

Sports Recovery & Performance – Hyperbaric Oxygen Therapy

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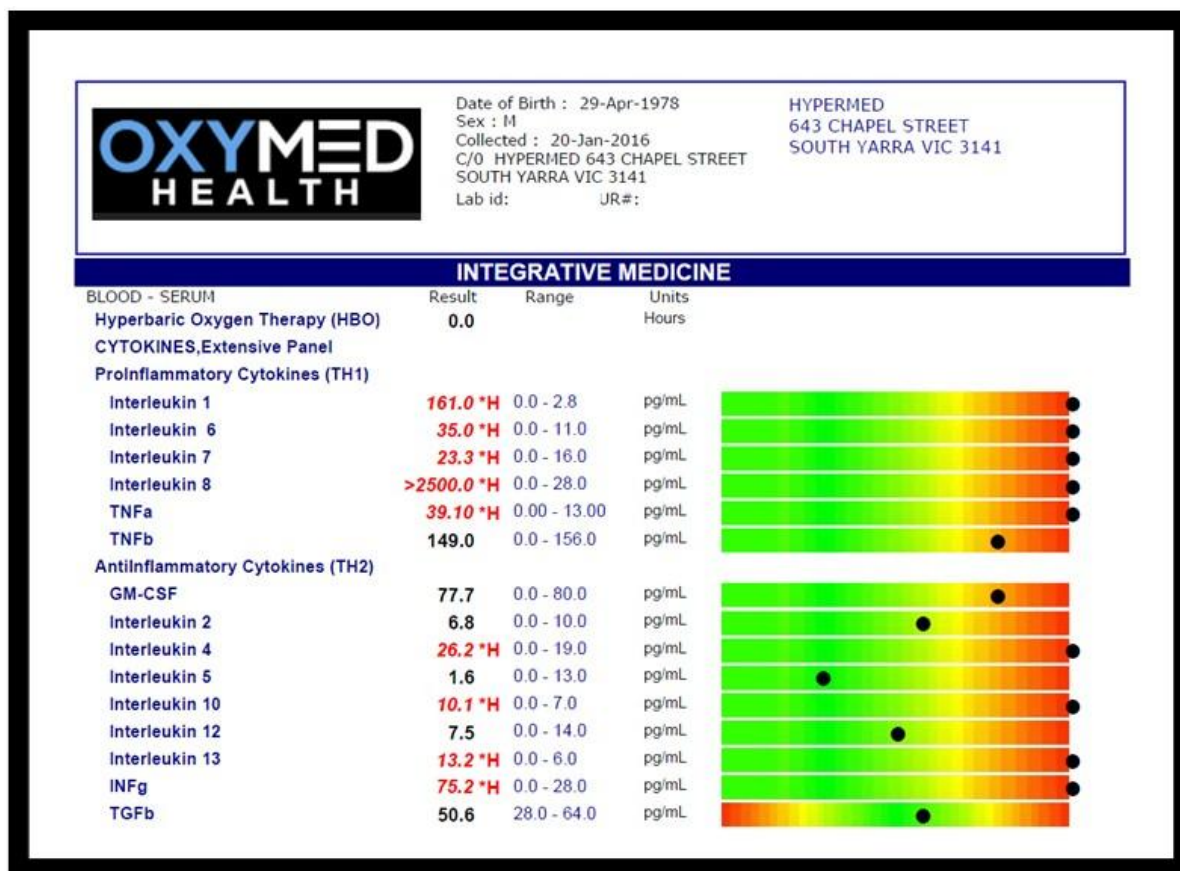
Sports Recovery & Performance

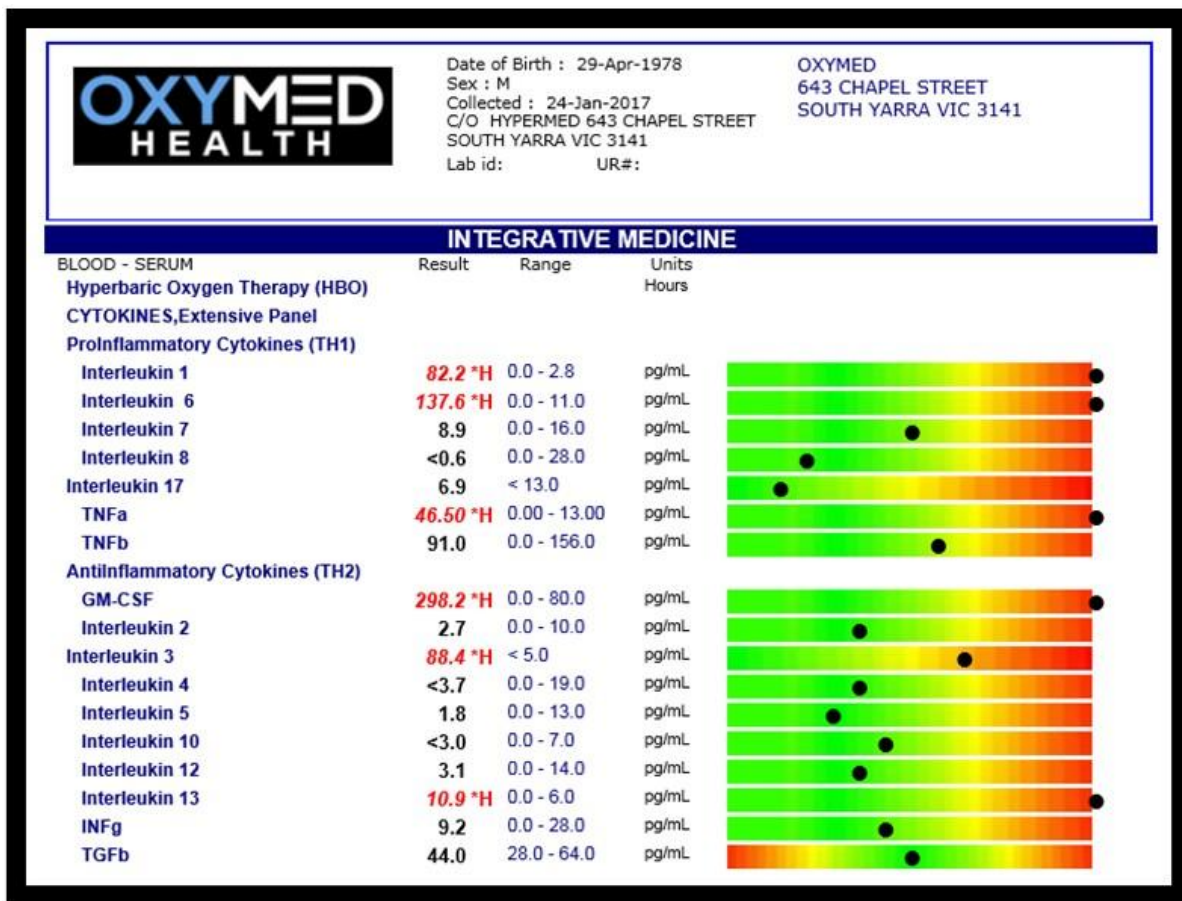
Elite athletes typically retire as a result of unresolved injury. The physical and mental toll of competing at the high-end level of sports culminates with the never-ending list of chronic injuries. Instead of enjoying their success, many athletes are invariably confronted with managing potentially serious health related challenges off the field of glory.

End of a Long Career

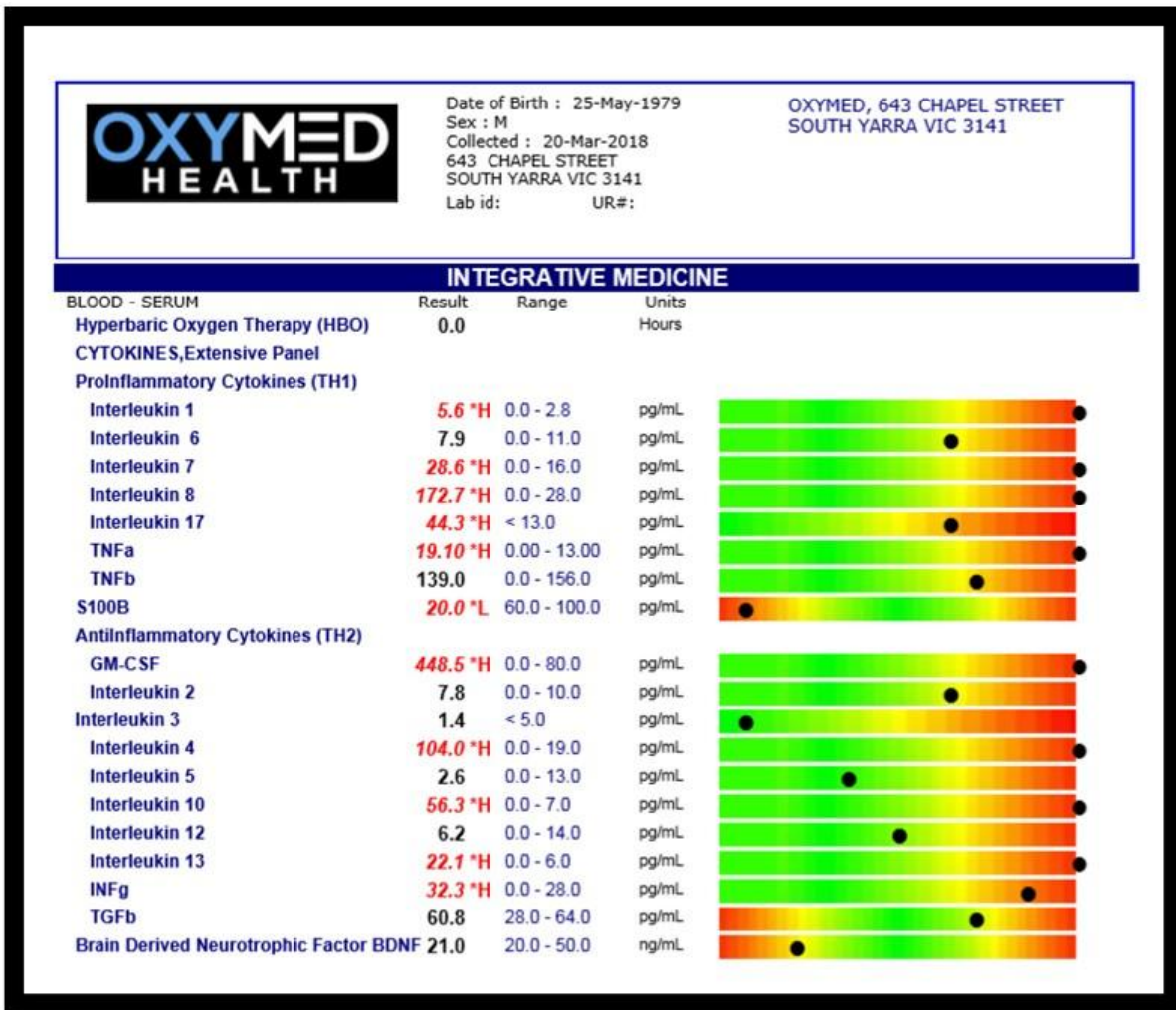
Performance, recovery, performance, recovery an endless cycle. Many elite athletes are in a chronic ‘cytokine storm’ – a prolonged phase of burn-out. International athletes are confronted with a range of metabolic challenges due to constant flights at altitude (reduced pressure and oxygen), time differences, chronic hypoxia, fatigue, lactic acid overload and sheer exhaustion. As tissue oxygenation diminishes (hypoxia) the secondary inflammatory changes and range of injuries increases dramatically. Tissues chronically exposed to hypoxia over express pro-inflammatory cytokine expressions. Over-expression of inflammatory cytokine gene clusters spread, potentiating autoimmune disorders [1].

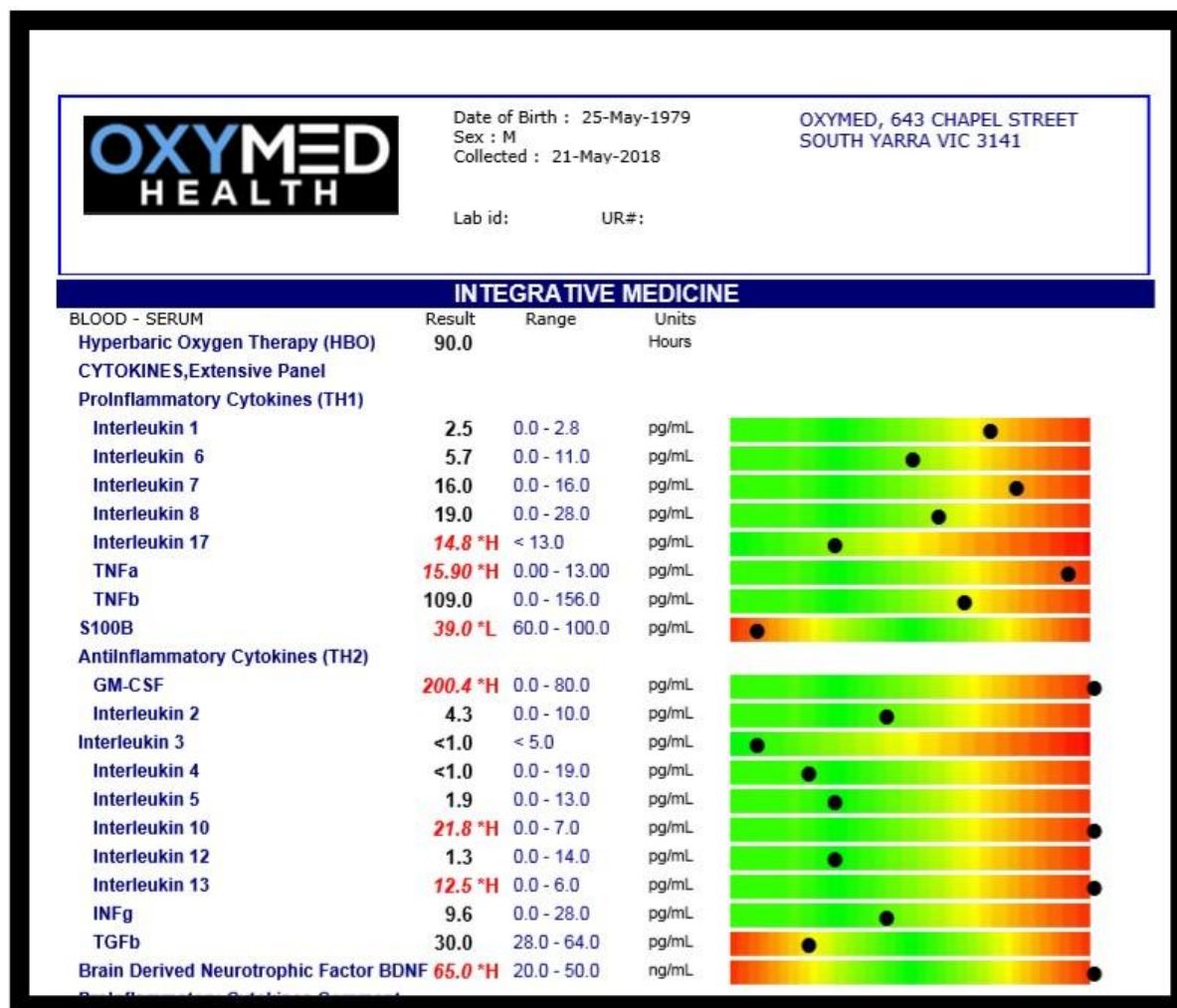
Case 1: Before & After





Case 2: Before & After





Cytokine Expressions

Cytokines are gene signal proteins and glycoproteins that ‘orchestrate’ proper immune responses including inflammation and anti-inflammatory pathways. Cytokines act as mediators and modulators and regulate immunological responses, hematopoietic development, and cell-to-cell communication as well as host responses to infectious agents and inflammatory stimuli. Cytokines are pleiotropic which refers to their ability to address multiple targets and physiological effects [1]. Cytokine production is tightly regulated, their homeostatic concentration in body fluids is low. However, if required, the concentration of cytokines can increase up to 1,000-fold [1]. In healthy individuals, cytokines are either not detectable or present at small concentrations in body fluid or tissues. Markers such as inflammatory cytokines are elevated after exercise in healthy individuals and return to baseline values within minutes to hours after exercise. Elevated concentrations of cytokines indicate activation of cytokine pathways associated with inflammation or disease progression. For this reason, cytokine measurements are both predictive and preventative to understand disease progression and monitor the effects of treatment.

Interleukin 1: (IL1) with IL6 and TNFα contribute to acute Cytokine Storm associated with COVID19 comorbidities [2]. IL1 is linked with systemic inflammation including ‘gut and brain connection’. Many athletes suffer with chronic irritable bowel syndrome (IBS). IL1 is linked in stroke with a reduction in the cerebral blood flow and increase in infarct volume [3]. Blockade of endothelin-1 receptors reversed this hypoperfusion, reduced tissue damage, and improved functional outcome. IL1 inhibits neurogenesis. IL1 is linked with synovial joint fluid degeneration and rheumatoid arthritis [4], IL1 is also linked in schizophrenia [5]. IL1 is linked in aggressive carcinoma progression including head and neck cancer, pancreatic cancer, thyroid cancer and bladder cancer [6].

Interleukin 6 (IL6): can increase up to a 1,000-fold during trauma and acute infection and is a major driver of COVID19 mortality [3]. IL6 is linked with chronic stress and may contribute to neurobehavioral complication [7]. IL6 is an important biomarker in monitoring inflammatory responses [8]. IL-6 is involved in the induction of acute phase responses and induction of fever [9]. IL6 is linked in chronic inflammatory arthritis and juvenile arthritis [10, 11]. Chronic elevation of serum IL-6 is associated with the progression of atherosclerosis in patients with vascular risk factors [11]. Elevated IL6 predicts cognitive decline and dementia [12]. IL6 is linked with chronic lumbar radicular pain. Persistent increase of IL1, IL6 and IL8 continues after disc herniation [9]. IL6 is linked with pathological pain associated with bone cancer, peripheral nerve injury, spinal cord injury, chemotherapy-induced peripheral neuropathy [8]. IL6 is a growth and survival factor in human glioblastoma cells and plays an important role in malignant progression.

Interleukin 8 (IL8): is linked with atherosclerosis and cerebrovascular disorders due to the micro-damage of the endothelial vascular wall [13]. IL8 is linked with activated microglia, as a primary source of neuroinflammation in concussion syndrome, head injury, alzheimers and other neurodegenerative diseases [14]. Hypoxia increased the release of IL8 which contributes to the aggressive spread of cancer cells [15].

Tumor Necrosis Factor Alpha: is a regulator of inflammatory responses. TNFα is linked with chronic pain syndromes, disc degeneration, failed surgery, arthritis, chronic tendinitis [16]. The hippocampus (role in learning and memory formation) is responsible for the growth and development of neurons (neurogenesis) after injury. TNFα is upregulated in the hippocampus and associated with neuropathic pain syndromes. TNFα suppresses brain-derived neurotrophic factor (BDNF) [17]. TNFα is linked with depression, neurodegeneration and maladaptive neuroplasticity.

S100B: is a marker of brain damage and BBB function. S100B is linked with depression disorders including dementia, alzheimers, stroke, traumatic brain injury, concussion, post traumatic stress disorders. S100B is associated with neural inflammation anywhere throughout the body [18, 19]. S100B is elevated in returning War Veterans suffering the effects of PTSD, TBI, and shock blast injuries. S100B elevation is associated with repeated hypobaric (long haul flights) hypoxic exposure resulting in blood-brain barrier dysfunction [20].

Hyperbaric Oxygen Therapy

HBOT is the application of two variables - increased pressure and increased oxygen. HBOT has profound effects on immune modulation in the presence of hypoxia. Mitochondrial respiration is the key to an athlete's recovery and performance. HBOT studies demonstrate improvement in cerebral hypo perfusion, neuroinflammation, gastrointestinal inflammation, immune dysregulation, oxidative stress, mitochondrial dysfunction and neurotransmitter abnormalities [21]. HBOT is typically the focus of injury repair and part of an active rehabilitation program. All professional athletes and weekend warriors carry a growing injury list. Personalised protocols based on cytokine testing empower both predictive and preventative strategies. Off season strategies employing HBOT will best prepare an athlete and team. HBOT pre-conditioning off season, pre and post-game will diminish the window intersecting peak performance and the risk of injury and recurrence. HBOT increases the plasma saturation of oxygen. Normal plasma oxygen by volume is 3% (0.3 ml O₂/100 ml blood), under HBOT conditions the O₂ saturation increases 10-20 times that of breathing room air at normal atmospheric pressure [22]. HBOT increases the production and circulation of progenitor stem cells specific to the individual [23-25]. Oxygen saturated plasma under pressure ‘soaks the tissue’. HBOT saturation into the central nervous system promotes the brain, spinal cord and peripheral nerves to better regulate biochemical reactions [26]. HBOT promotes rapid recovery from soft tissue injuries. HBOT increased the number of proliferating and differentiating satellite cells, and the amount of regenerated muscle fibers [27]. HBOT inhibits chronic circulating

infections in the blood plasma [28]. HBOT impacts underlying gut issues and sensitivities. All athletes suffer exposure to air borne pathogens, hypobaric hypoxic international flights and crowded environments [20]. HBOT has strong anti-inflammatory properties decreasing the pro-inflammatory cascade including IL1, IL6, IL7, IL8, TNF α , and S100B. HBOT has been shown to increase the counter-inflammatory IL4, IL10 and IL13 levels [22]. HBOT elevates IL10 and Brain Derived Neurotrophic Factor (BDNF) required for repetition skill neuroplasticity [18].

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