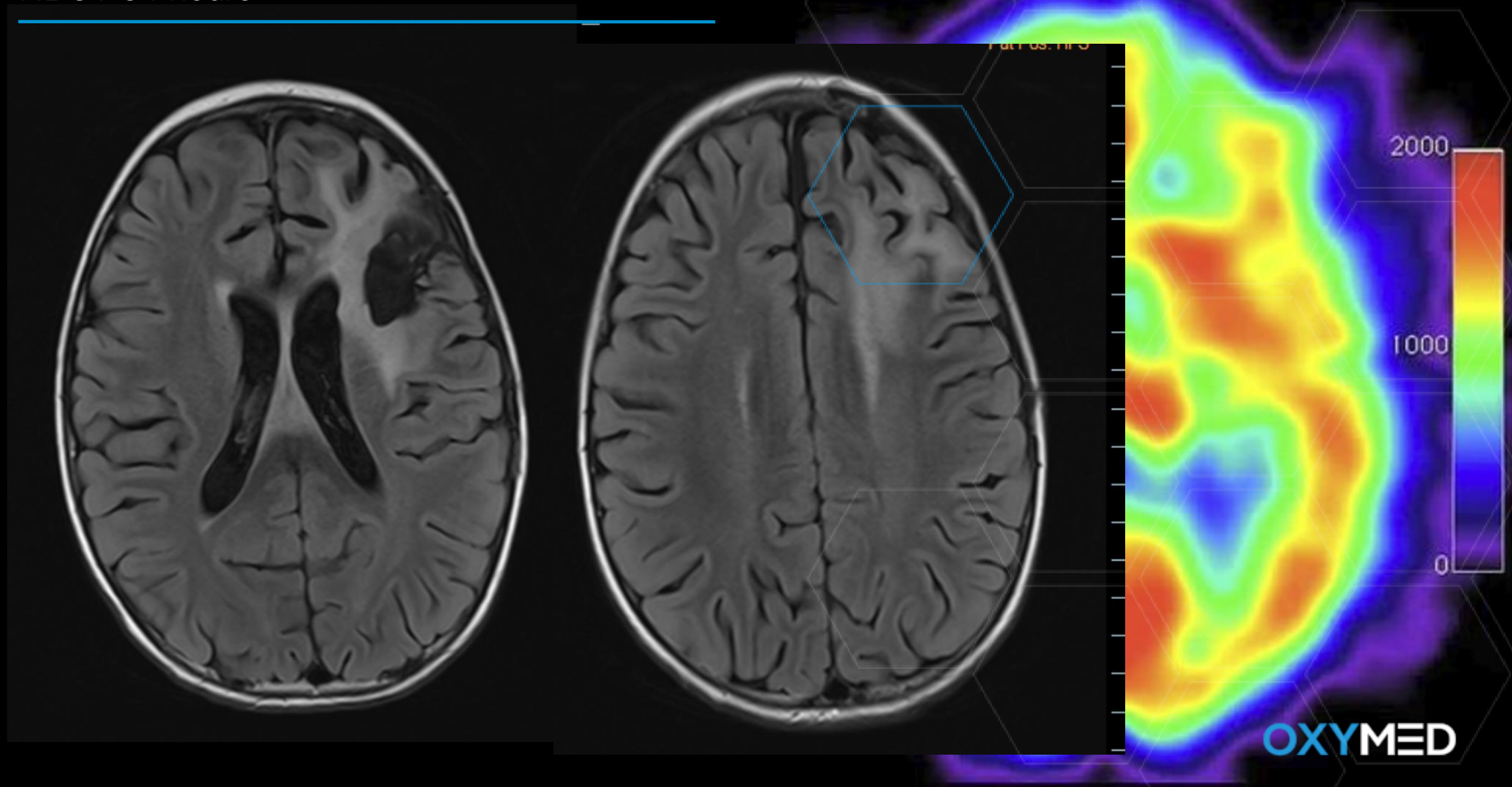
OXYMED
643 CHAPEL STREET
SOUTH YARRA VIC 3141

INTEGRATIVE MEDICINE

BLOOD - SERUM	Result	Range	Units
CYTOKINES,Extensive Panel			
ProInflammatory Cytokines (TH1)			
Interleukin 1	*3463.0 *H*	0.0 - 2.8	pg/mL
Interleukin 6	*1252.0 *H*	0.0 - 11.0	pg/mL
Interleukin 7	*67.8 *H*	0.0 - 16.0	pg/mL
Interleukin 8	*>2500.0 *H*	0.0 - 28.0	pg/mL
Interleukin 17	10.4	< 13.0	pg/mL
TNFa	*816.00 *H*	0.00 - 13.00	pg/mL
TNFb	*164.0 *H*	0.0 - 156.0	pg/mL
S100B	*151.8 *H*	60.0 - 100.0	pg/mL
AntiInflammatory Cytokines (TH2)			
GM-CSF	*1620.0 *H*	0.0 - 80.0	pg/mL
Interleukin 2	7.4	0.0 - 10.0	pg/mL
Interleukin 3	<3.2	< 5.0	pg/mL
Interleukin 4	*127.2 *H*	0.0 - 19.0	pg/mL
Interleukin 5	3.8	0.0 - 13.0	pg/mL
Interleukin 10	*99.7 *H*	0.0 - 7.0	pg/mL
Interleukin 12	7.9	0.0 - 14.0	pg/mL
Interleukin 13	*21.7 *H*	0.0 - 6.0	pg/mL
INFg	25.8	0.0 - 28.0	pg/mL
TGFb	57.7	28.0 - 64.0	pg/mL
Brain Derived Neurotrophic Factor BDNF	33.2	20.0 - 50.0	ng/mL



HBOT 84 hours



Cytokine Profile – 106 hours HBOT

OXYMED HEALTH

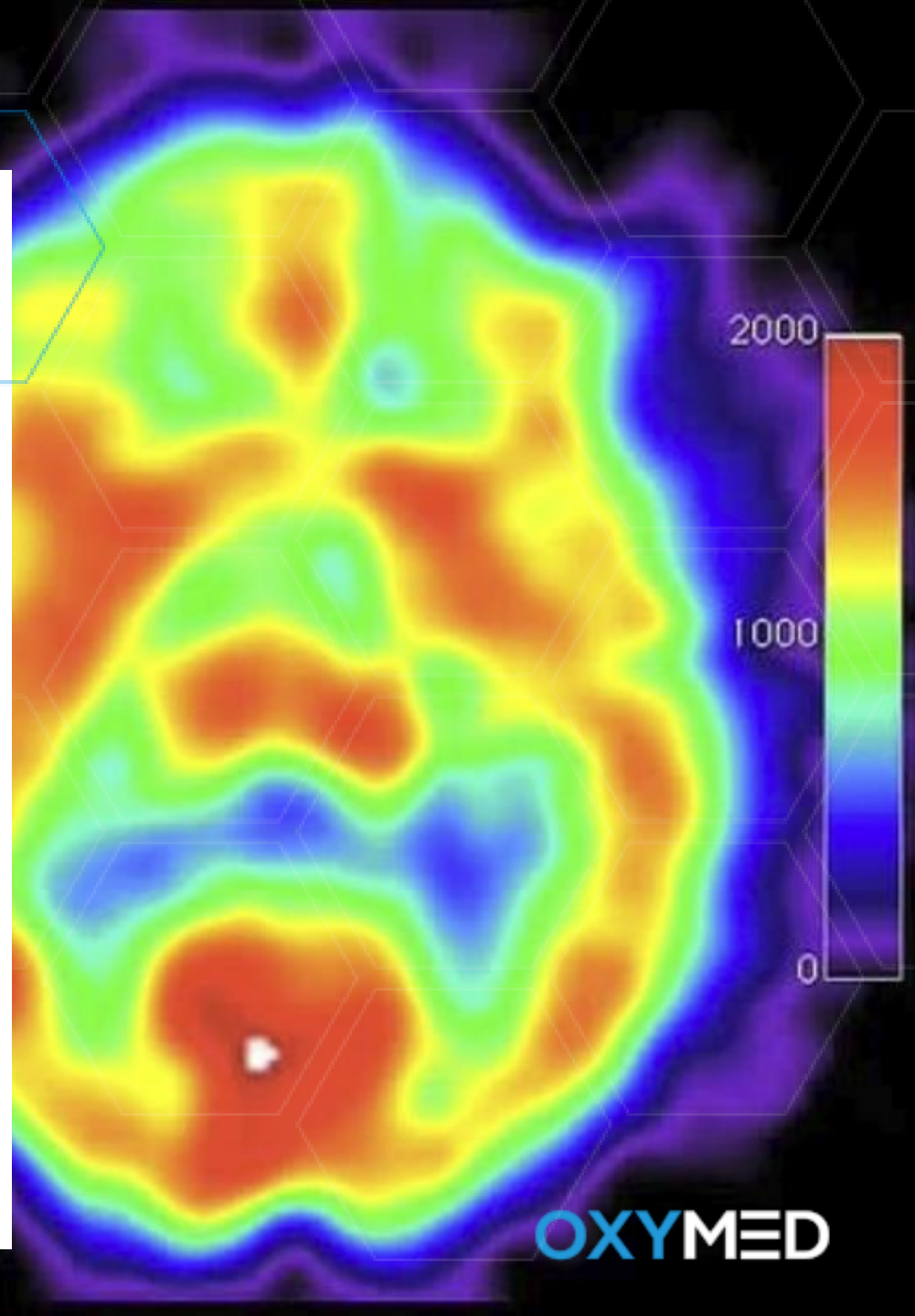
Date of Birth : 21-Oct-2009
Sex : M
Collected : 02-May-2017

Lab id: UR#:

OXYMED, 643 CHAPEL STREET
SOUTH YARRA VIC 3141

INTEGRATIVE MEDICINE

BLOOD - SERUM	Result	Range	Units
Hyperbaric Oxygen Therapy (HBO)	106.0		Hours
CYTOKINES,Extensive Panel			
ProInflammatory Cytokines (TH1)			
Interleukin 1	1.9	0.0 - 2.8	pg/mL
Interleukin 6	4.9	0.0 - 11.0	pg/mL
Interleukin 7	*24.8* *H	0.0 - 16.0	pg/mL
Interleukin 8	*48.4* *H	0.0 - 28.0	pg/mL
Interleukin 17	7.8	< 13.0	pg/mL
TNFa	10.80	0.00 - 13.00	pg/mL
TNFb	144.0	0.0 - 156.0	pg/mL
S100B	*13.6* *L	60.0 - 100.0	pg/mL
AntiInflammatory Cytokines (TH2)			
GM-CSF	*1510.3* *H	0.0 - 80.0	pg/mL
Interleukin 2	3.6	0.0 - 10.0	pg/mL
Interleukin 3	<3.0	< 5.0	pg/mL
Interleukin 4	*44.4* *H	0.0 - 19.0	pg/mL
Interleukin 5	1.8	0.0 - 13.0	pg/mL
Interleukin 10	*14.8* *H	0.0 - 7.0	pg/mL
Interleukin 12	2.4	0.0 - 14.0	pg/mL
Interleukin 13	*7.1* *H	0.0 - 6.0	pg/mL
INFg	17.7	0.0 - 28.0	pg/mL
TGFb	50.2	28.0 - 64.0	pg/mL
Brain Derived Neurotrophic Factor BDNF	*382.0* *H	20.0 - 50.0	ng/mL



Cytokine Profiles – before and after HBOT

OXYMED HEALTH

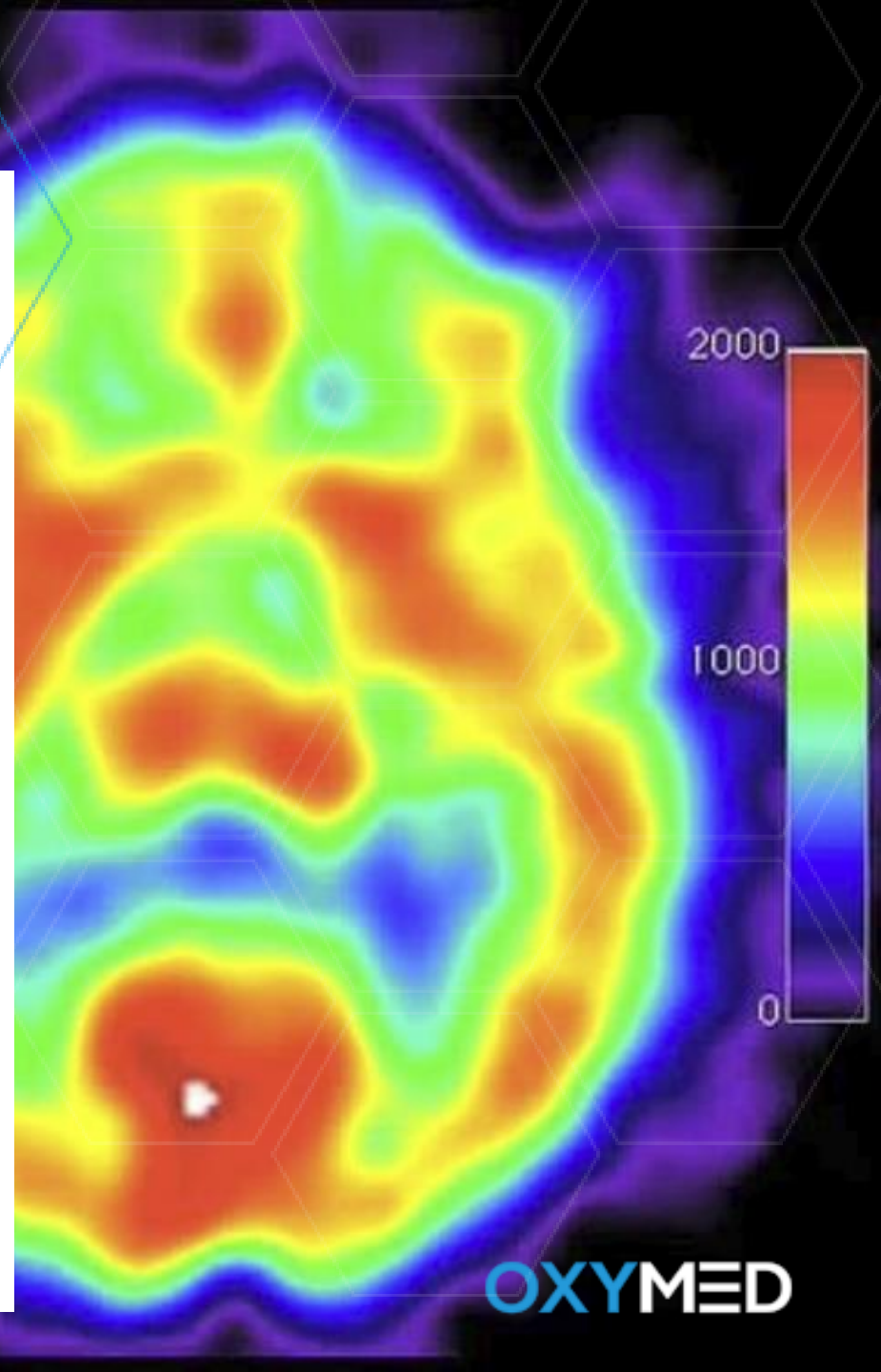
Date of Birth : 21-Oct-2009
Sex : M
Collected : 01-Mar-2017
02-May-2017

Lab id: UR#:

OXYMED
643 CHAPEL STREET
SOUTH YARRA VIC 3141

INTEGRATIVE MEDICINE

BLOOD - SERUM	Result	Hours		
CYTOKINES,Extensive Panel	0	106.0		
ProInflammatory Cytokines (TH1)				
Interleukin 1	*3463.0* *H	1.9	0.0 - 2.8	pg/mL
Interleukin 6	*1252.0* *H	4.9	0.0 - 11.0	pg/mL
Interleukin 7	*67.8* *H	*24.8* *H	0.0 - 16.0	pg/mL
Interleukin 8	*>2500.0* *H	*48.4* *H	0.0 - 28.0	pg/mL
Interleukin 17	10.4	7.8	< 13.0	pg/mL
TNFa	*816.00* *H	10.80	0.00 - 13.00	pg/mL
TNFb	*164.0* *H	144.0	0.0 - 156.0	pg/mL
S100B	*151.8* *H	*13.6* *L	60.0 - 100.0	pg/mL
AntiInflammatory Cytokines (TH2)				
GM-CSF	*1620.0* *H	*1510.3* *H	0.0 - 80.0	pg/mL
Interleukin 2	7.4	3.6	0.0 - 10.0	pg/mL
Interleukin 3	<3.2	<3.0	< 5.0	pg/mL
Interleukin 4	*127.2* *H	*44.4* *H	0.0 - 19.0	pg/mL
Interleukin 5	3.8	1.8	0.0 - 13.0	pg/mL
Interleukin 10	*99.7* *H	*14.8* *H	0.0 - 7.0	pg/mL
Interleukin 12	7.9	2.4	0.0 - 14.0	pg/mL
Interleukin 13	*21.7* *H	*7.1* *H	0.0 - 6.0	pg/mL
INFg	25.8	17.7	0.0 - 28.0	pg/mL
TGFb	57.7	50.2	28.0 - 64.0	pg/mL
Brain Derived Neurotrophic Factor BDNF	33.2	*382.0* *H	20.0 - 50.0	ng/mL



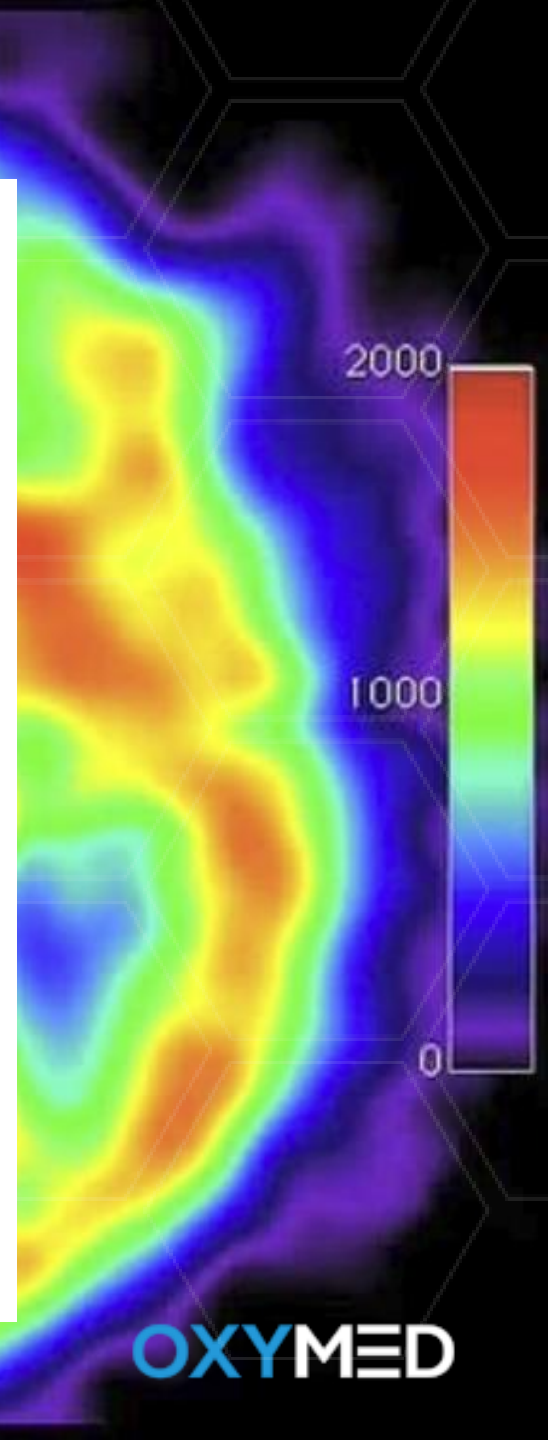
HBOT & Glioblastoma Multiforme

Med Gas Res.

2018 Apr 18;8(1):24-28. doi: 10.4103/2045-9912.229600. eCollection 2018 Jan-Mar.

Hyperbaric Oxygen Therapy as adjunctive strategy in treatment of glioblastoma multiforme.

- Glioblastoma multiforme (GBM) is the most common type of malignant intracranial tumor in adults. Tumor tissue hypoxia, high mitotic rate, and rapid tumor spread account for its poor prognosis. Hyperbaric oxygen therapy (HBOT) may improve the sensitivity of radio-chemotherapy by increasing oxygen tension within the hypoxic regions of the neoplastic tissue. This review summarizes the research of HBOT applications within the context of experimental and clinical GBM.
- Limited clinical trials and preclinical studies suggest that radiotherapy immediately after HBOT enhances the effects of radiotherapy in some aspects.
- HBOT also is able to strengthen the anti-tumor effect of chemotherapy when applied together.
- HBOT is well tolerated in the GBM patients and does not significantly increase toxicity. However, HBOT applied by itself as curative strategy against GBM is controversial in preclinical studies and has not been evaluated rigorously in GBM patients.
- In addition to HBOT favorably managing the therapeutic resistance of GBM, future research needs to focus on the multimodal or cocktail approaches to treatment, as well as molecular strategies targeting GBM stem cells.



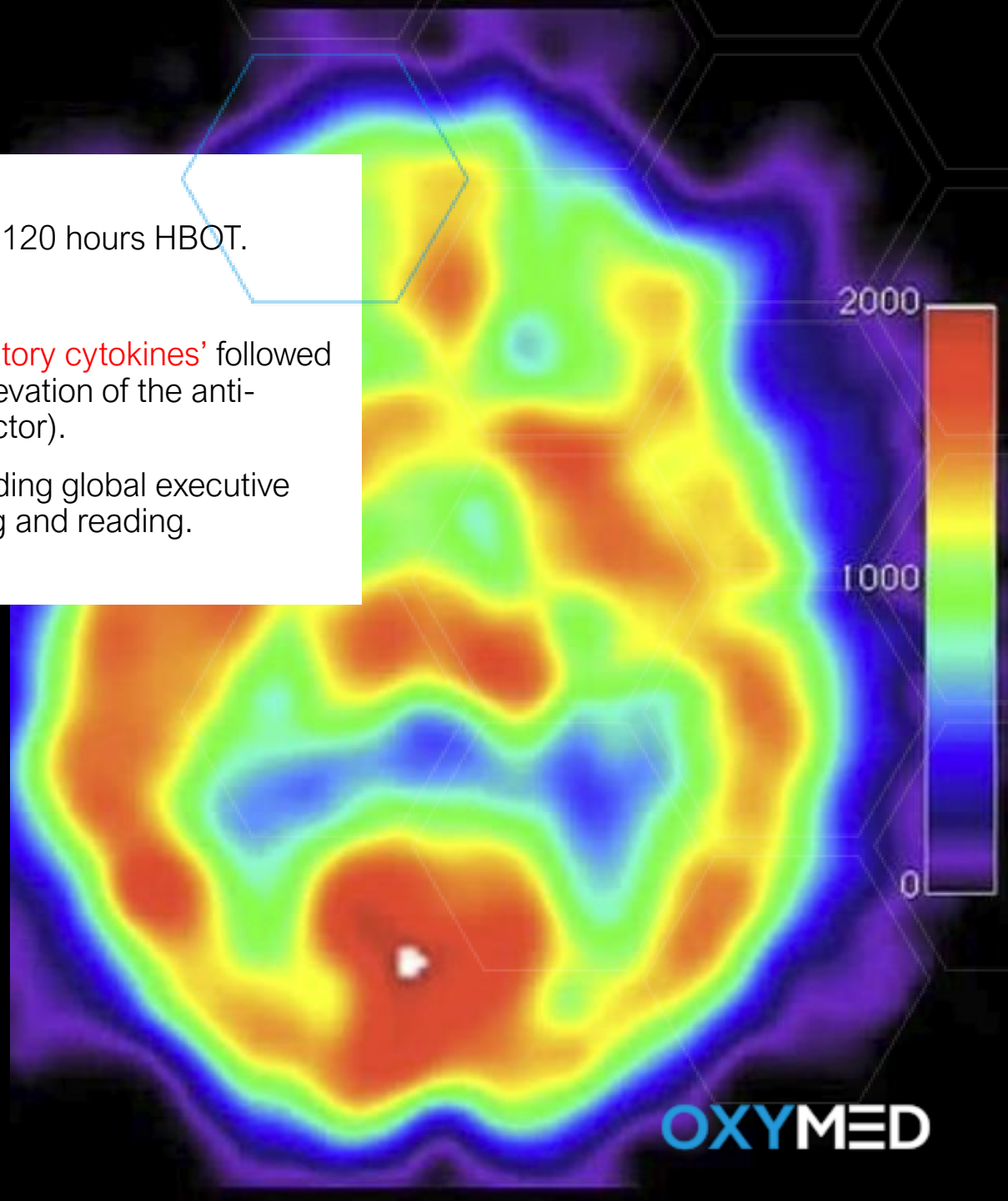
OXYMED

Case Study – Autism

Case Study DC age 7 Autism

Cytokine Testing pre-HBOT, then again at 70-hours HBOT and final test at 120 hours HBOT.

- **High functional autism, non-social, non-verbal.**
- Typically between 50-70 hours of HBOT, there is a 'washout of inflammatory cytokines' followed by reduction of the inflammatory markers corresponding with notable elevation of the anti-inflammatory cytokines including BDNF (Brain Derived Neurotrophic Factor).
- After 120-hours of HBOT, young DC has significant improvements including global executive functioning, talking, instructional responsive, social interactions, drawing and reading.



Autism Case Study DC – before HBOT

OXYMED HEALTH

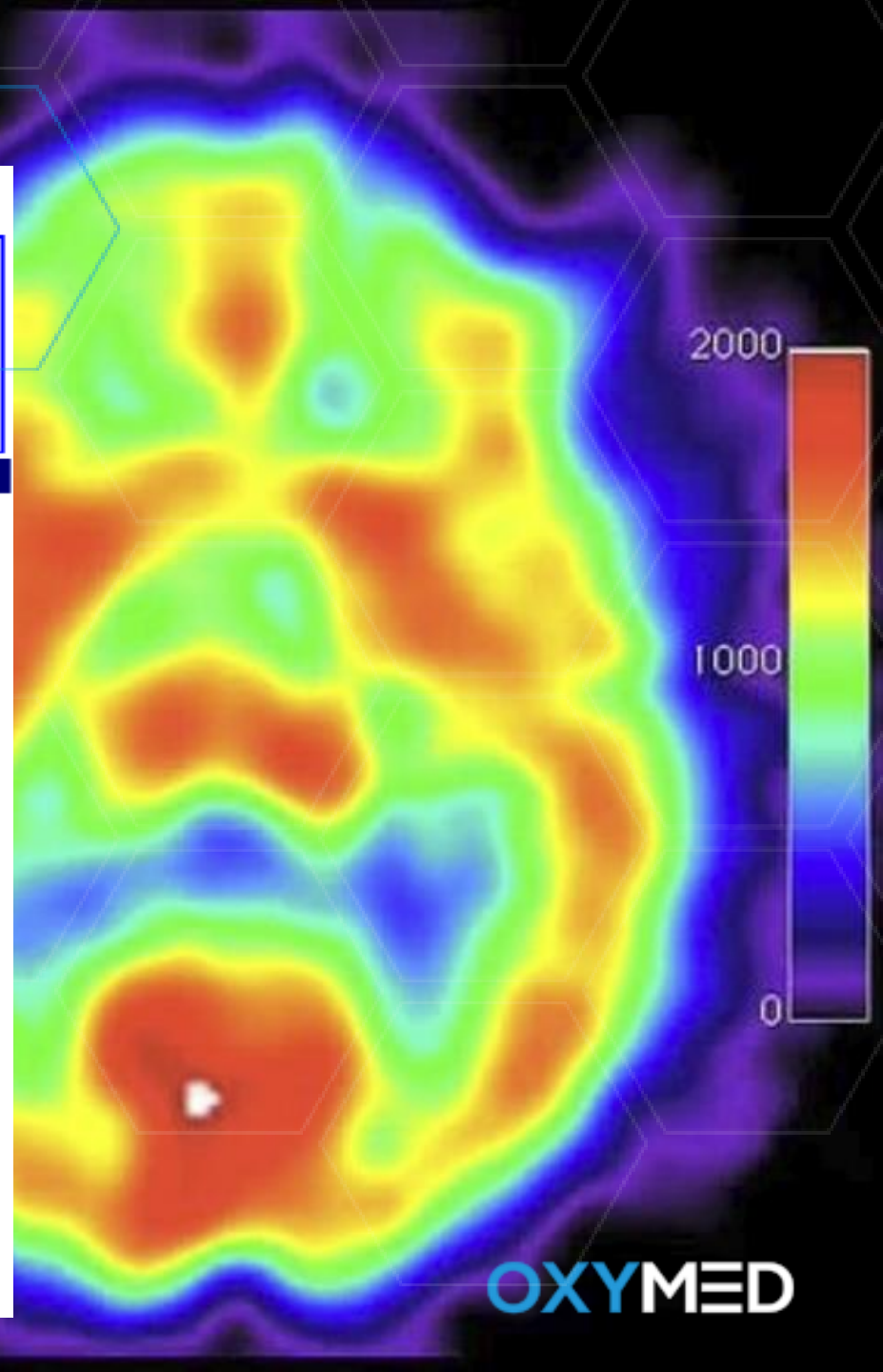
Date of Birth : 20-Nov-2013
Sex : M
Collected : 16-Jan-2018

Lab id: UR#:

OXYMED, 643 CHAPEL STREET
SOUTH YARRA VIC 3141

INTEGRATIVE MEDICINE

BLOOD - SERUM	Result	Range	Units
CYTOKINES,Extensive Panel			
ProInflammatory Cytokines (TH1)			
Interleukin 1	*6.2 *H*	0.0 - 2.8	pg/mL
Interleukin 6	6.7	0.0 - 11.0	pg/mL
Interleukin 7	*22.7 *H*	0.0 - 16.0	pg/mL
Interleukin 8	*106.3 *H*	0.0 - 28.0	pg/mL
Interleukin 17	*26.7 *H*	< 13.0	pg/mL
TNFa	*21.60 *H*	0.00 - 13.00	pg/mL
TNFb	98.0	0.0 - 156.0	pg/mL
S100B	*>5000.0 *H*	60.0 - 100.0	pg/mL
AntiInflammatory Cytokines (TH2)			
GM-CSF	*1217.7 *H*	0.0 - 80.0	pg/mL
Interleukin 2	7.3	0.0 - 10.0	pg/mL
Interleukin 3	*9.6 *H*	< 5.0	pg/mL
Interleukin 4	*30.6 *H*	0.0 - 19.0	pg/mL
Interleukin 5	5.4	0.0 - 13.0	pg/mL
Interleukin 10	*35.3 *H*	0.0 - 7.0	pg/mL
Interleukin 12	*15.6 *H*	0.0 - 14.0	pg/mL
Interleukin 13	*24.0 *H*	0.0 - 6.0	pg/mL
INFg	18.5	0.0 - 28.0	pg/mL
TGFb	36.4	28.0 - 64.0	pg/mL
Brain Derived Neurotrophic Factor BDNF	47.0	20.0 - 50.0	ng/mL



Autism Case Study DC – 70 hours HBOT

OXYMED HEALTH

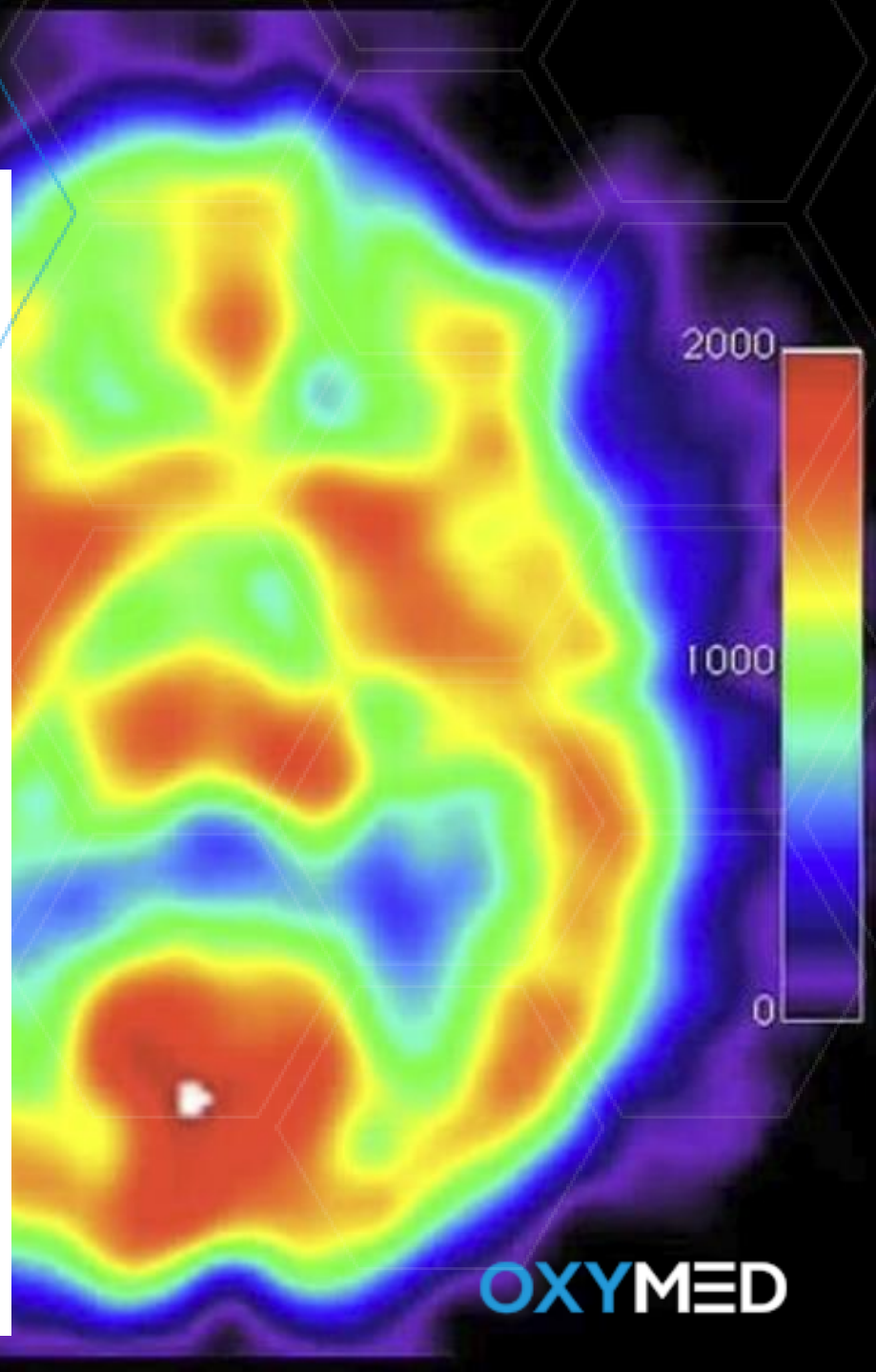
Date of Birth : 20-Nov-2012
Sex : M
Collected : 26-Feb-2018

OXYMED, 643 CHAPEL STREET
SOUTH YARRA VIC 3141

Lab id: UR#:

INTEGRATIVE MEDICINE

BLOOD - SERUM	Result	Range	Units
Hyperbaric Oxygen Therapy (HBO)	70.0		Hours
CYTOKINES,Extensive Panel			
ProInflammatory Cytokines (TH1)			
Interleukin 1	945.6 *H	0.0 - 2.8	pg/mL
Interleukin 6	<0.4	0.0 - 11.0	pg/mL
Interleukin 7	43.9 *H	0.0 - 16.0	pg/mL
Interleukin 8	>2500.0 *H	0.0 - 28.0	pg/mL
Interleukin 17	16.6 *H	< 13.0	pg/mL
TNFa	213.90 *H	0.00 - 13.00	pg/mL
TNFb	123.0	0.0 - 156.0	pg/mL
S100B	639.0 *H	60.0 - 100.0	pg/mL
AntiInflammatory Cytokines (TH2)			
GM-CSF	1710.3 *H	0.0 - 80.0	pg/mL
Interleukin 2	9.8	0.0 - 10.0	pg/mL
Interleukin 3	1.3	< 5.0	pg/mL
Interleukin 4	34.7 *H	0.0 - 19.0	pg/mL
Interleukin 5	4.7	0.0 - 13.0	pg/mL
Interleukin 10	56.9 *H	0.0 - 7.0	pg/mL
Interleukin 12	18.2 *H	0.0 - 14.0	pg/mL
Interleukin 13	30.0 *H	0.0 - 6.0	pg/mL
INFg	23.1	0.0 - 28.0	pg/mL
TGFb	33.0	28.0 - 64.0	pg/mL
Brain Derived Neurotrophic Factor BDNF	39.0	20.0 - 50.0	ng/mL



Autism Case Study DC – 120 hours HBOT

OXYMED HEALTH

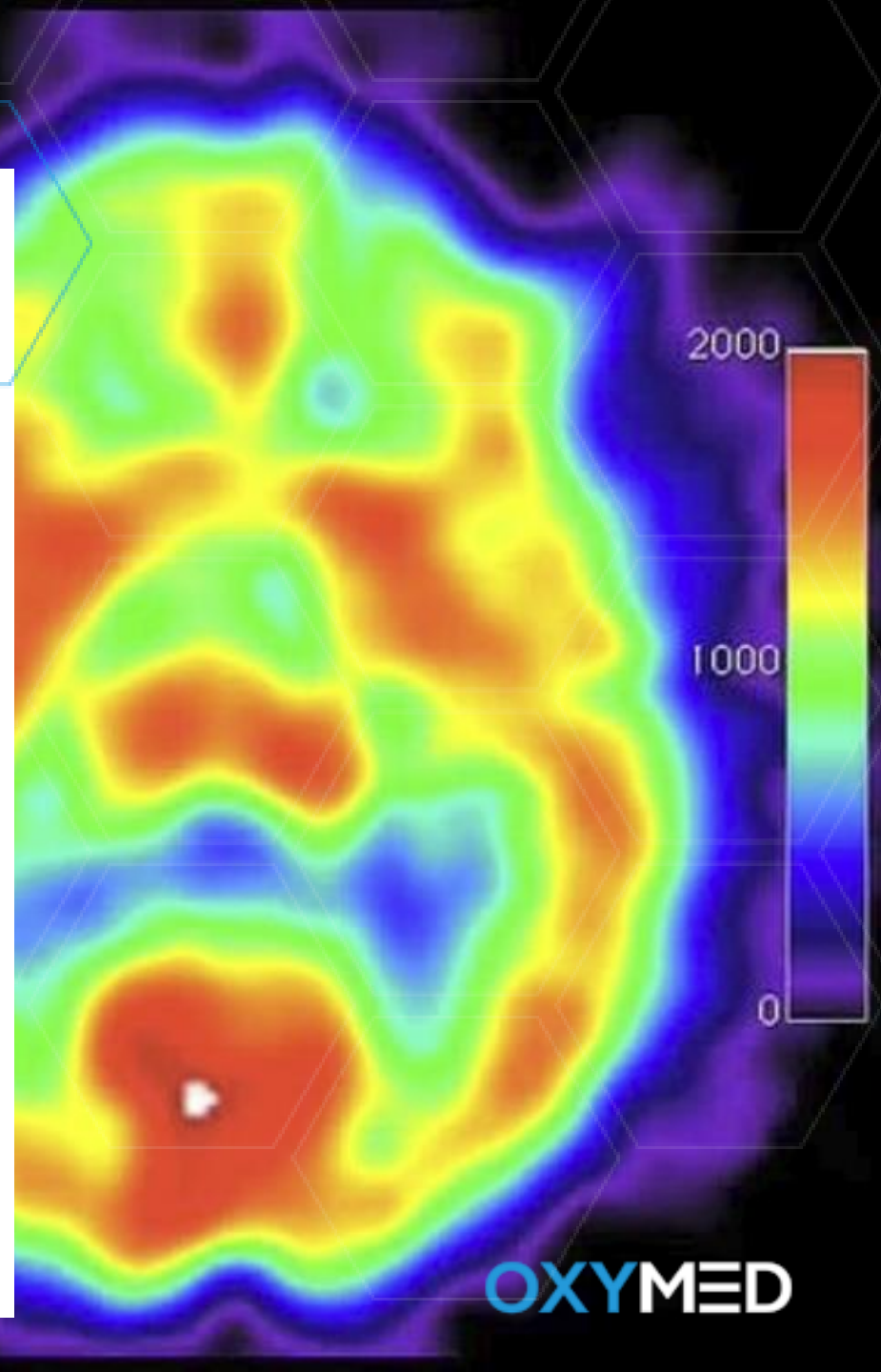
Date of Birth : 20-Nov-2012
Sex : M
Collected : 16-Apr-2018

Lab id: UR#:

OXYMED, 643 CHAPEL STREET
SOUTH YARRA VIC 3141

INTEGRATIVE MEDICINE

BLOOD - SERUM	Result	Range	Units
Hyperbaric Oxygen Therapy (HBO)	120.0		Hours
CYTOKINES,Extensive Panel			
ProInflammatory Cytokines (TH1)			
Interleukin 1	*9.6* *H	0.0 - 2.8	pg/mL
Interleukin 6	7.2	0.0 - 11.0	pg/mL
Interleukin 7	*34.3* *H	0.0 - 16.0	pg/mL
Interleukin 8	*317.9* *H	0.0 - 28.0	pg/mL
Interleukin 17	*33.0* *H	< 13.0	pg/mL
TNFa	*33.40* *H	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	*514.0* *H	0.0 - 80.0	pg/mL
Interleukin 2	9.6	0.0 - 10.0	pg/mL
Interleukin 3	<1.0	< 5.0	pg/mL
Interleukin 4	*61.3* *H	0.0 - 19.0	pg/mL
Interleukin 5	5.2	0.0 - 13.0	pg/mL
Interleukin 10	*34.0* *H	0.0 - 7.0	pg/mL
Interleukin 12	13.1	0.0 - 14.0	pg/mL
Interleukin 13	*37.3* *H	0.0 - 6.0	pg/mL
INFg	28.0	0.0 - 28.0	pg/mL
TGFb	37.0	28.0 - 64.0	pg/mL
Brain Derived Neurotrophic Factor BDNF	*52.0* *H	20.0 - 50.0	ng/mL



Autism Case Study DC – before and after HBOT

OXYMED HEALTH

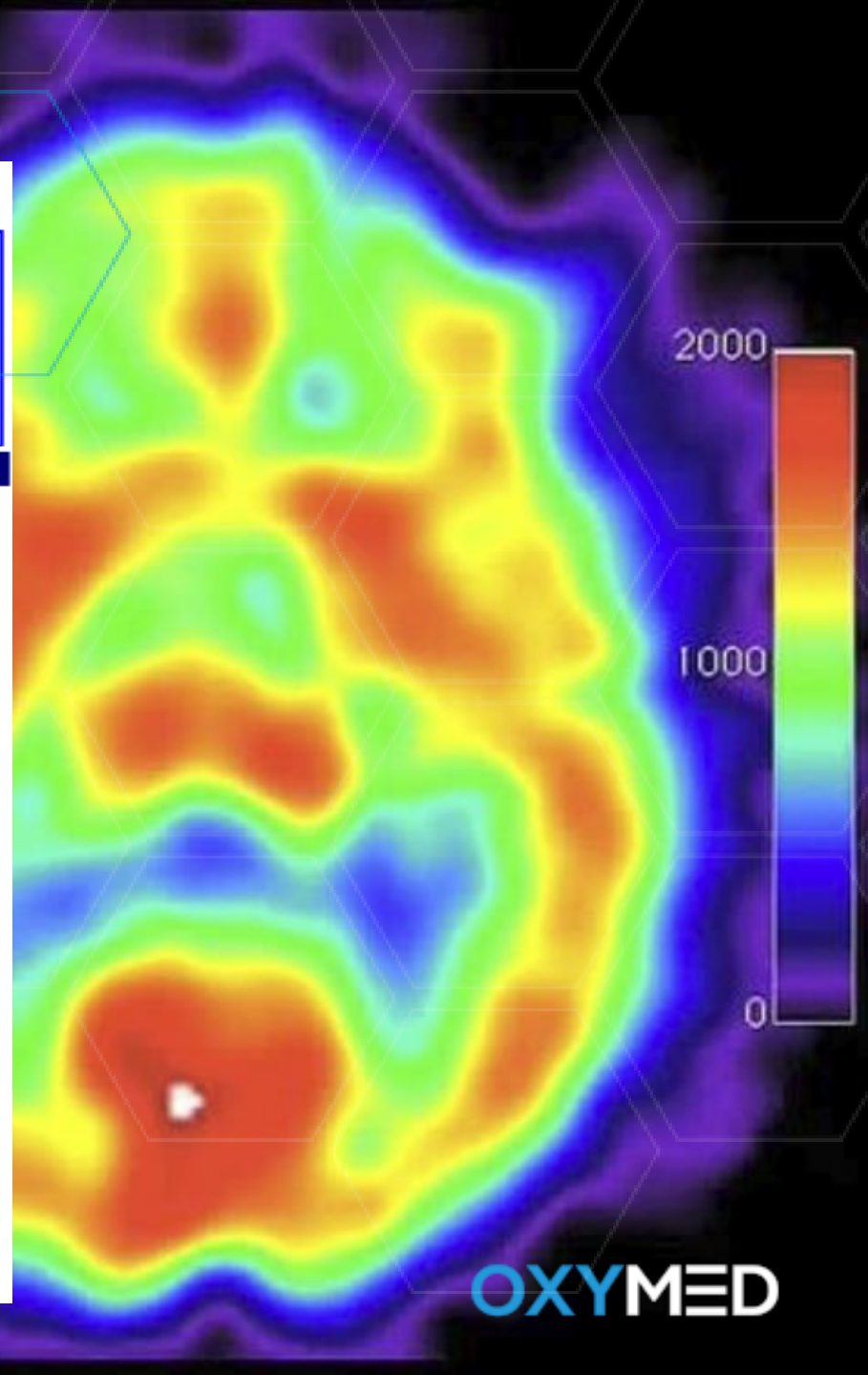
Date of Birth : 20-Nov-2013
Sex : M
Collected : 16-Jan-2018

OXYMED, 643 CHAPEL STREET
SOUTH YARRA VIC 3141

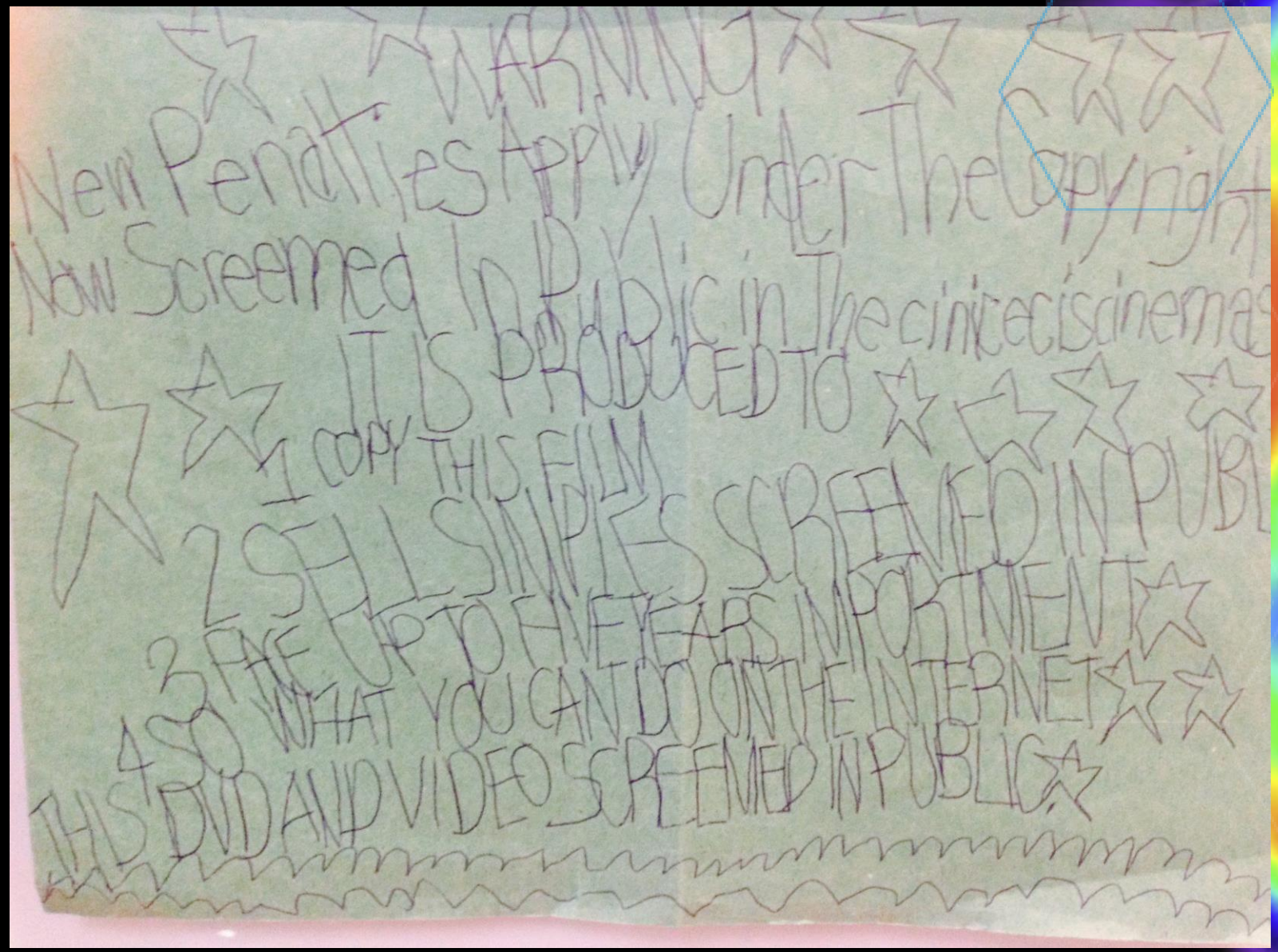
Lab id: UR#:

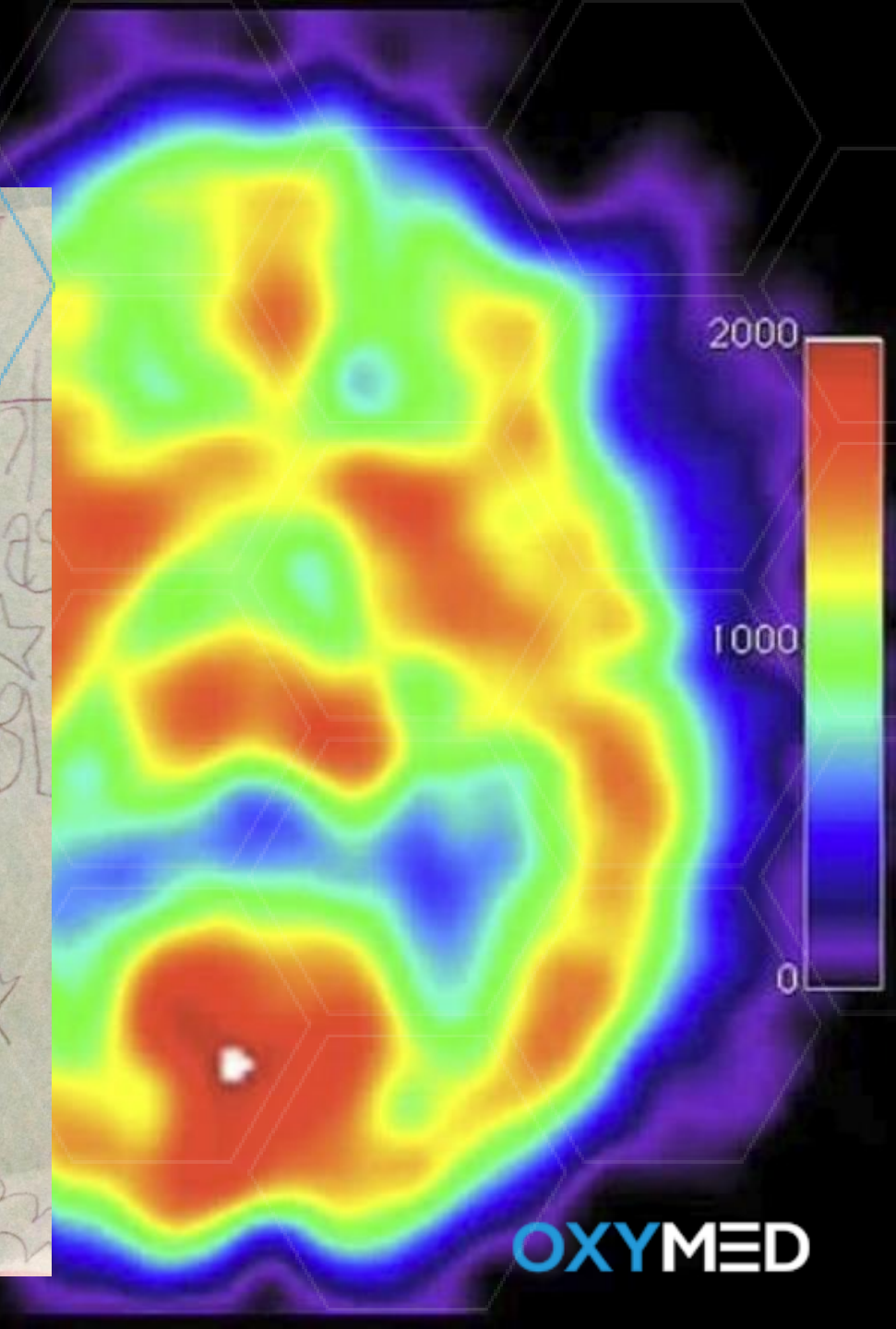
INTEGRATIVE MEDICINE

BLOOD - SERUM	Result	70.0	120.0	
CYTOKINES,Extensive Panel				
ProInflammatory Cytokines (TH1)				
Interleukin 1	*6.2* *H	*945.6* *H	*9.6* *H	0.0 - 2.8
Interleukin 6	6.7	<0.4	7.2	0.0 - 11.0
Interleukin 7	*22.7* *H	*43.9* *H	*34.3* *H	0.0 - 16.0
Interleukin 8	*106.3* *H	*>2500.0* *H	*317.9* *H	0.0 - 28.0
Interleukin 17	*26.7* *H	*16.6* *H	*33.0* *H	< 13.0
TNFa	*21.60* *H	*213.90* *H	*33.40* *H	0.00 - 13.00
TNFb	98.0	123.0	93.0	0.0 - 156.0
S100B	*>5000.0* *H	*639.0* *H	*<10.0* *L	60.0 - 100.0
AntiInflammatory Cytokines (TH2)				
GM-CSF	*1217.7* *H	*1710.3* *H	*514.0* *H	0.0 - 80.0
Interleukin 2	7.3	9.8	9.6	0.0 - 10.0
Interleukin 3	*9.6* *H	1.3	<1.0	< 5.0
Interleukin 4	*30.6* *H	*34.7* *H	*61.3* *H	0.0 - 19.0
Interleukin 5	5.4	4.7	5.2	0.0 - 13.0
Interleukin 10	*35.3* *H	*56.9* *H	*34.0* *H	0.0 - 7.0
Interleukin 12	*15.6* *H	*18.2* *H	13.1	0.0 - 14.0
Interleukin 13	*24.0* *H	*30.0* *H	*37.3* *H	0.0 - 6.0
INFg	18.5	23.1	28.0	0.0 - 28.0
TGFb	36.4	33.0	37.0	28.0 - 64.0
Brain Derived Neurotrophic Factor BDNF	47.0	39.0	*52.0* *H	20.0 - 50.0

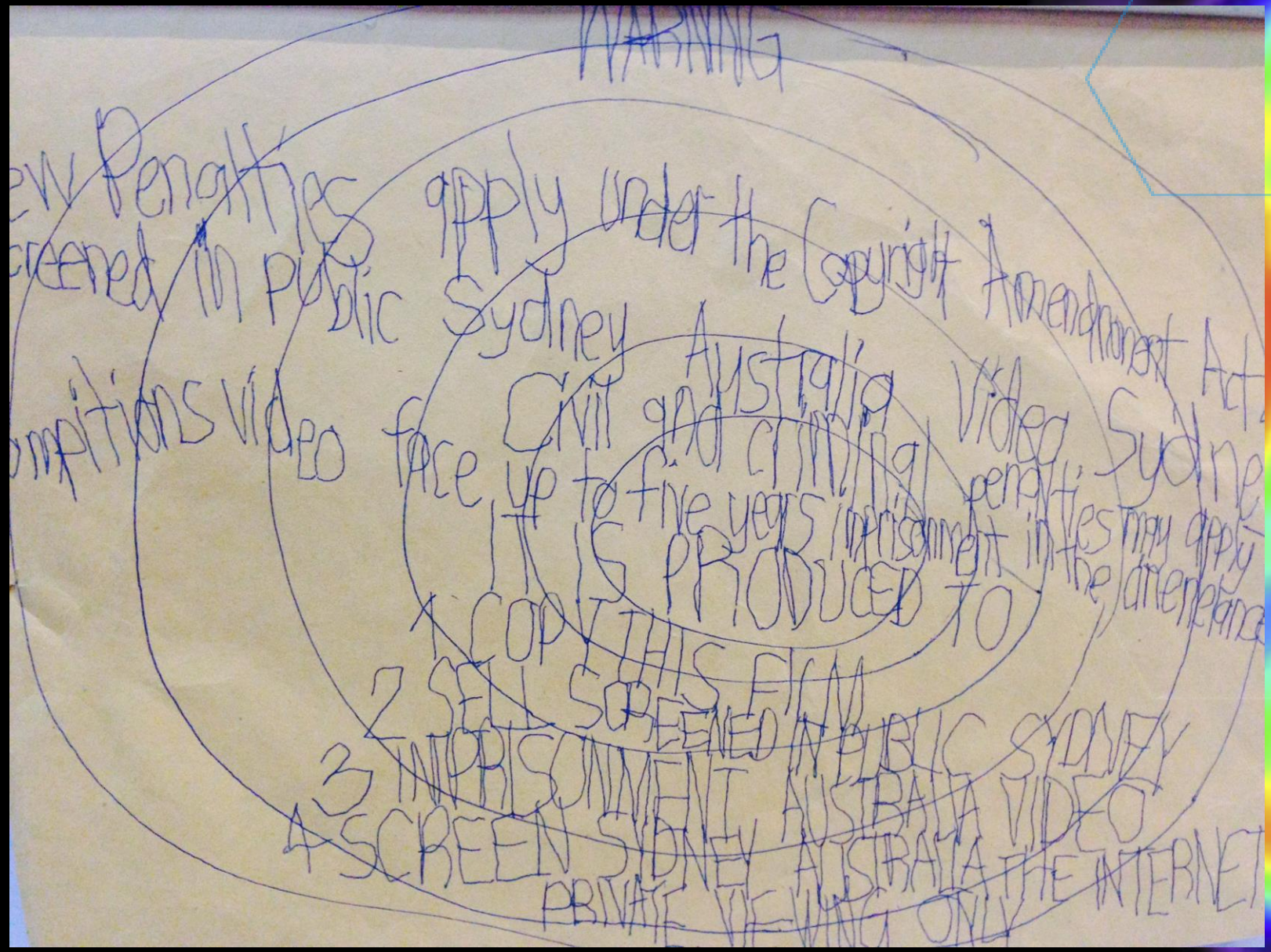


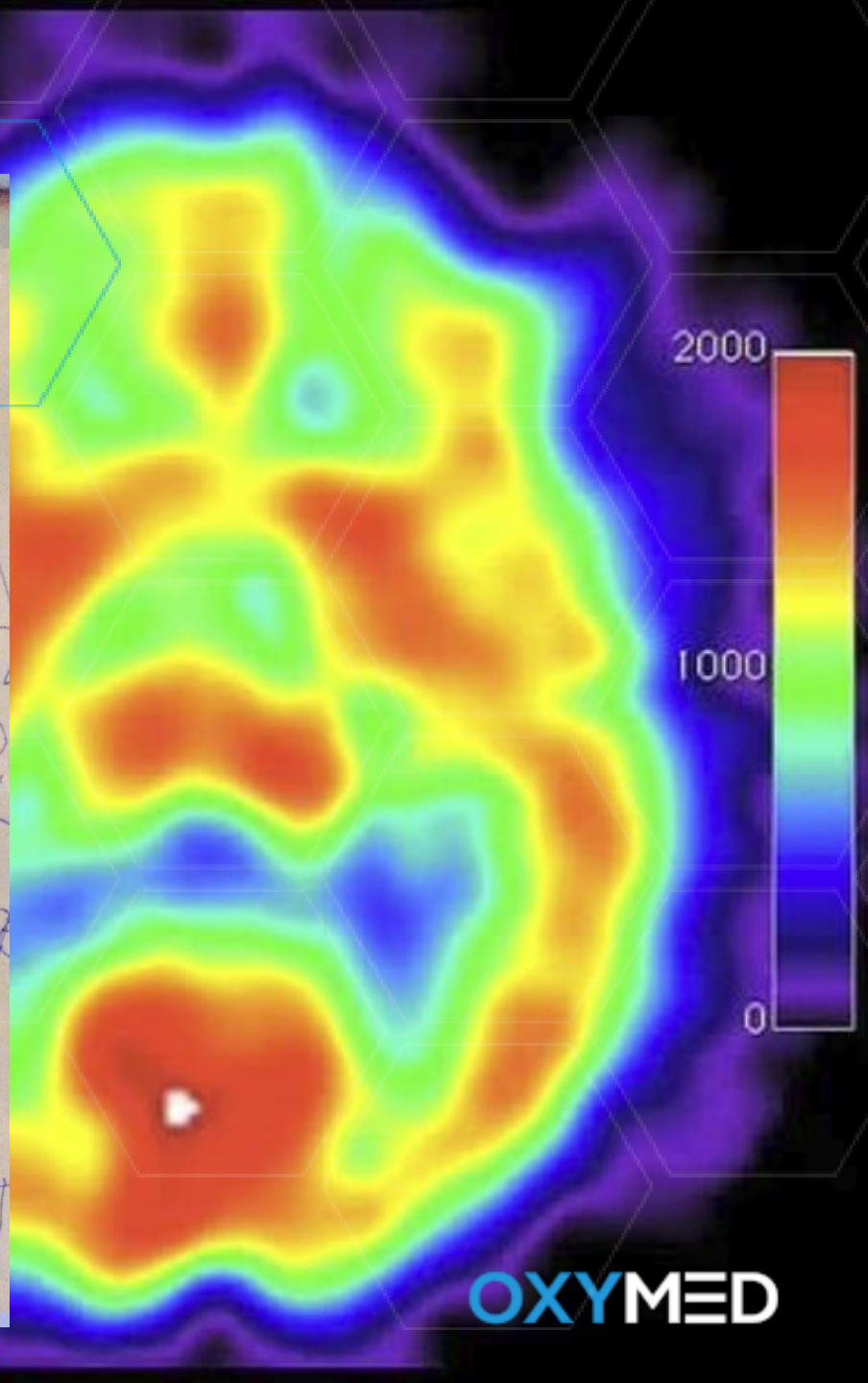
Autism ADHD - Case Study JP – after 45 hours HBOT



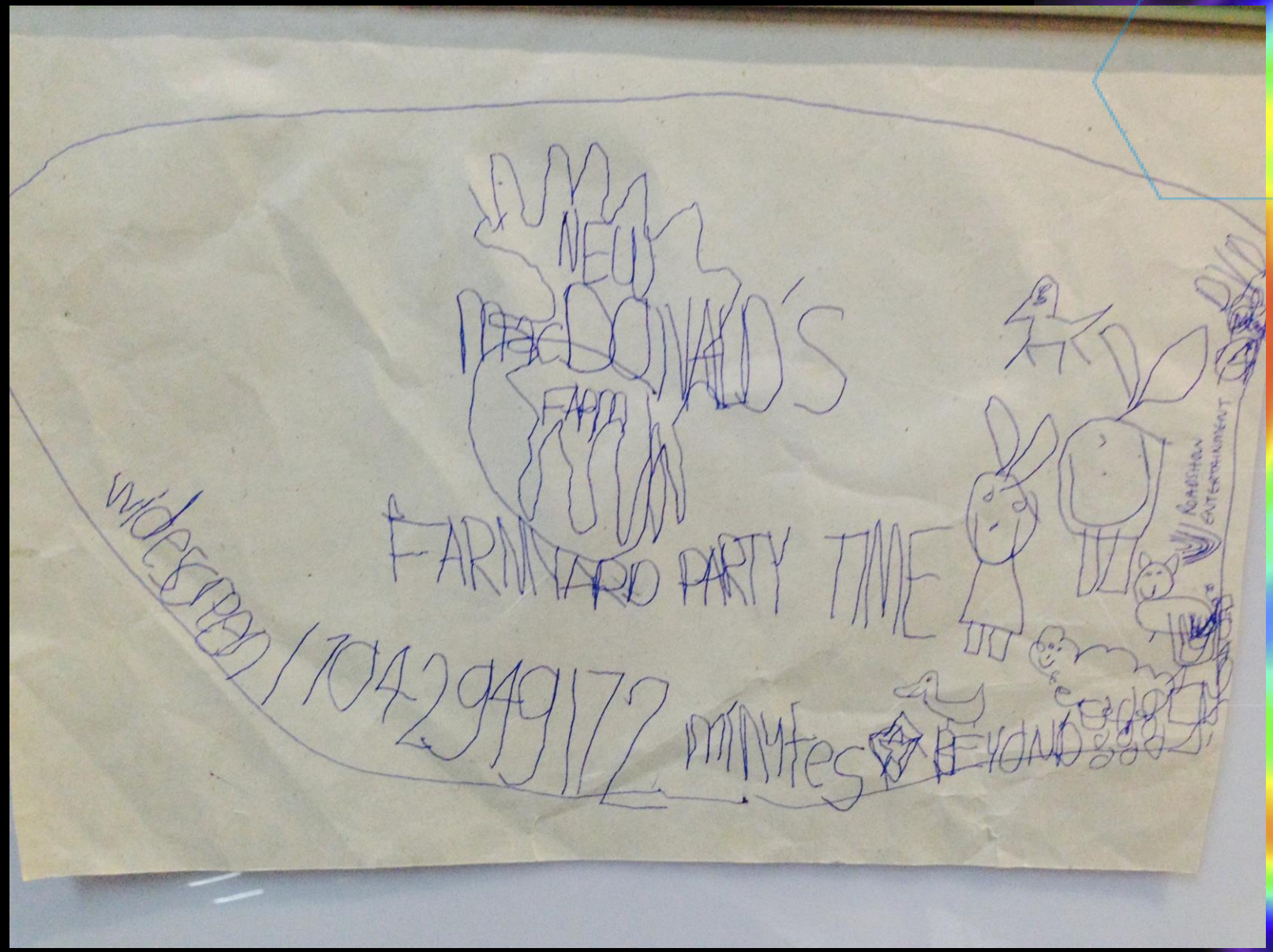


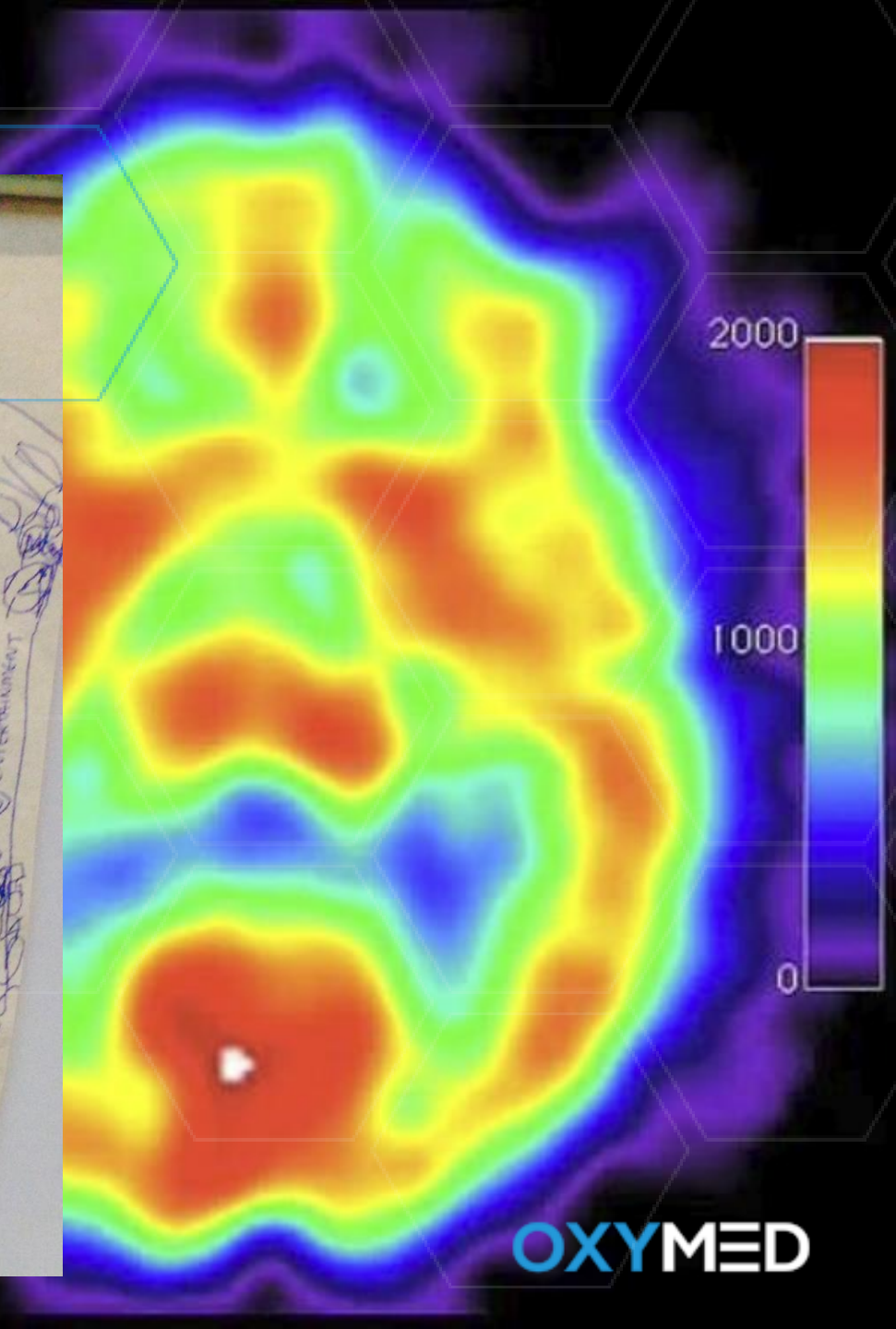
Case Study JP – after 45 hours HBOT





Case Study JP – after 45 hours HBOT





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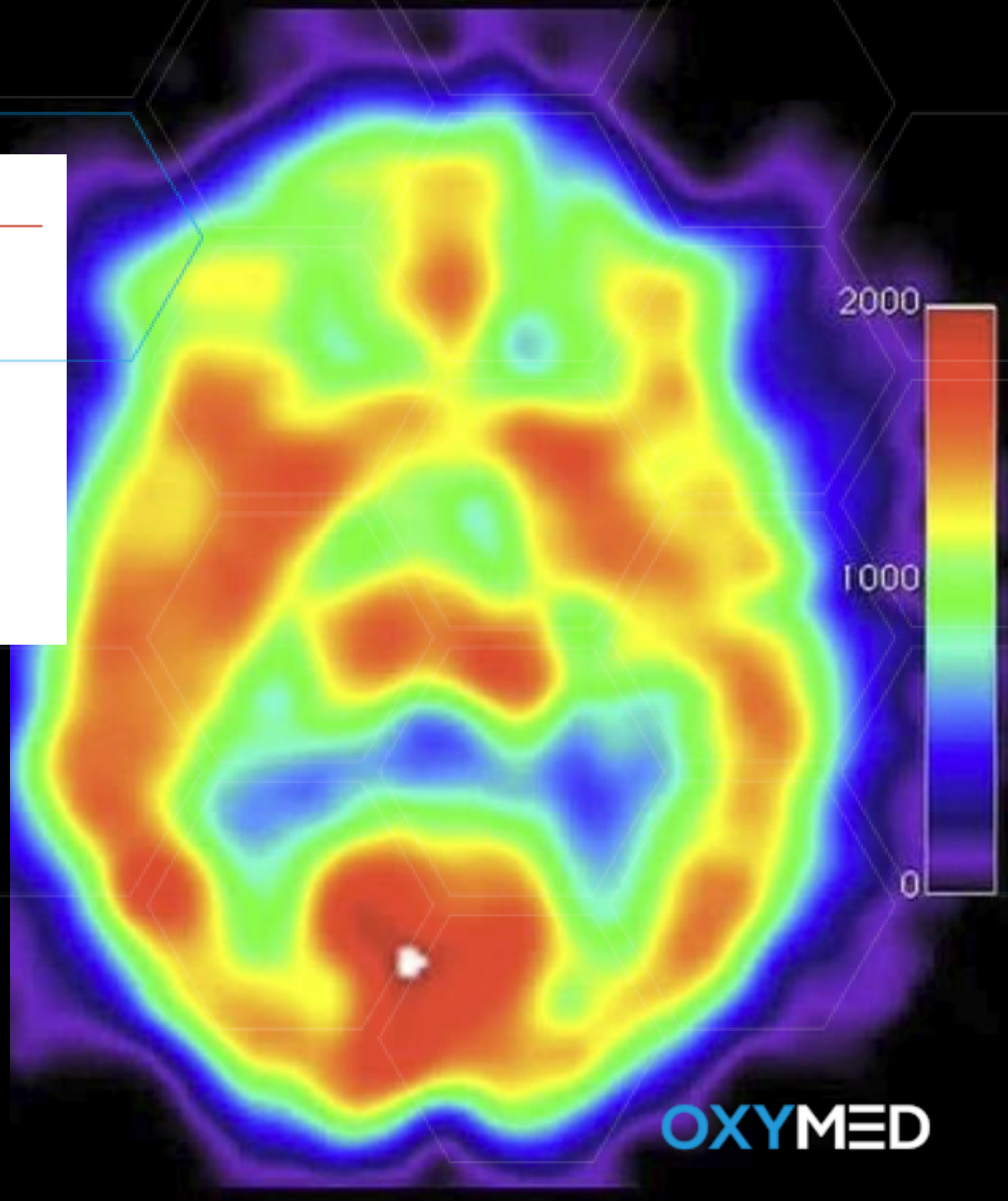
Cerebrolysin – Brain Derived Neurotrophic Factor

WELCOME TO CEREBROLYSIN®

Cerebrolysin® is a multi-modal neuropeptide drug which improves the brain's ability for self-repair by stimulating neurorecovery.

Cerebrolysin® is used for treatment of ischemic and hemorrhagic stroke, traumatic brain injuries (TBI), different forms of dementia (vascular dementia, Alzheimer's disease) and cognitive disorders and to prevent cognitive decline after brain injuries.

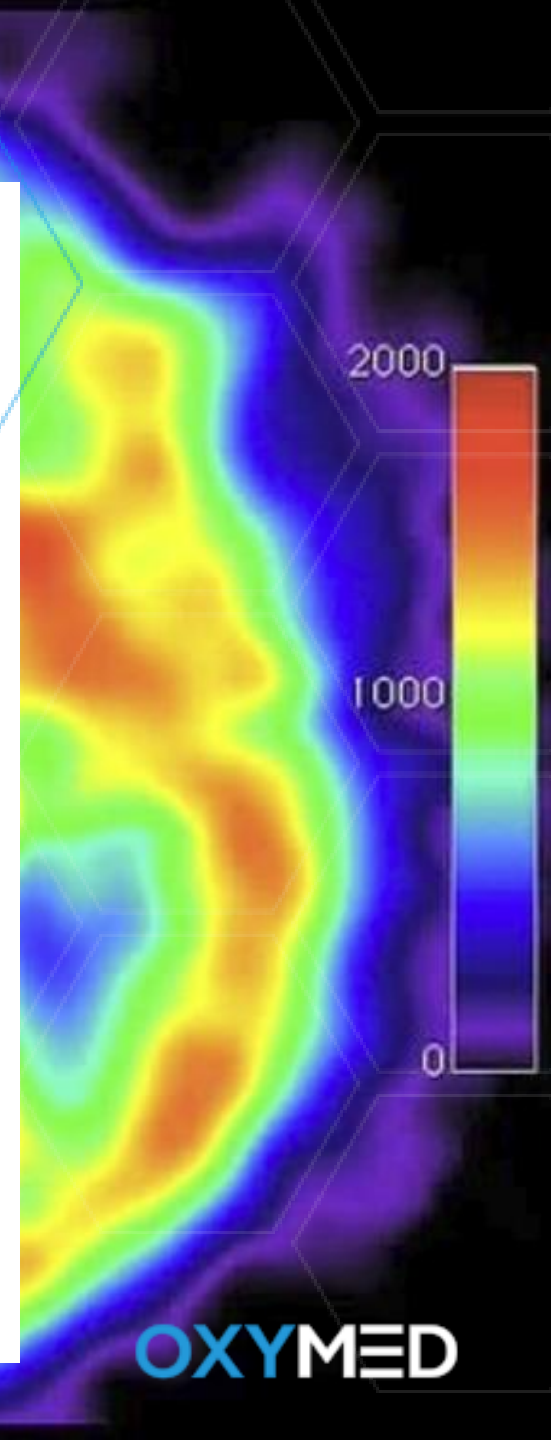
READ MORE



Cerebrolysin – Brain Derived Neurotrophic Factor

www.oxymed.com.au/cerebrolysin

- Brain derived peptide with neurotrophic factors – brain derived neurotrophic factor (BDNF), glial cell line derived neurotrophic factor (GDNF), nerve growth factor (NGF), ciliary neurotrophic factor (CNTF) and other peptide fragments.
- CL neurotrophic modulation shown to improve cognitive performance and global function in numerous neurodegenerative disorders and mental illness with 'increased daily quality living'.
- CL potentiates brain alpha activity, reduces slow EEG delta frequencies; improved memory performance in healthy elderly humans, suggesting CL activates cerebral mechanisms related to attention and memory processes.
- CL improves cognitive deficits and global function in patients with mild to moderate progressive neurodegenerative disease including Multiple Sclerosis, Parkinson's Disease, Alzheimer's Disease, Dementia, Acute and Chronic Stroke victims.
- CL improves post-acute traumatic brain injury; childhood autism (89%) and cerebral palsy.
- CL attenuates motor neuron damage in spinal cord and nerve root damage – with significant motor recovery. Neuro-immunotrophic reducing chronic nerve cell inflammation in both acute traumatic and chronic progressive neurodegenerative diseases (progressive arthritis).
- Neuroprotective and neurorestorative properties, demonstrates 'anti-aging' with benefits 'improving cognition, memory function, brain metabolism with capacity.



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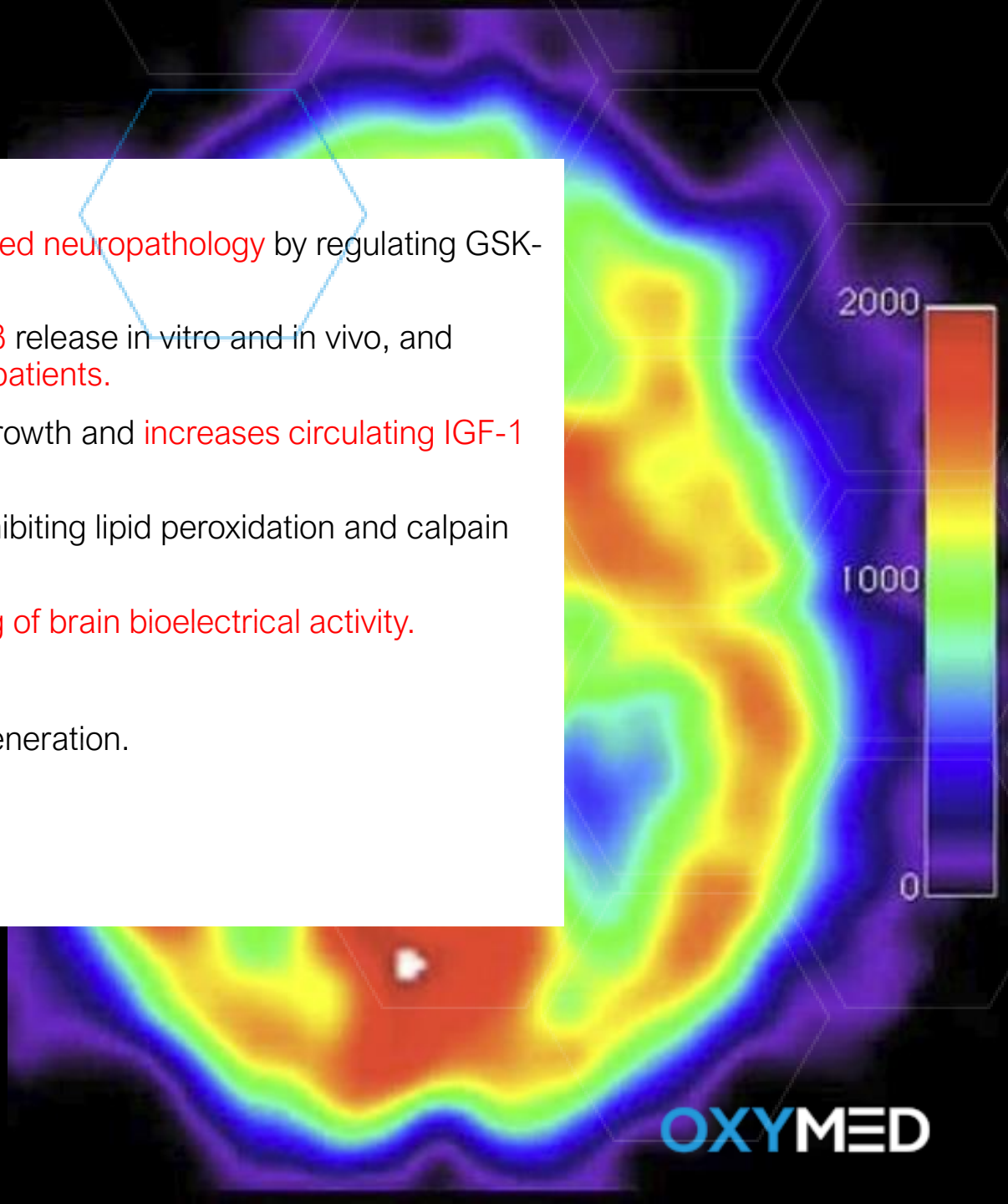
Cerebrolysin – Brain Derived Neurotrophic Factor

In pharmacodynamic studies it has been demonstrated that Cerebrolysin:

- Reduces brain Aβ deposition, tau phosphorylation and Aβ- and tau-related neuropathology by regulating GSK-3β and CDK-5 activity.
- Modulates neuroinflammation, attenuating microglia activation and IL-1β release in vitro and in vivo, and reducing the elevated serum levels of TNF-α and TNF receptor-1 in AD patients.
- Displays neurotrophic-like actions on neuronal survival and neurite outgrowth and increases circulating IGF-1 and BDNF levels in humans.
- Protects against oxidative and excitotoxic damage, at least in part by inhibiting lipid peroxidation and calpain activation.
- Enhances the supply of glucose to the brain and ameliorates the slowing of brain bioelectrical activity.
- Promotes neural plasticity and prevents dendritic and synaptic loss.
- Promotes neuronal survival protecting neurons from apoptosis and degeneration.
- Stimulates neurogenesis, probably through Akt activation.
- Improves learning and memory.



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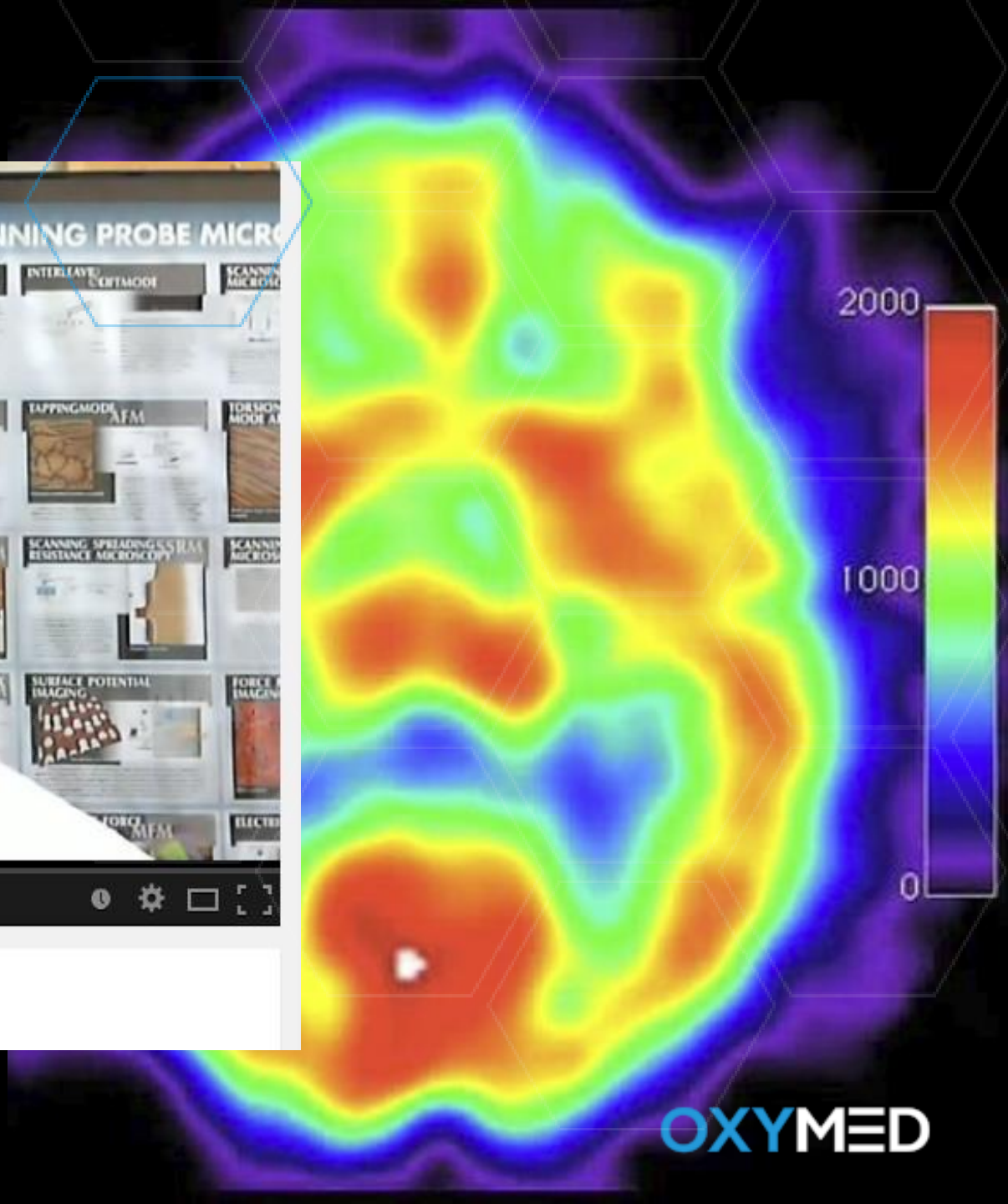
Prof Dominic D’Agostino - Influencer

Starving Cancer with Dr. Dominic D'Agostino



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Ketogenic Diet & HBOT

The Ketogenic Diet and Hyperbaric Oxygen Therapy Prolong Survival in Mice with Systemic Metastatic Cancer

Angela M. Poff[1]*, Csilla Ari[1], Thomas N. Seyfried[2], Dominic P. D'Agostino[1]

1 Department of Molecular Pharmacology and Physiology, University of South Florida, Tampa, Florida, United States of America, 2 Department of Biology, Boston College, Chestnut Hill, Massachusetts, United States of America

Abstract

Introduction: Abnormal cancer metabolism creates a glycolytic-dependency which can be exploited by lowering glucose availability to the tumor. The ketogenic diet (KD) is a low carbohydrate, high fat diet which decreases blood glucose and elevates blood ketones and has been shown to slow cancer progression in animals and humans. Abnormal tumor vasculature creates hypoxic pockets which promote cancer progression and further increase the glycolytic-dependency of cancers. Hyperbaric oxygen therapy (HBO_2T) saturates tumors with oxygen, reversing the cancer promoting effects of tumor hypoxia. Since these non-toxic therapies exploit overlapping metabolic deficiencies of cancer, we tested their combined effects on cancer progression in a natural model of metastatic disease.

Methods: We used the firefly luciferase-tagged VM-M3 mouse model of metastatic cancer to compare tumor progression and survival in mice fed standard or KD *ad libitum* with or without HBO_2T (2.5 ATM absolute, 90 min, 3x/week). Tumor growth was monitored by *in vivo* bioluminescent imaging.

Results: KD alone significantly decreased blood glucose, slowed tumor growth, and increased mean survival time by 56.7% in mice with systemic metastatic cancer. While HBO_2T alone did not influence cancer progression, combining the KD with HBO_2T elicited a significant decrease in blood glucose, tumor growth rate, and 77.9% increase in mean survival time compared to controls.

Conclusions: KD and HBO_2T produce significant anti-cancer effects when combined in a natural model of systemic metastatic cancer. Our evidence suggests that these therapies should be further investigated as potential non-toxic treatments or adjuvant therapies to standard care for patients with systemic metastatic disease.

Citation: Poff AM, Ari C, Seyfried TN, D'Agostino DP (2013) The Ketogenic Diet and Hyperbaric Oxygen Therapy Prolong Survival in Mice with Systemic Metastatic Cancer. PLoS ONE 8(6): e65522. doi:10.1371/journal.pone.0065522

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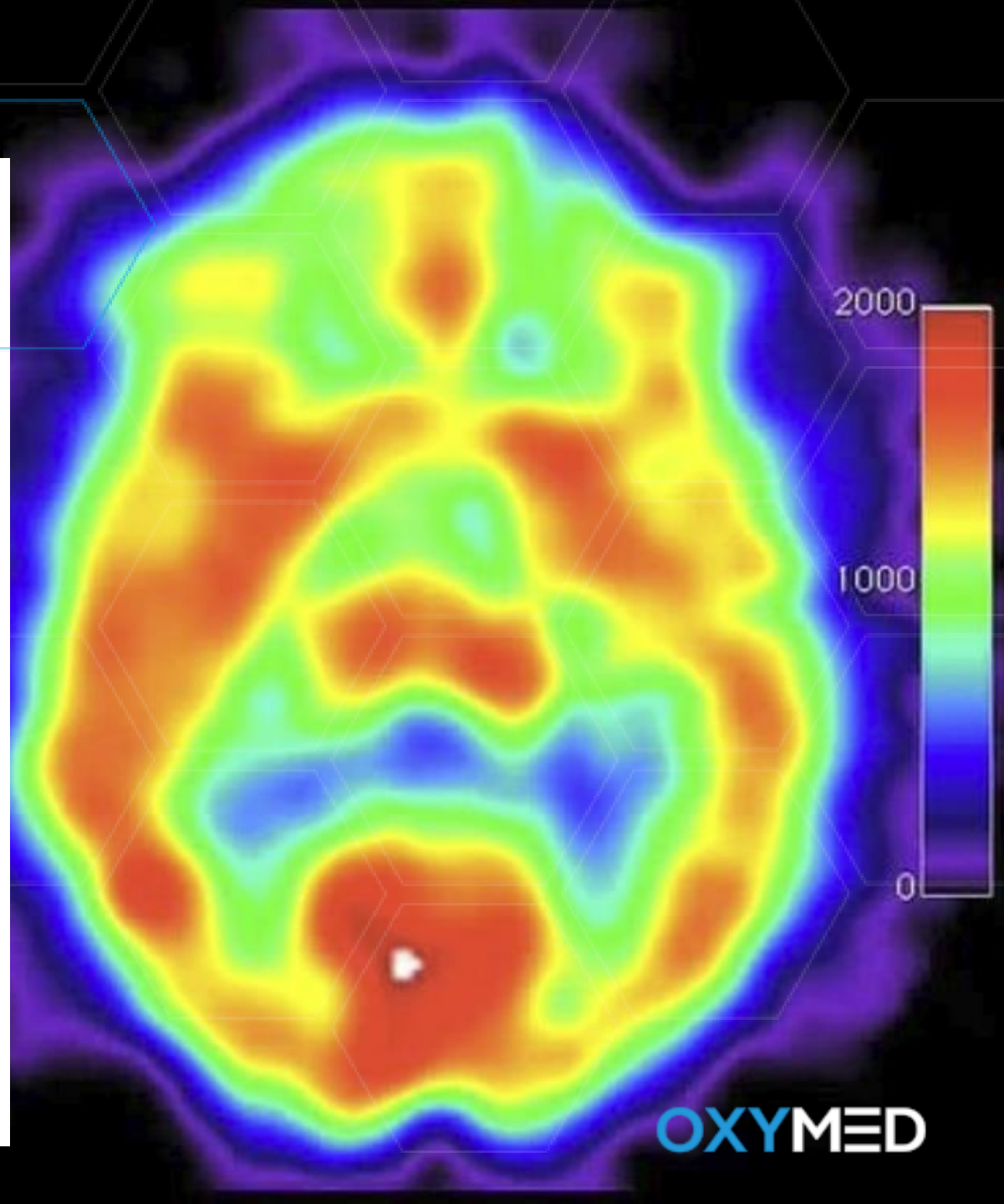
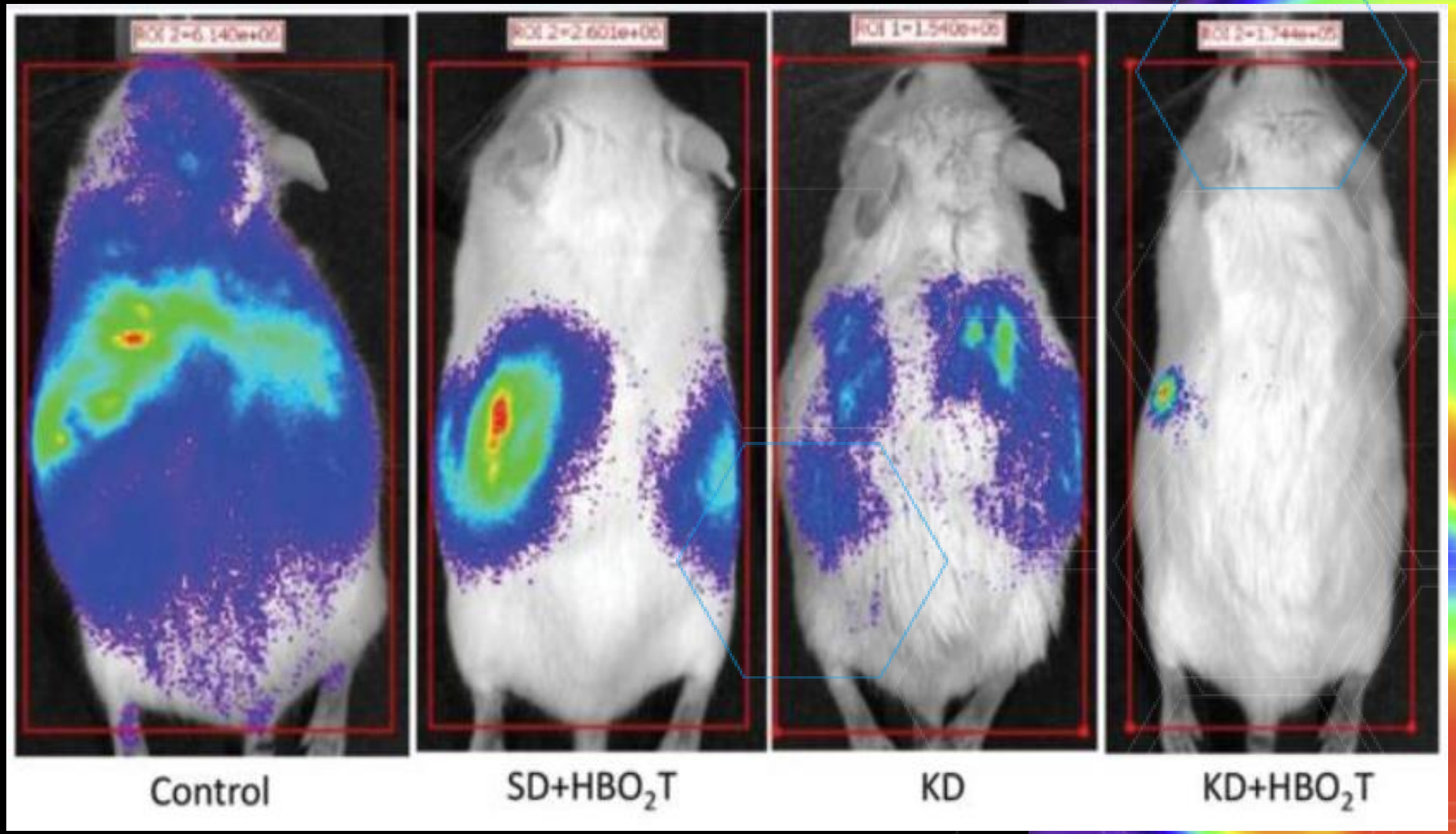
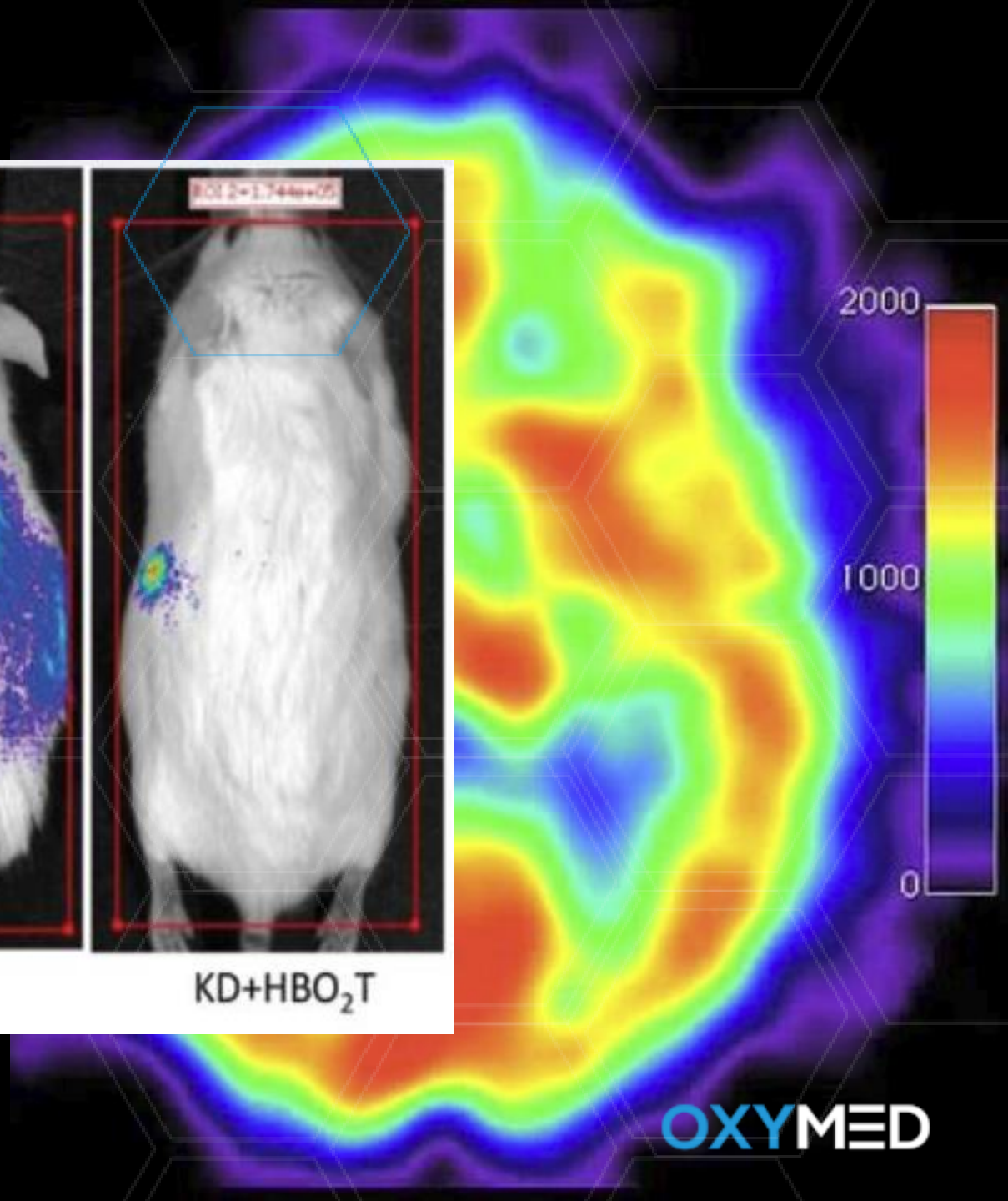
Competing Interests: The authors have declared that no competing interests exist.

* E-mail: abennett@health.usf.edu



Ketogenic Diet & HBOT





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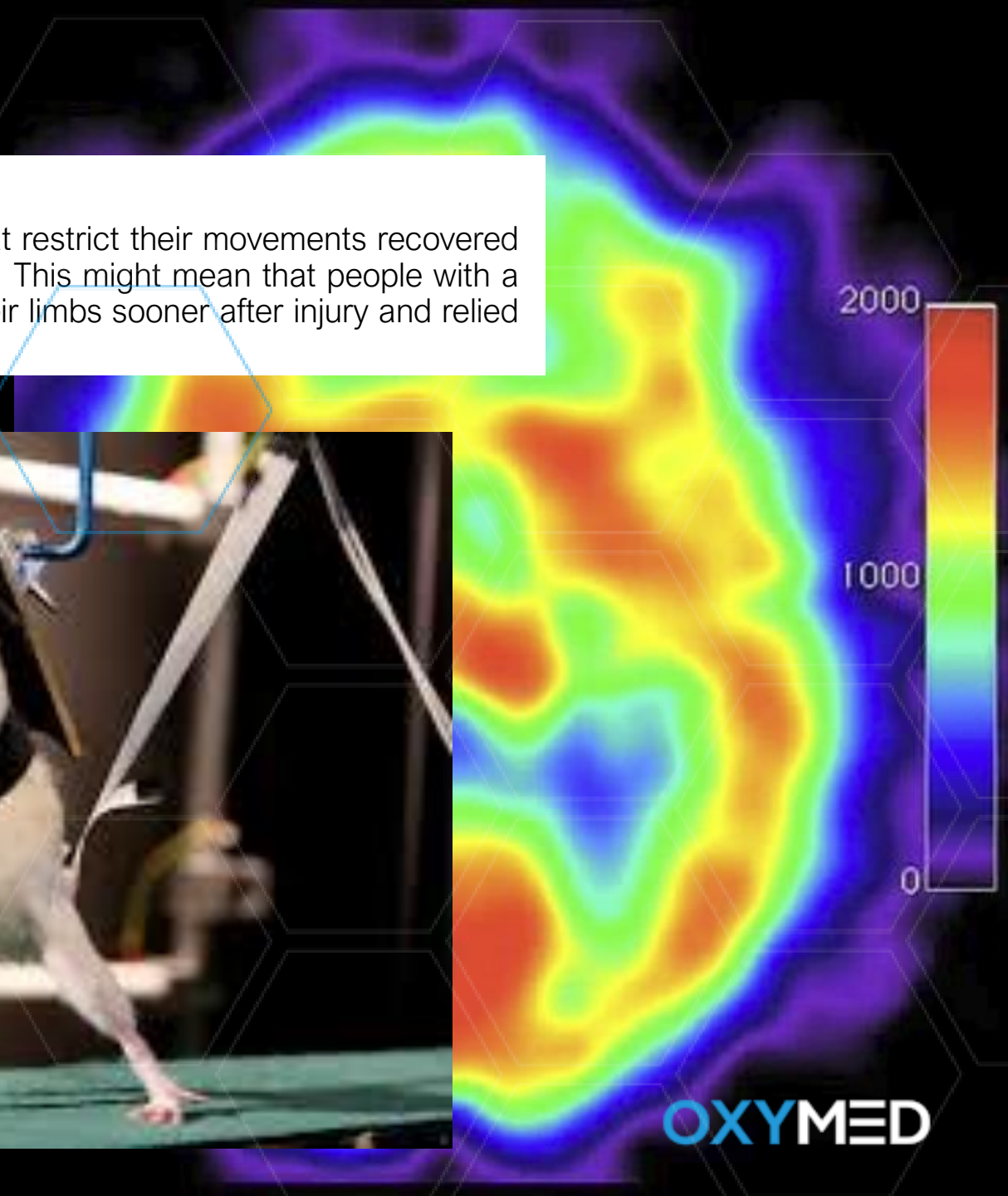
Do wheel chairs inhibit recovery?

National Neurotrauma Society Symposium in Orlando, Florida

NewScientist Aug 2008 - INJURED rats strapped to tiny "wheelchairs" that restrict their movements recovered less limb function and coordination than those left to fend for themselves. This might mean that people with a spinal cord injury would recover better if they were encouraged to use their limbs sooner after injury and relied less on wheelchairs.



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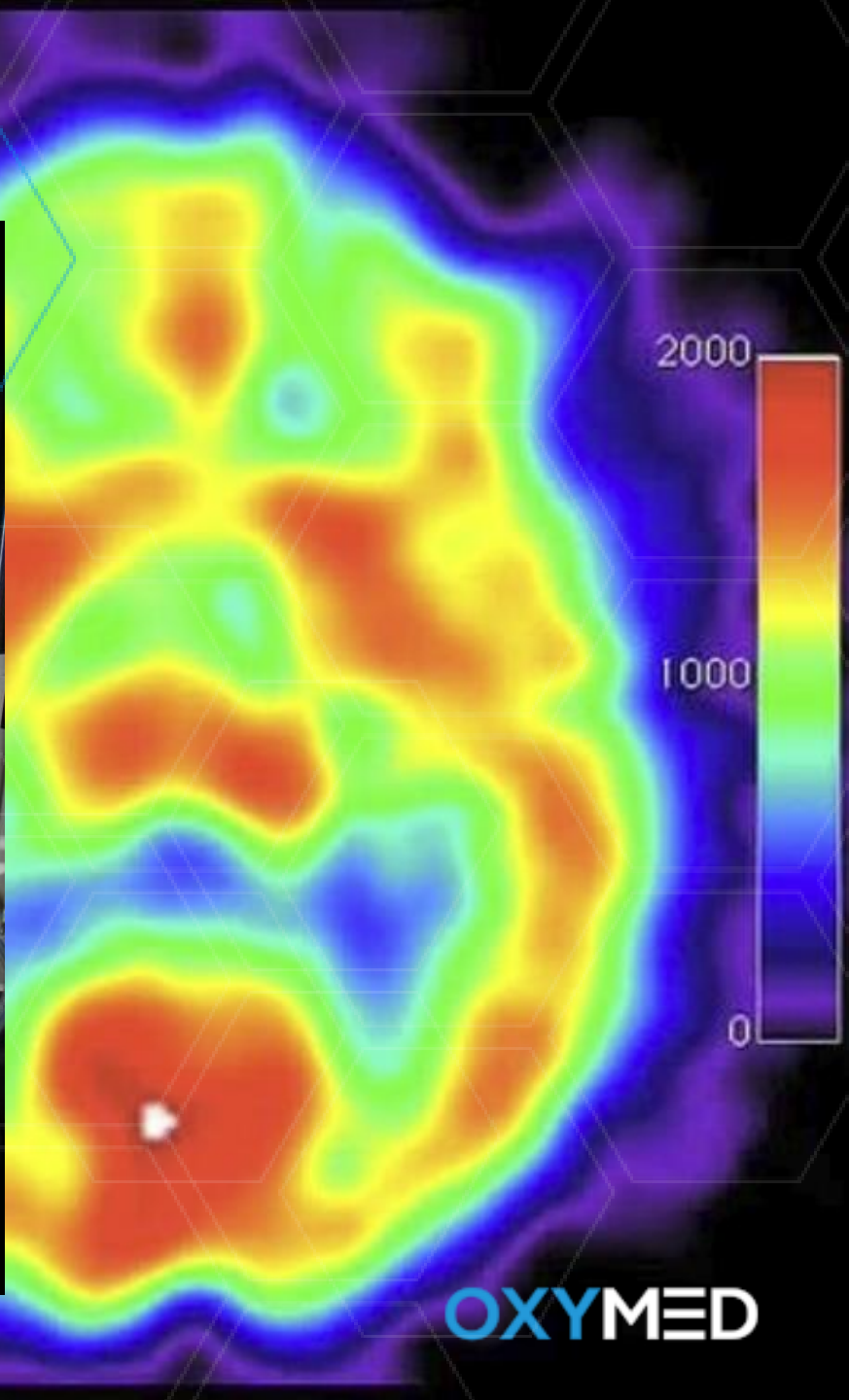
Robotic Exoskeleton Assisting Neuroplasticity



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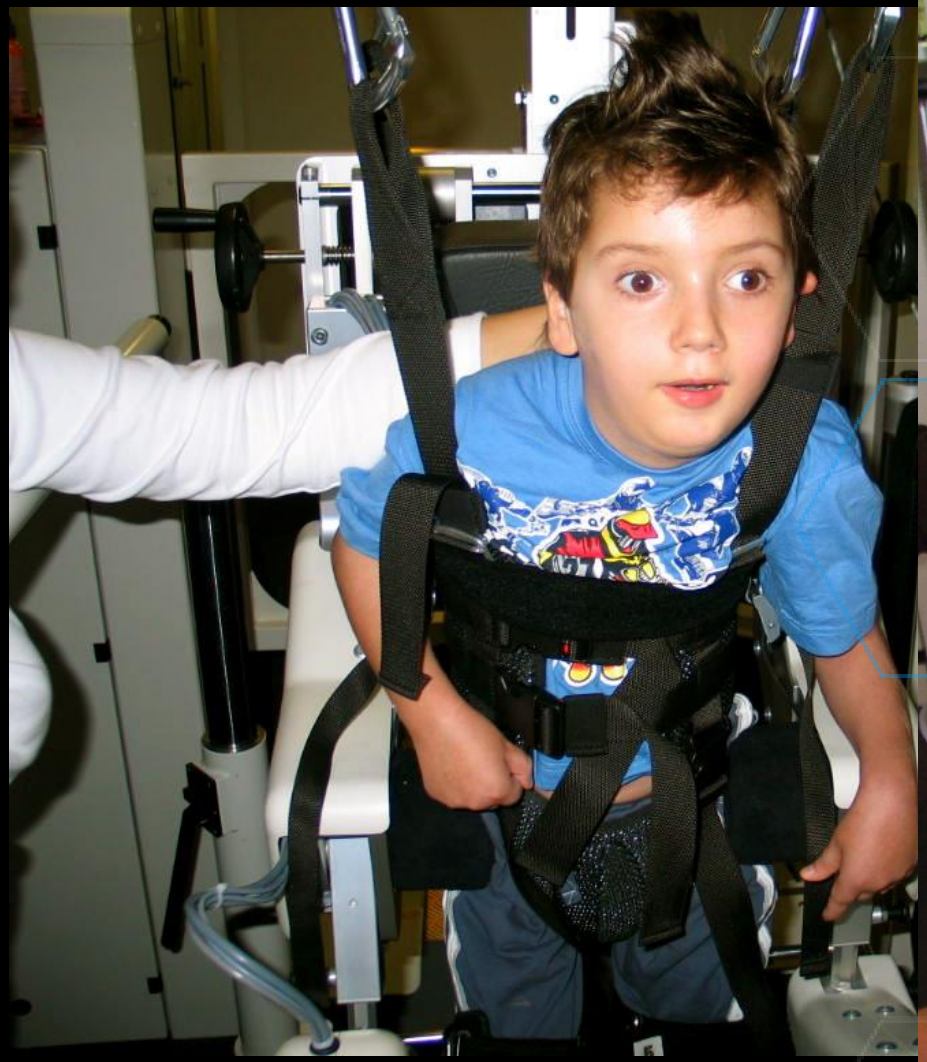
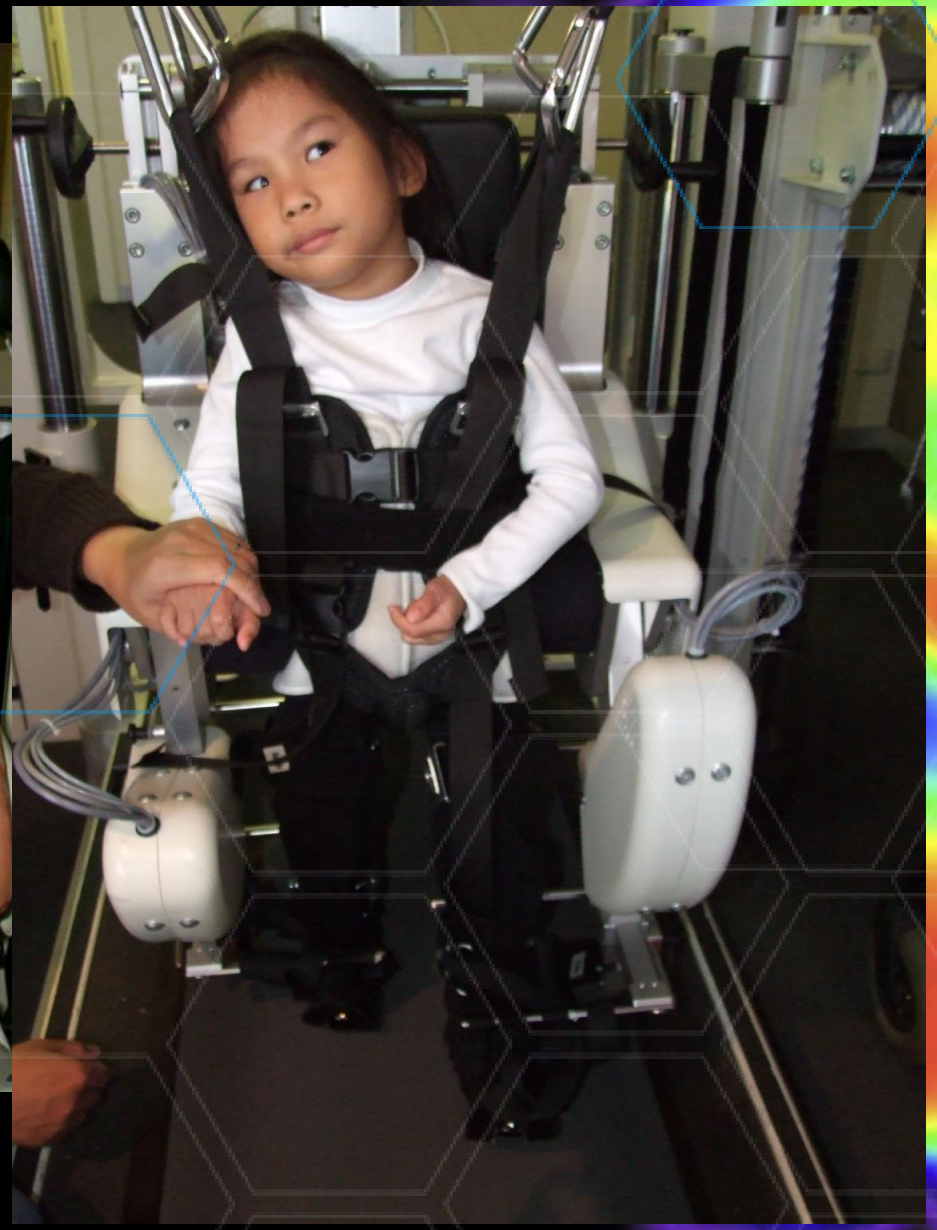
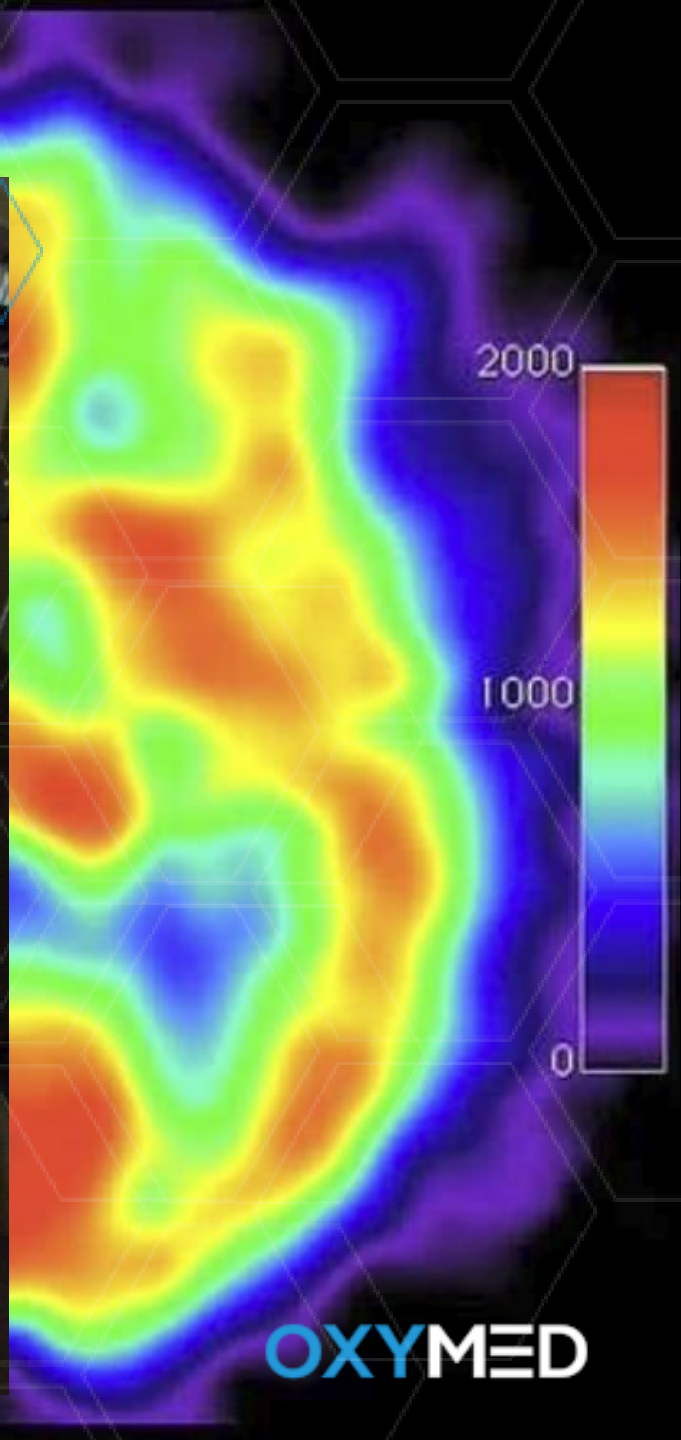
Activity Based Neurorehabilitation







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Functional Neuroplasticity

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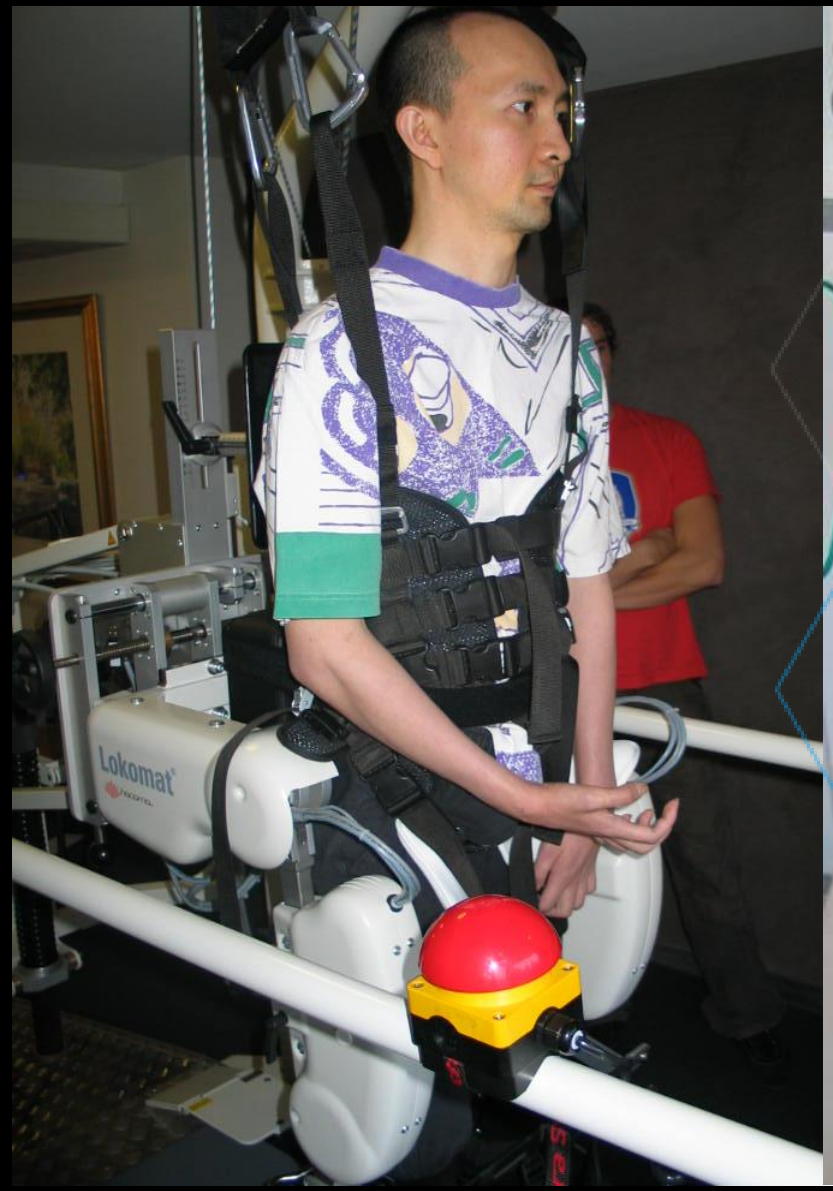
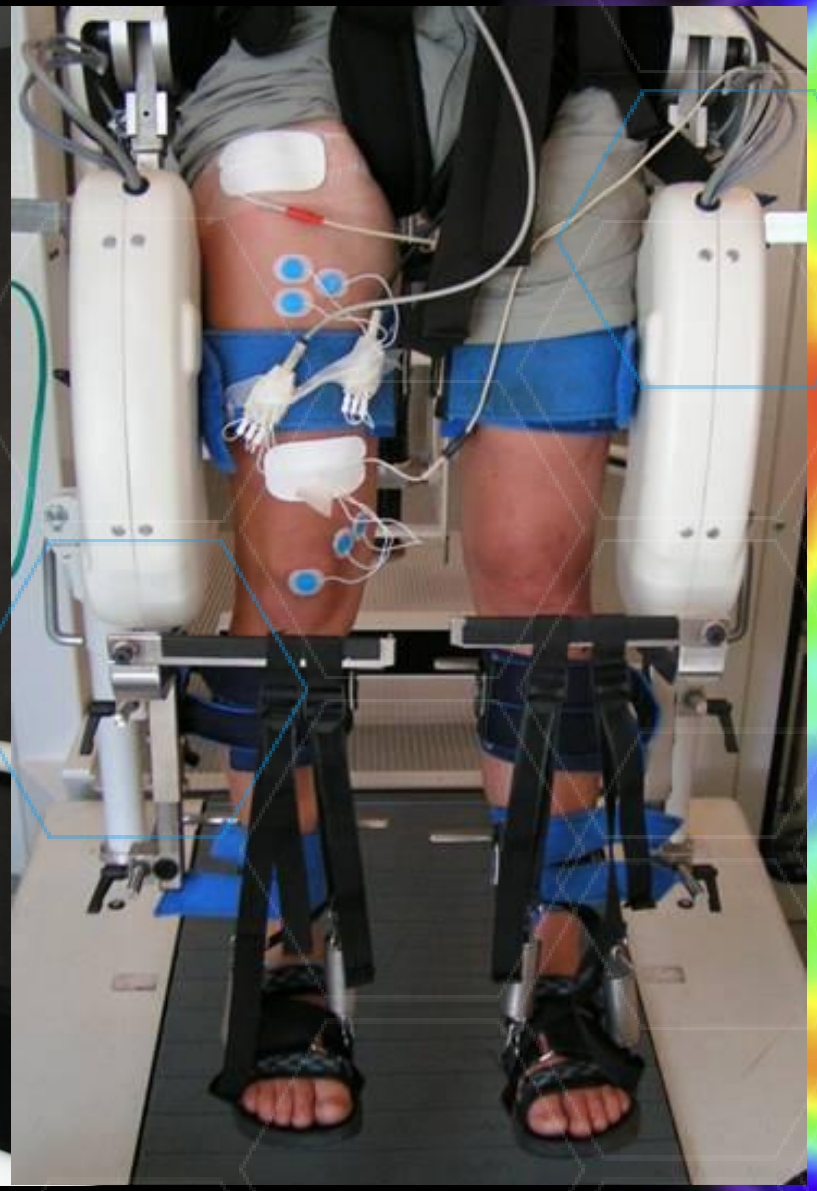
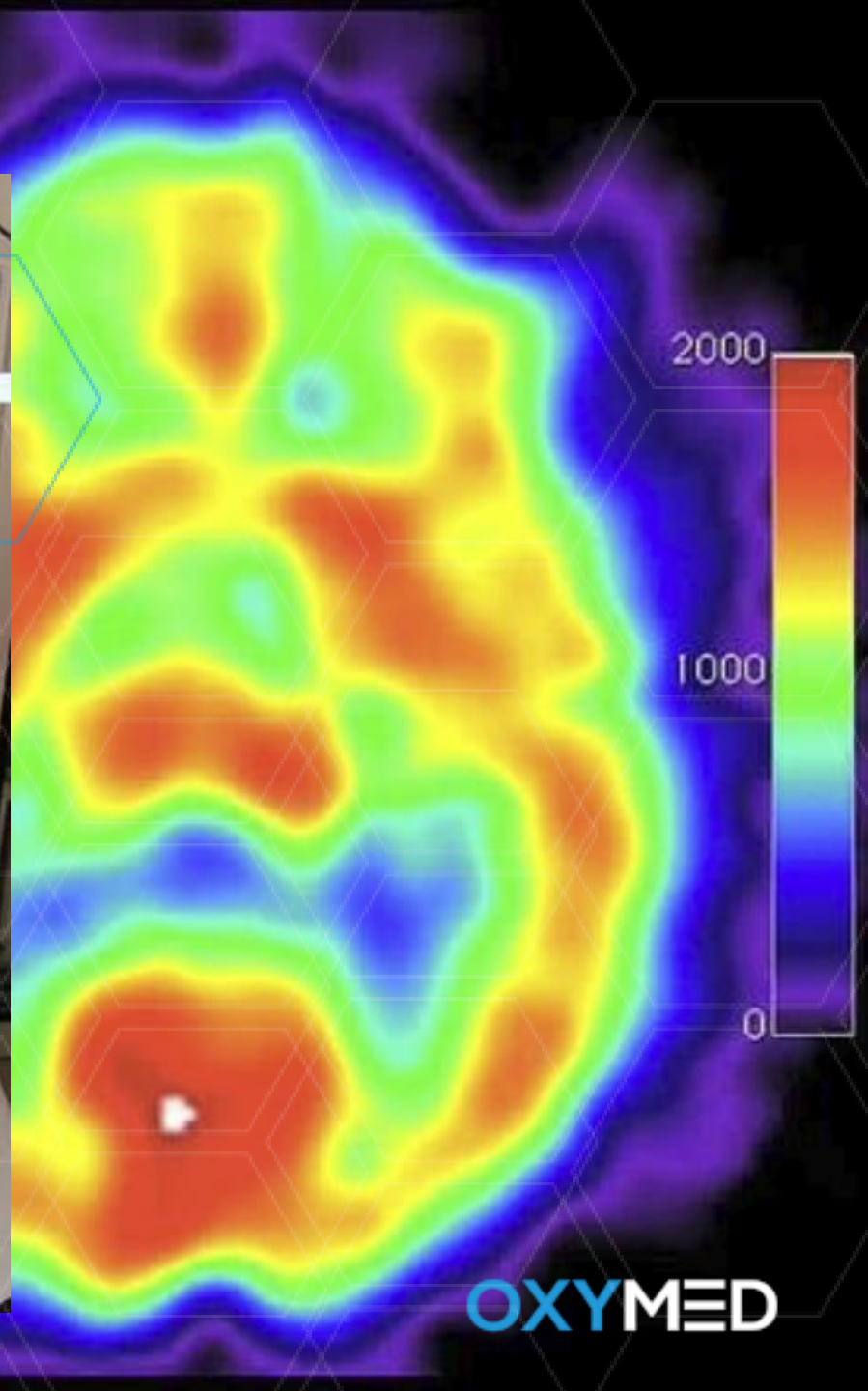
Exoskeleton Neural Priming







Next Generation Exoskeletons

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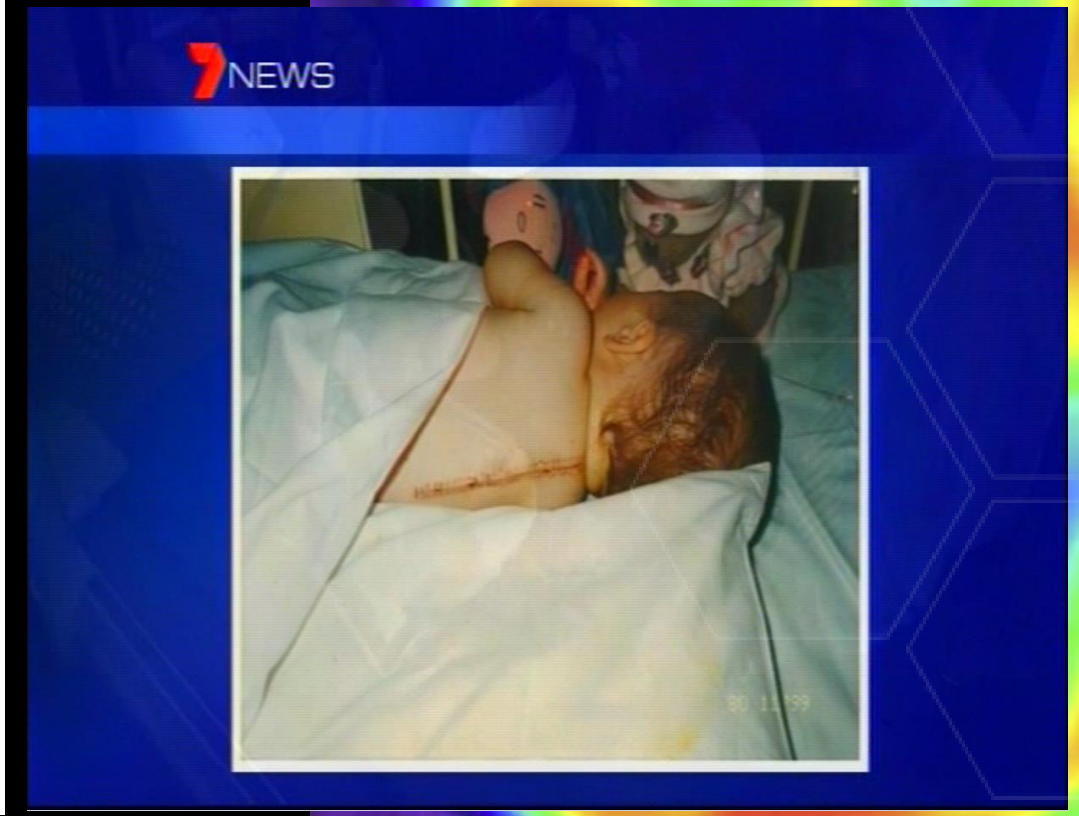
Case Study – T4 complete

Young Isabel has never walked.

At 8-months age Isabel, was operated on for a benign tumour resulting in her becoming a ‘T4 complete paraplegic’.



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Syringomyelia, cord atrophy

MRI 13-03-07:

“Comparison is made with the last examination dated 10-10-05. Again, demonstrating the enhancing intraspinal lesion posteriorly in the spinal canal at the level of T4-T6. It is again seen to measure 2.8cm cranio caudal and 1cm AP. The T2 imaging again shows cord atrophy with hydromyelia from T1 to at least the inferior border of the lesion”.

Channel 7 News - You Tube - OXYMED

https://youtu.be/f6ICtNzU6gM

https://youtu.be/ZfzWSKIKcvM

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Case Study – T12 Spinal Cord Injury

Kyle was told he would “never walk again”.

Now Kyle is walking limited distance with the assistance of calipers.

https://youtu.be/tml5KjZEBp0?t=5

2000

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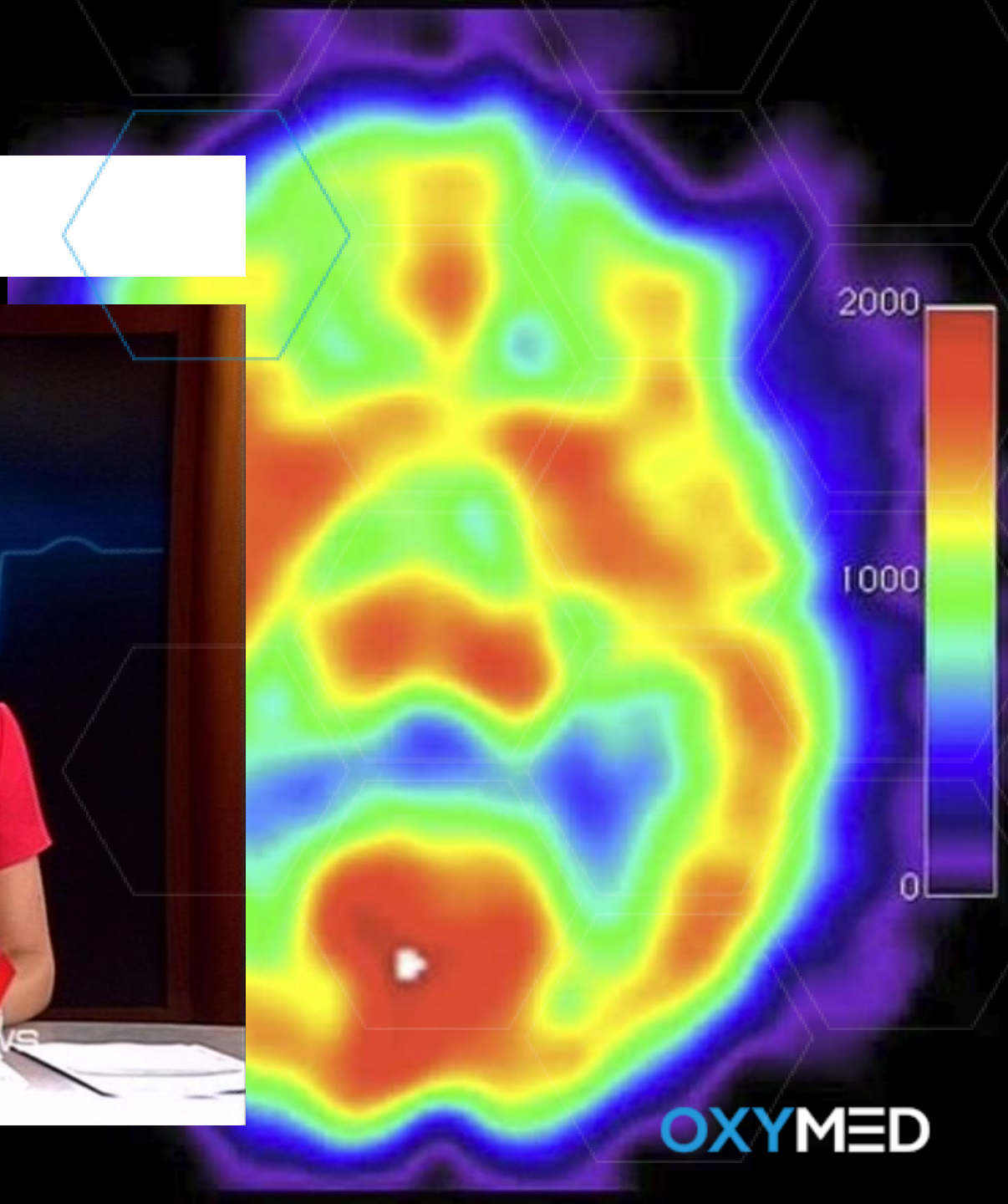
Case Study – Stroke Victim

Marco - massive arteriovenous malformation (AVM).

https://youtu.be/OMqty6COs8E?t=39



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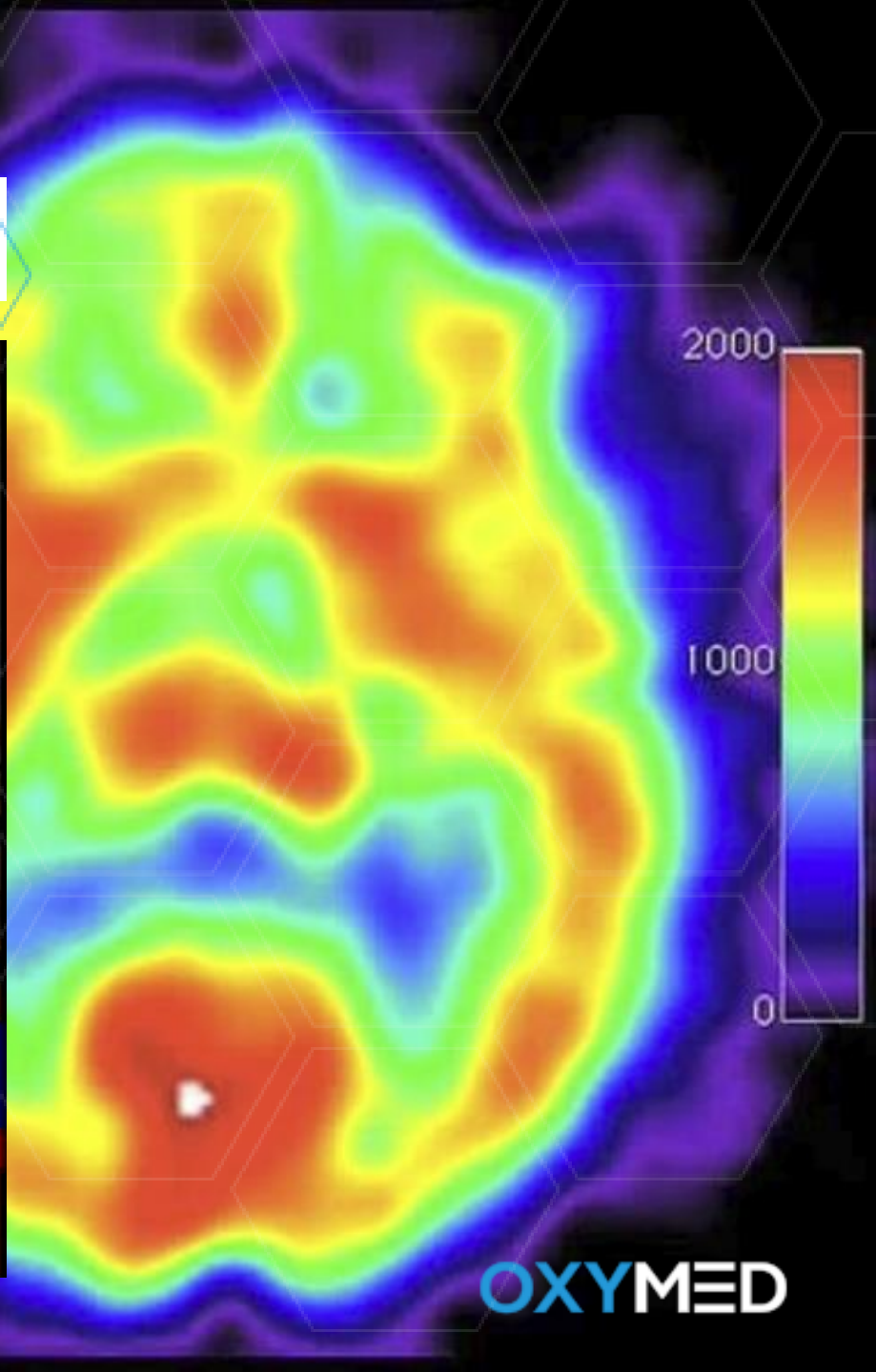
Case Study – Drowning Victim

Young Chloe – near drowning victim.

https://youtu.be/3XCL1mocf1c



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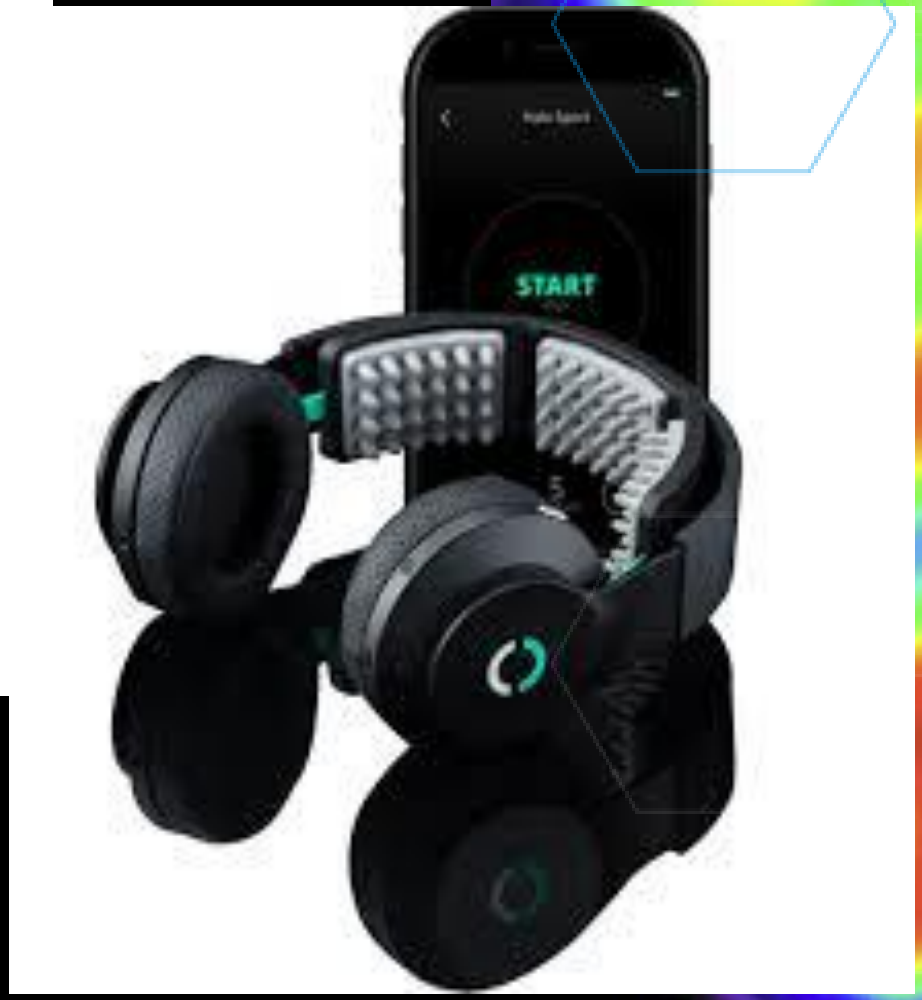
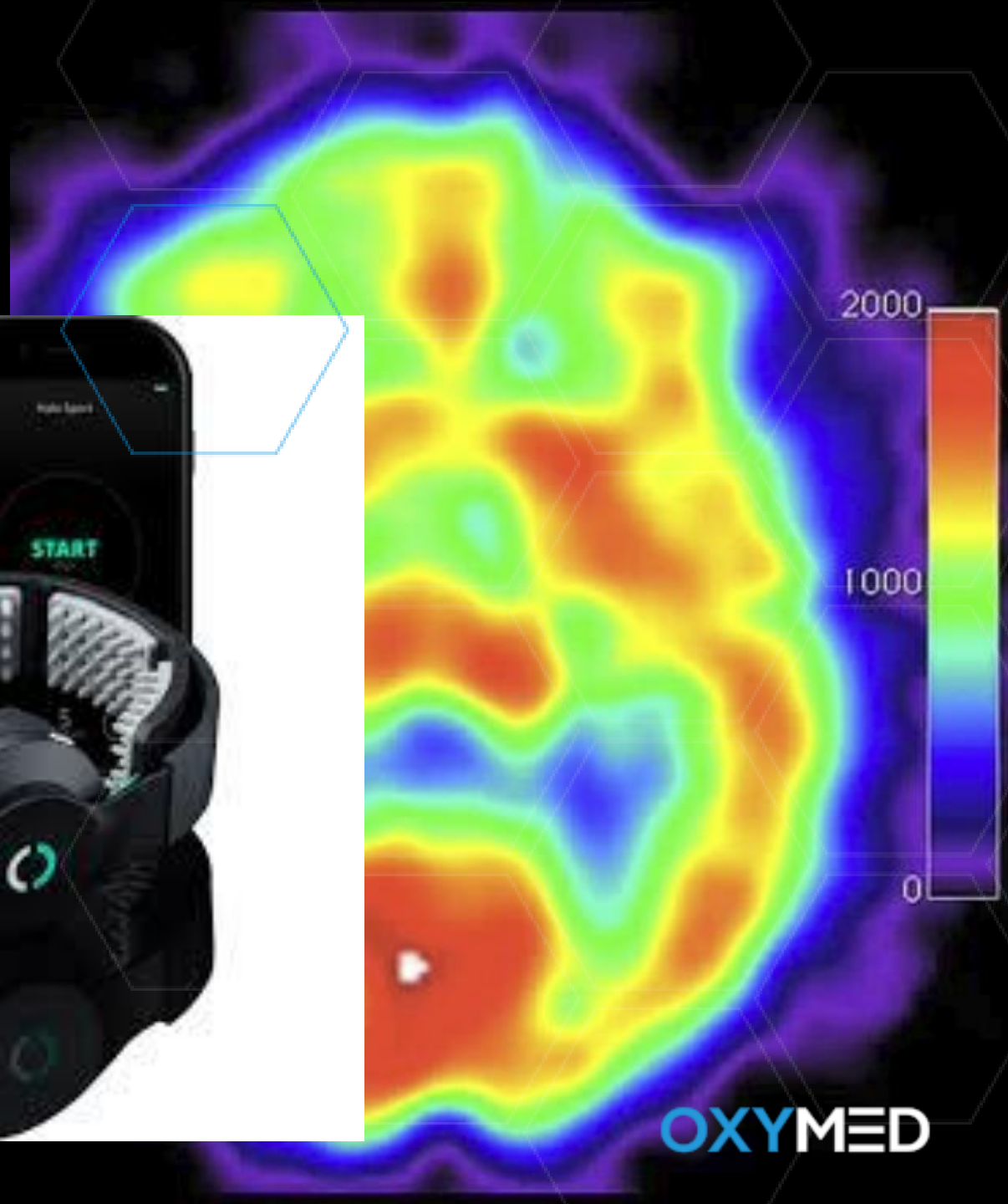
Neural Priming





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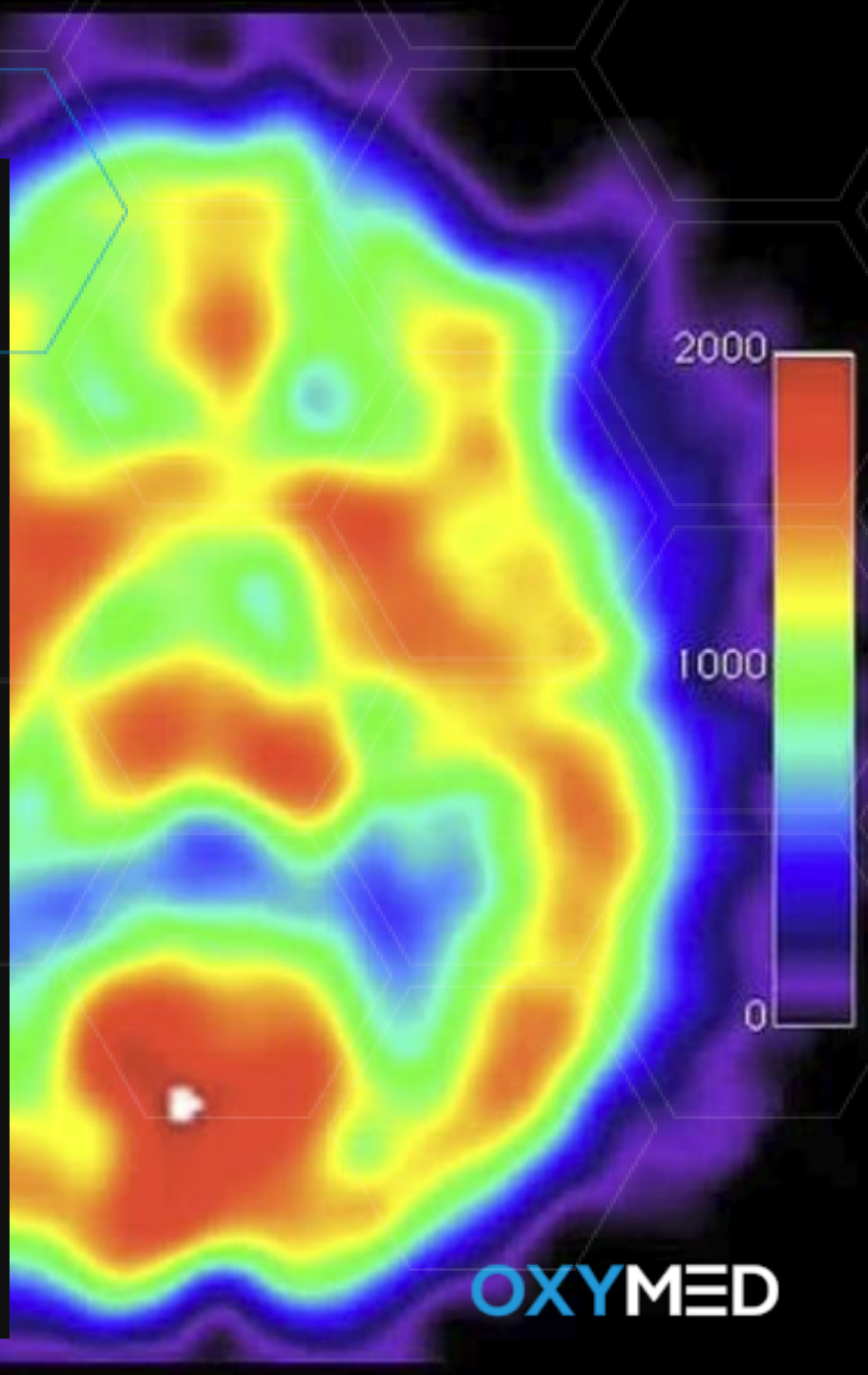
Neural Priming

Neuropriming is the process of using electrical stimulation during movement-based training to build stronger, more optimized connections between your brain and muscles.

This process induces a temporary state of hyper-learning or "hyperplasticity" in the brain, which refines the brain's ability to learn and adapt to training. This allows you to see better results, faster.



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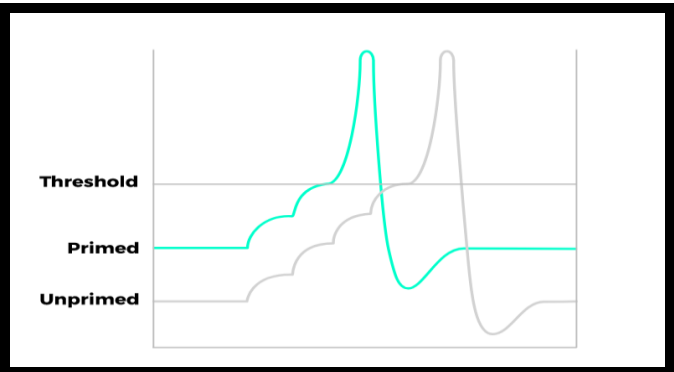
Athletes - Neural Priming

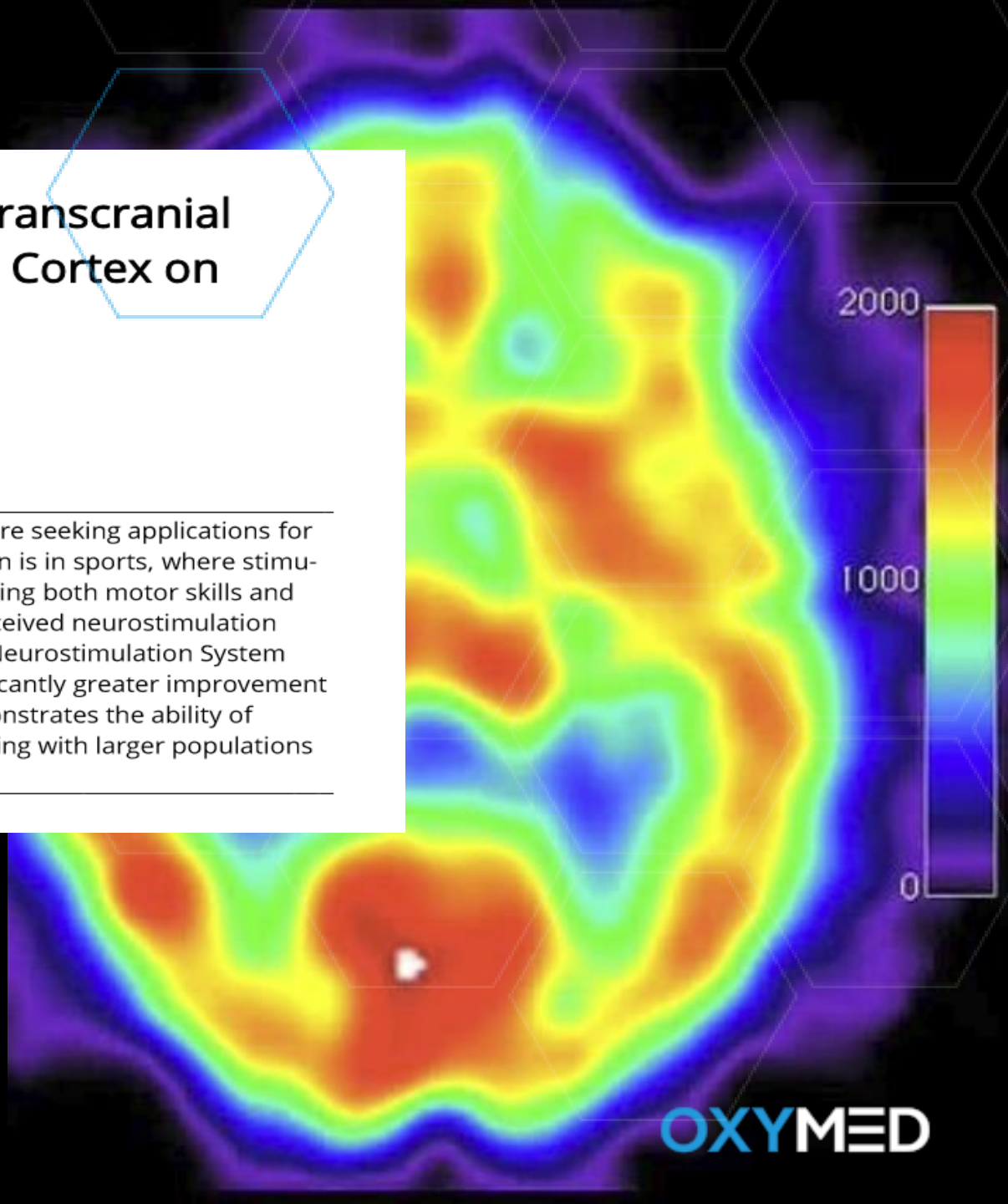
A Real-World Investigation into the Benefits of Transcranial Direct Current Stimulation to the Primary Motor Cortex on Muscular Performance in Elite Athletes

Halo Neuroscience

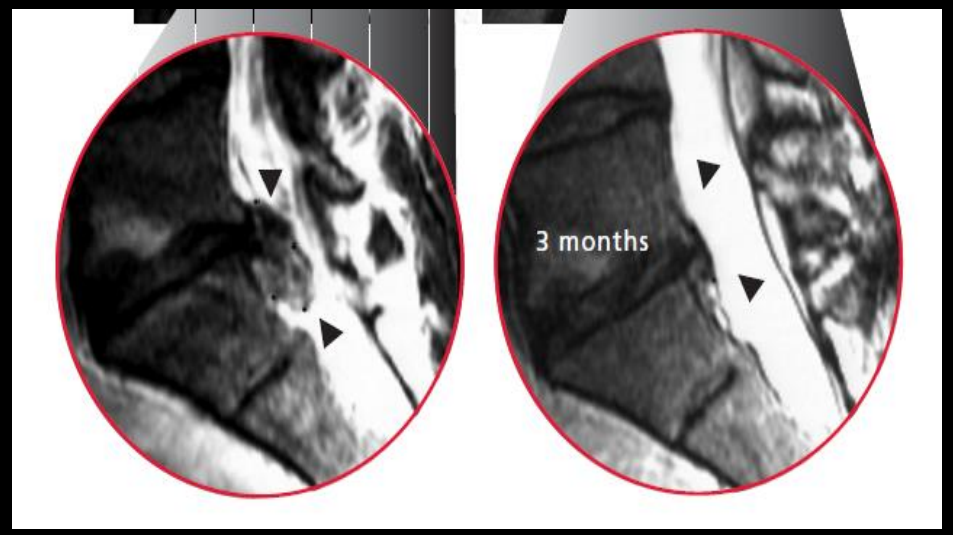
February 10, 2016

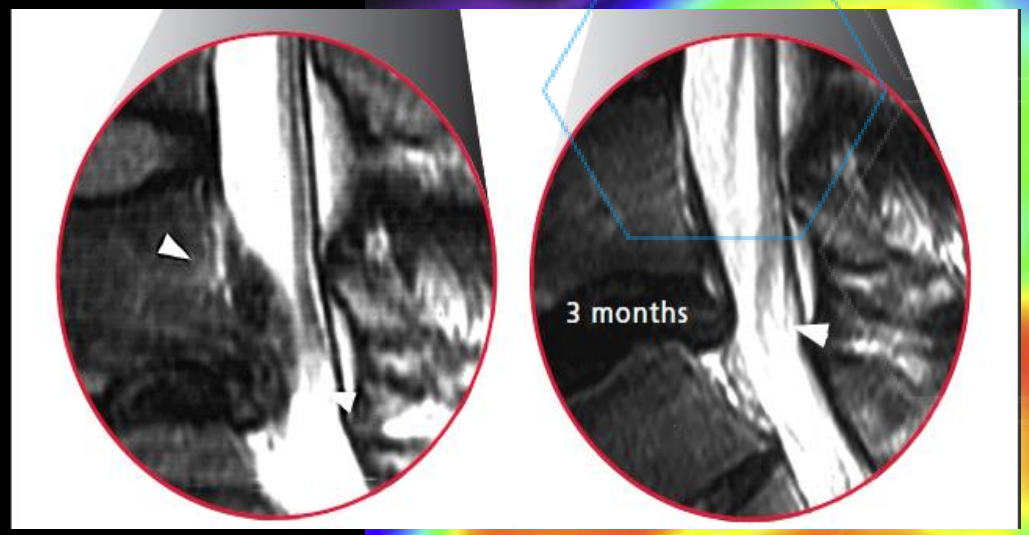
ABSTRACT: As interest in non-invasive brain stimulation grows, many potential users are seeking applications for the heightened learning state associated with this technology. One possible application is in sports, where stimulation has shown promising results in the form of increased training efficiency, improving both motor skills and raw power. In this study, athletes training for strength- and power-intensive sports received neurostimulation treatment in the form of transcranial direct current stimulation (tDCS) from the Halo Neurostimulation System during their normal training routine. Athletes who received stimulation showed significantly greater improvement in their jumping ability compared to non-stimulation athletes. The current study demonstrates the ability of non-invasive brain stimulation to improve athletic performance; however, further testing with larger populations and sham controls is needed in the future.

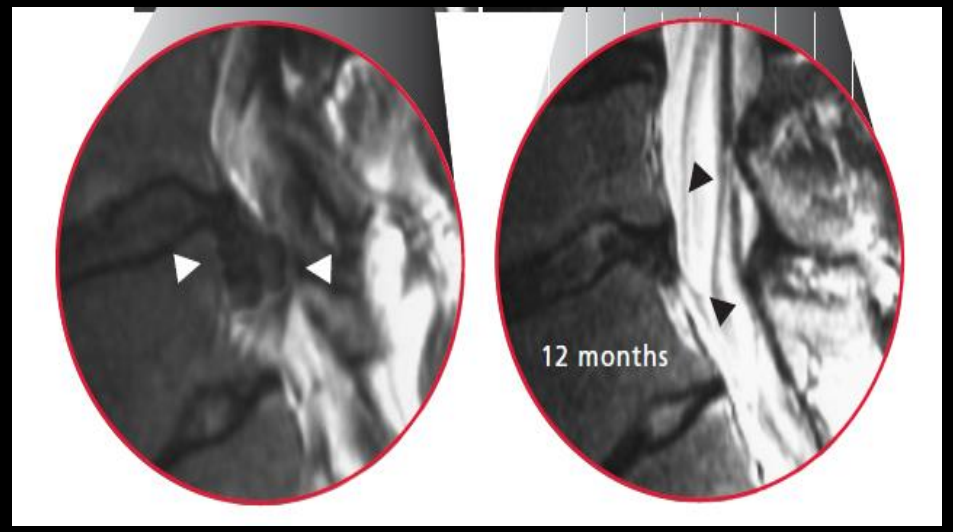


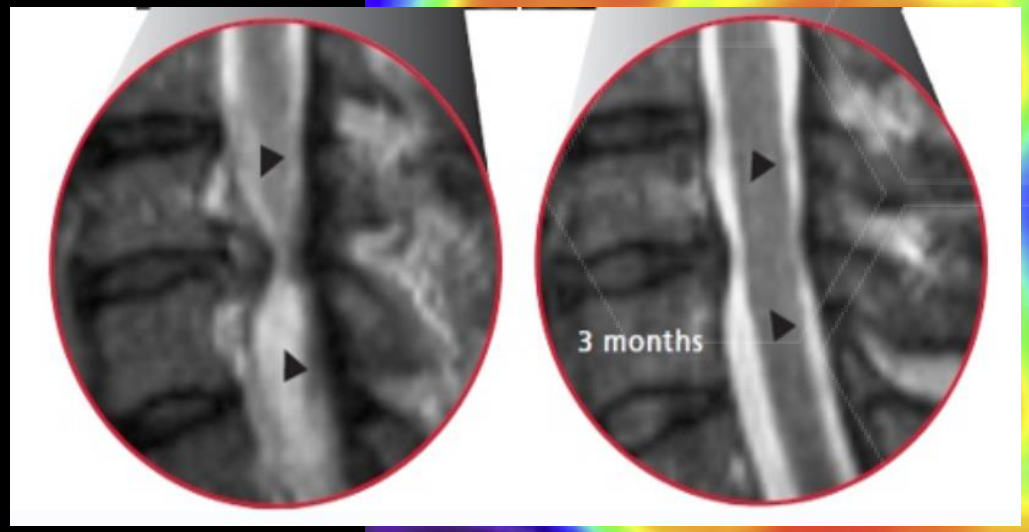


Chronic Pain, Disc Prolapse, Failed Back Surgery









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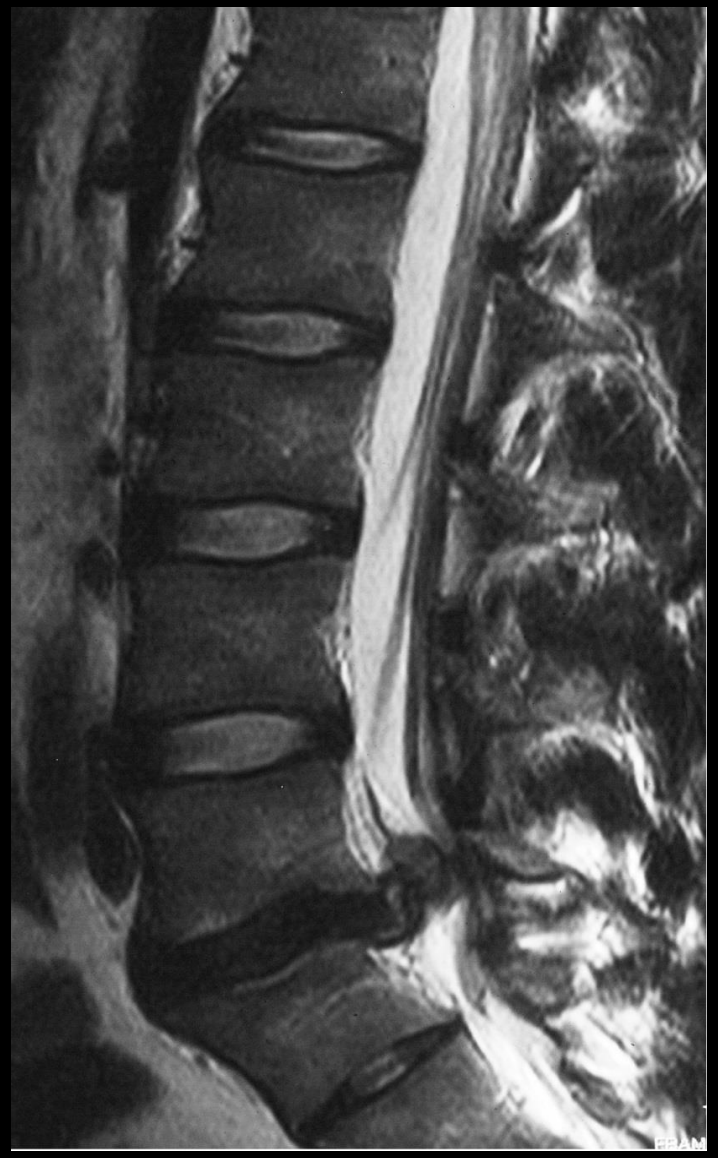
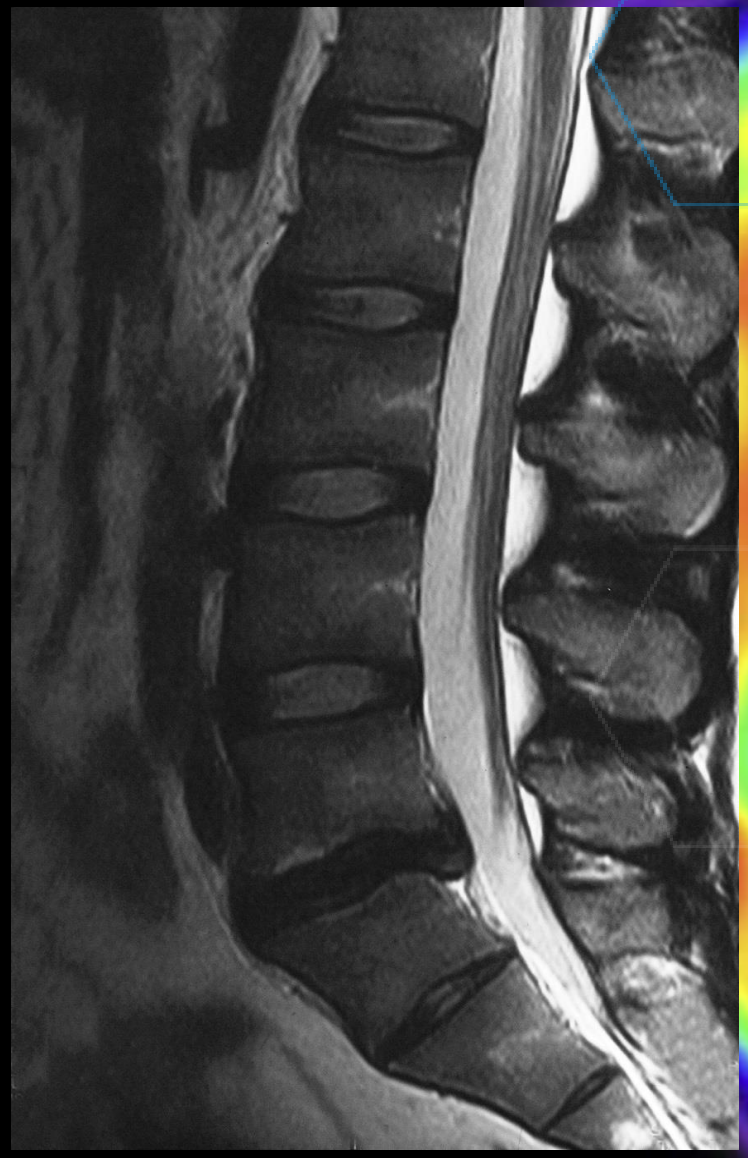
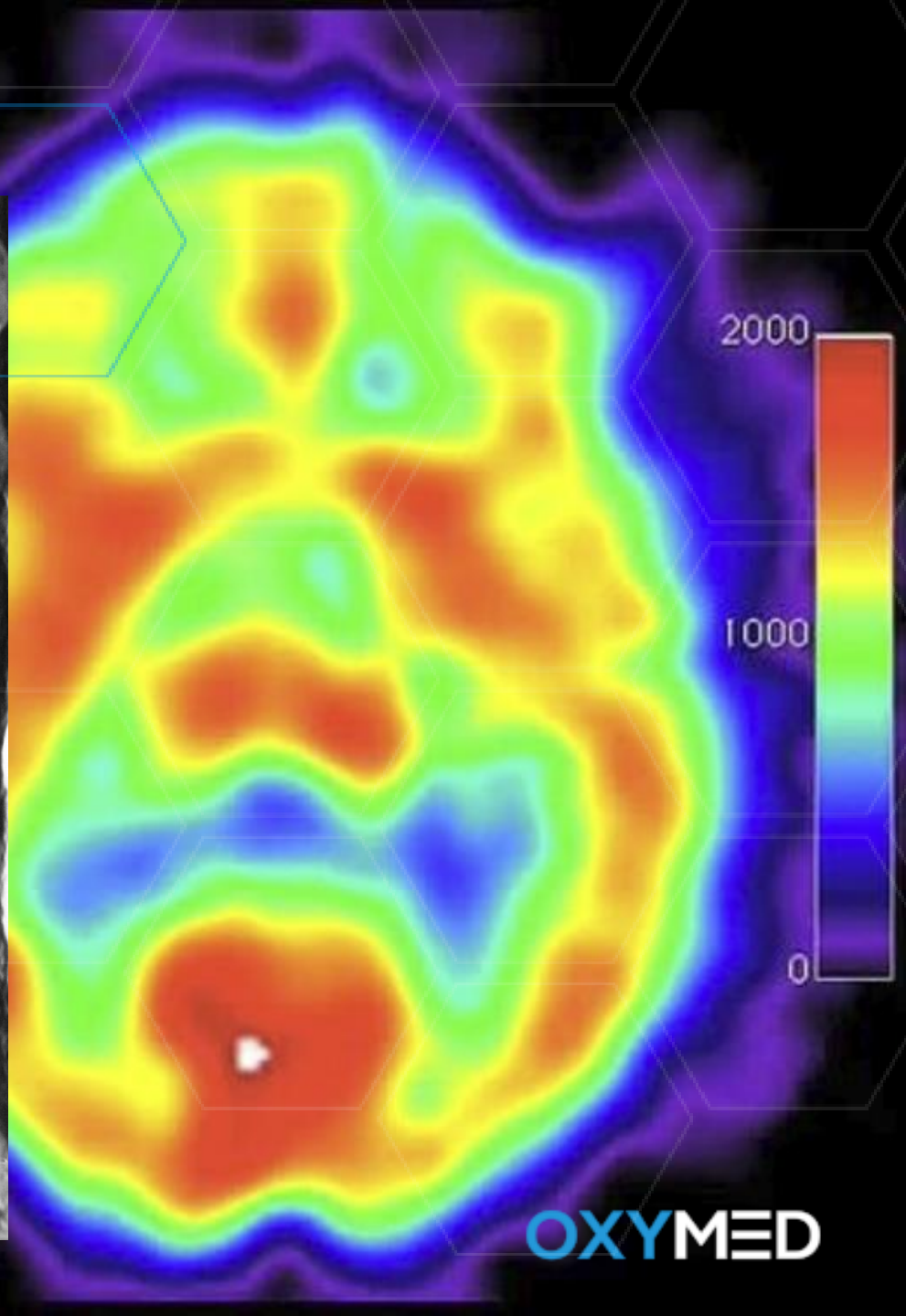
Disc Sequestration







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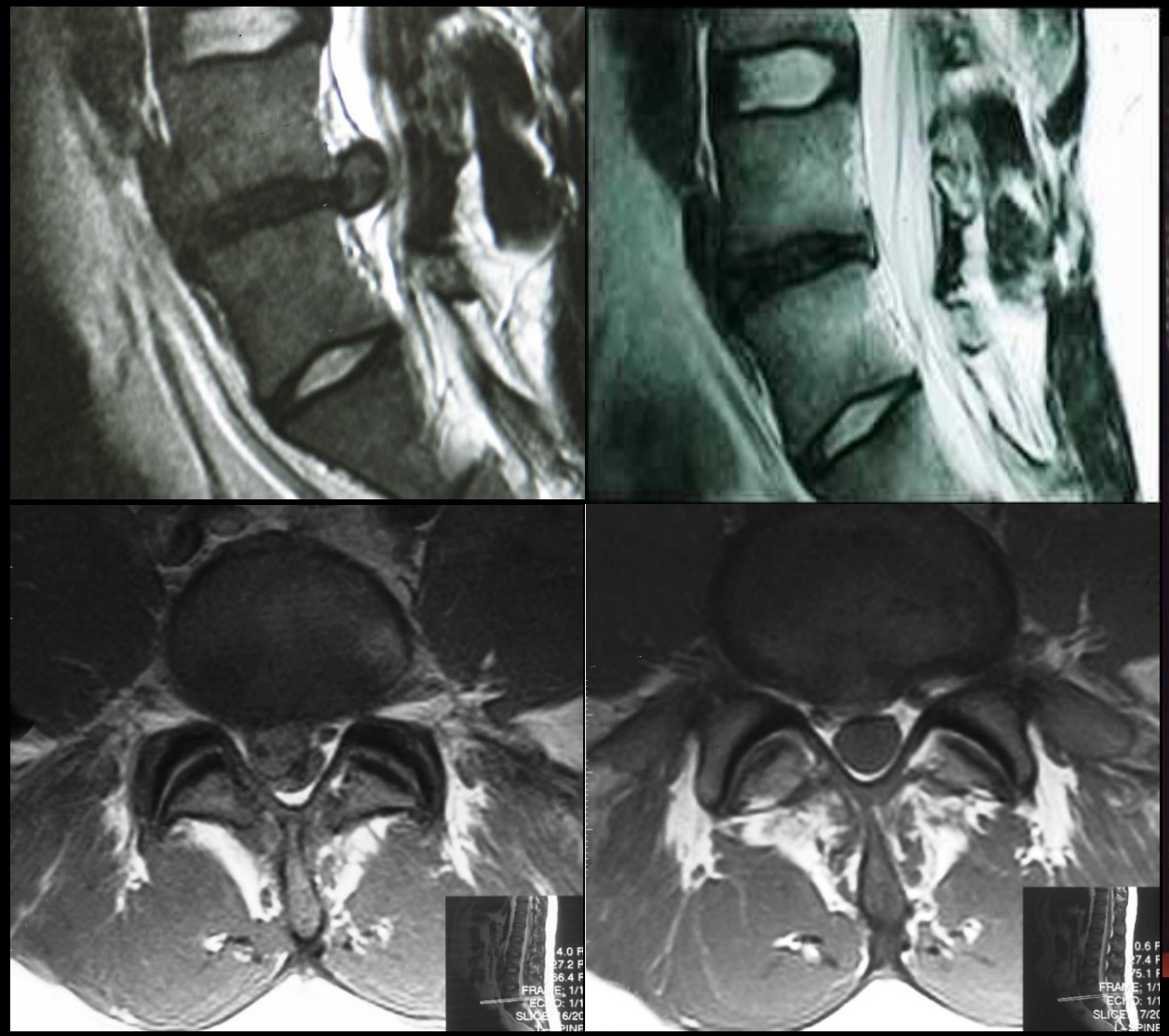
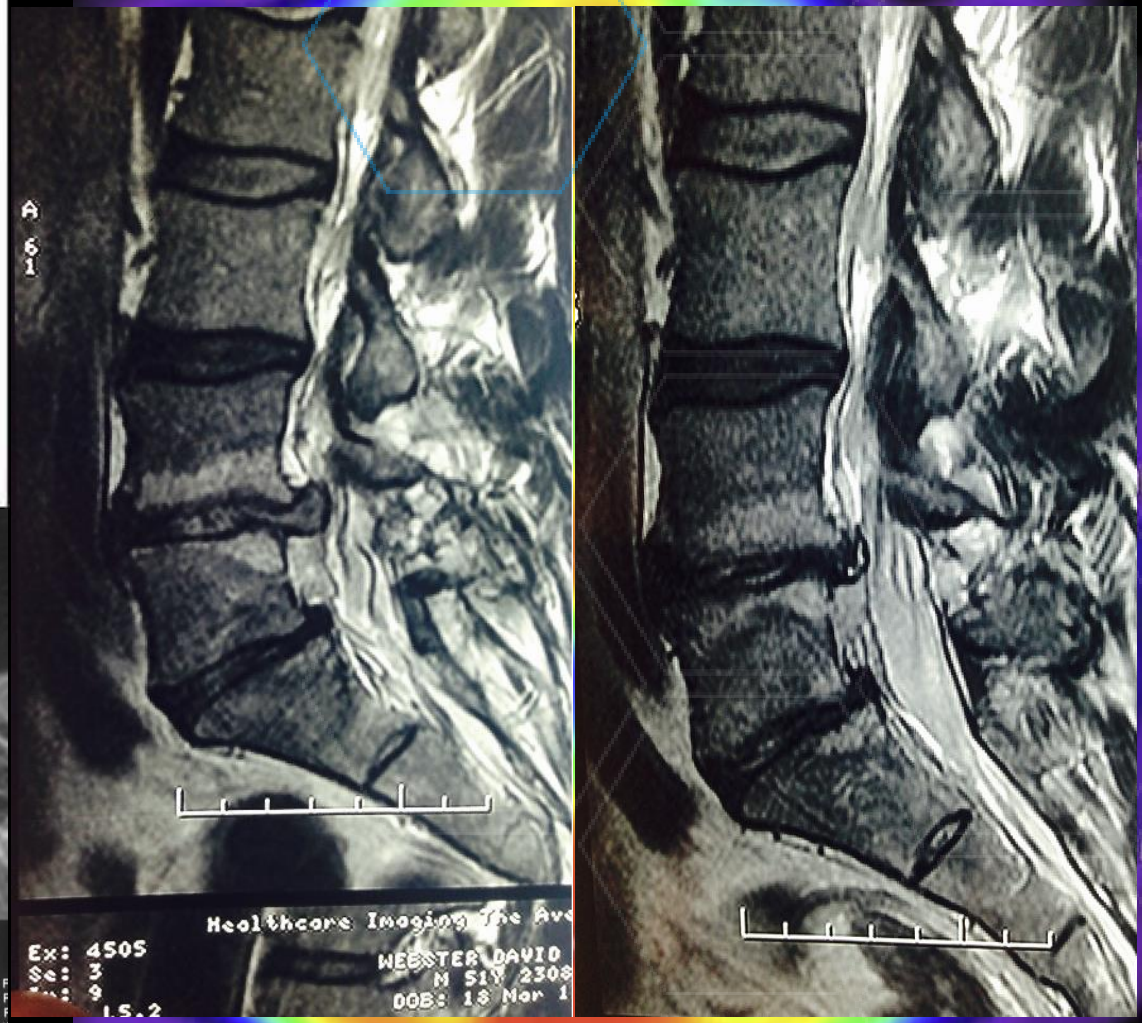
Disc Prolapse, Failed Back Surgery





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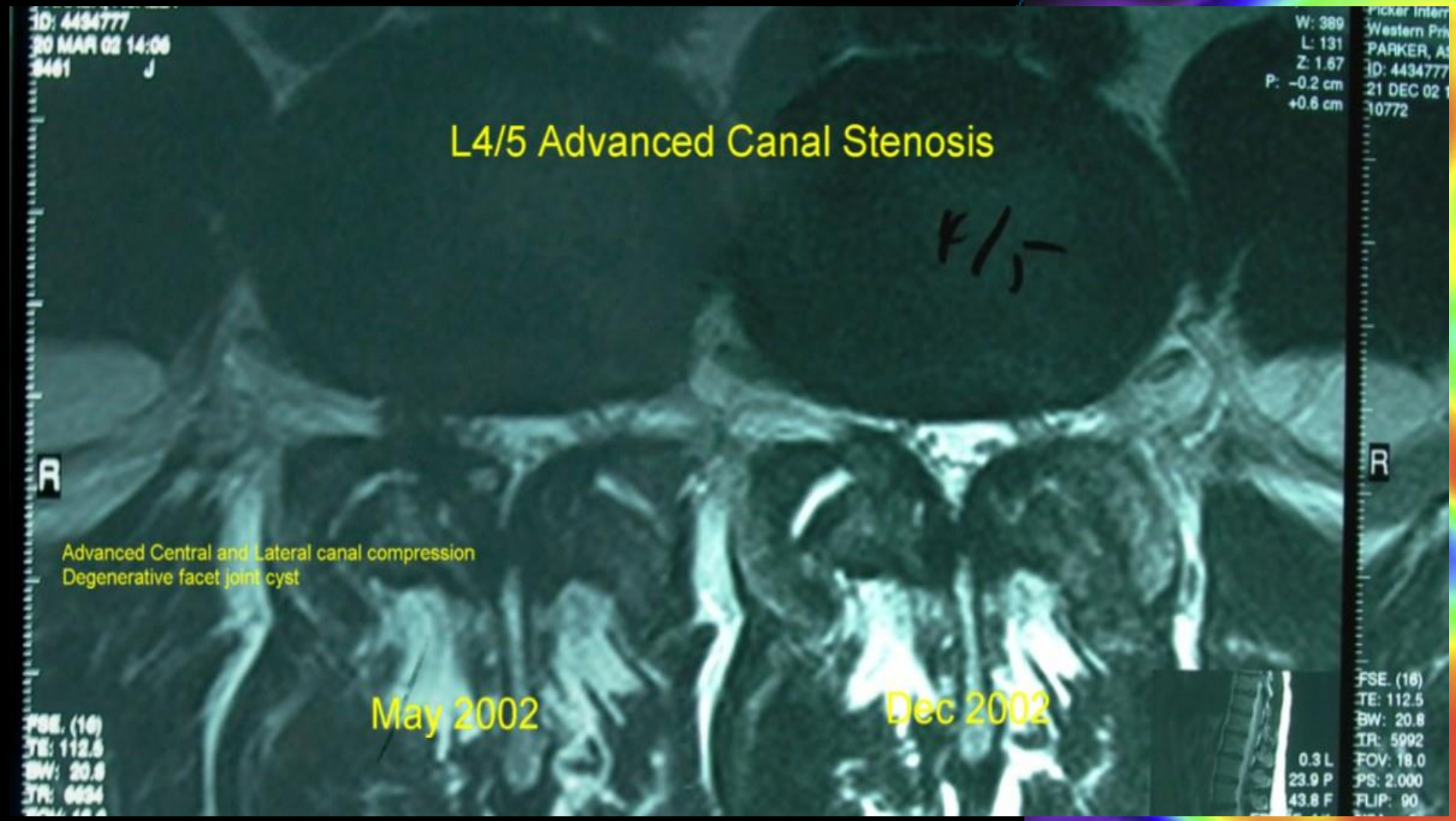
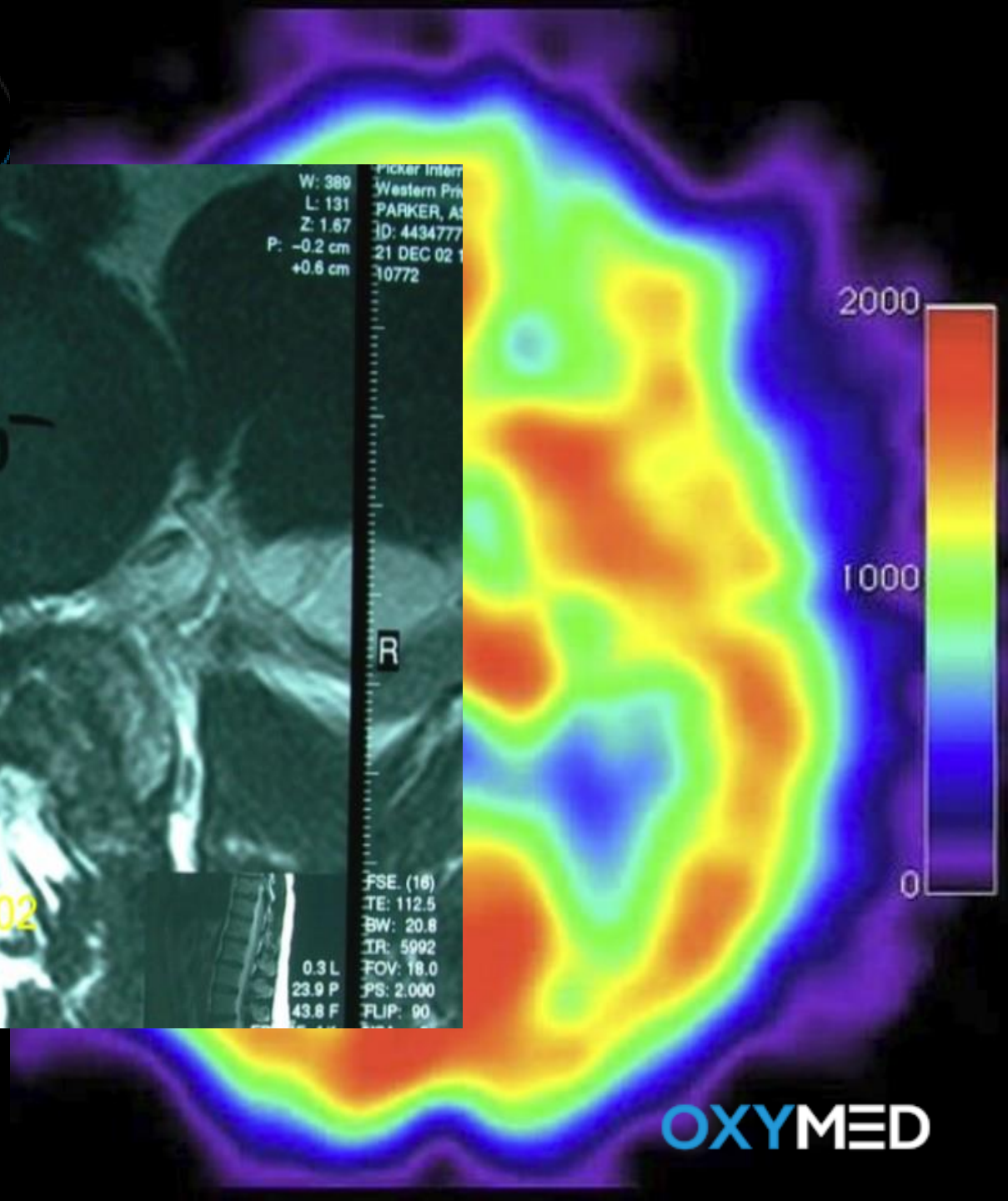
Degenerative Canal Stenosis





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OXYMED Heal the Warriors – OXYGEN100

HEAL THE WARRIORS

Traumatic Brain Injury (TBI) | Post-Concussion Syndrome (PCS)
Post-Traumatic Stress Disorder (PTSD)

Hyperbaric Oxygen Therapy - Fighting to treat the effects of TBI and PTSD

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MALCOLM HOOPER