THE INTERNATIONAL HYPERBARICS ASSOCIATION, INC.

The Pressure Point

Volume 8

Issue 12

December 2007

Global Toy Concern

Chinese Santa and His Elves

Growing concerns about lead, chemical toxicity and other harmful effects to children this Christmas

Editorial: For some time now it has become common knowledge that anything with small components, electronics and especially children's toys have become tainted with lead, chemical toxins and hazardous drugs and material.

The recent headlines in the papers have been spelling out the various harms that await purchasers of children's play jewelry, crafting items (i.e. aquadots), Mattel toys—lead poisoning, coma, death.

These toys all share one common gene—China.

Dora

Now, it is true that we live in a global market, and that trade with various countries encourages neighborly accord—even with a country willing to possibly nuke us! It is the neighborly thing to do.

How has it come to this—China taking over Christmas—and most every other day in our lives, for that matter?

In one word: OVERREGULATION. At face value this might seem to be the opposite of the problem, meaning that if we regulated Chinese toys better, the bad ones would not be getting in, right? Yes, in a way.

Our dependence on Chinese goods has been caused by over-regulation of companies here in the United States. Yes, this statement will make for interesting political talk, but it still stands that our children are the ones holding the bag of toxic Chinese goods.

Like everything else in America, we have outsourced the manufacture of most everything because the costs of manufacturing those things IN America have become exorbitant. Those costs include labor,

(Continued on page 4)

Inside This Issue

Gastro News

J Gastroenterol Hepatol. 2007 Nov; 22 (11):2042-6
17914992

Hyperbaric oxygen therapy (HBOT) for severe acute pancreatitis

Christopher Christophi , Ian Millar, Mehrdad Nikfarjam, Vijayaragavan Muralidharan, Caterina Malcontenti-Wilson

Despite improvements in the supportive management of severe acute pancreatitis over the last decade, the morbidity and mortality rate remains high. The main feature of this condition is pancreatic necrosis leading to sepsis, with both localized and systemic inflammatory response syndromes.

Early pathophysiological changes of the pancreas include alterations in microcirculation, ischemia reperfusion injury, and leukocyte and cytokine activation. The efficacy of hyperbaric oxygen (HBO) therapy in improving these pathophysiological disturbances is documented for various conditions. However, its effect in the treatment of severe acute pancreatitis is undetermined.

This report documents the case of a 56-year-old woman presenting with severe acute pancreatitis treated by HBO therapy. The severity of disease was based on an Acute Physiology and Chronic Health Evaluation (APACHE II) illness grading score of 11 and a Baltazar based computed tomography severity

"HBO therapy for severe acute pancreatitis appeared to be safe and may have a role in improving treatment outcomes."

(Continued on page 4)

Today.com

Inflammation is cause of insulin resistance

Researchers at the University of California, San Diego (UCSD) School of Medicine have discovered that inflammation provoked by immune cells called macrophages leads to insulin resistance and Type 2 diabetes.

Their discovery may pave the way to novel drug development to fight the epidemic of Type 2 diabetes associated with obesity, the most prevalent metabolic disease worldwide.

In recent years, it has been theorized that chronic, low-grade tissue inflammation related to obesity contributes to insulin resistance, the major cause of Type 2 diabetes. In research done in mouse models, the UCSD scientists proved that, by disabling the macrophage inflammatory pathway, insulin resistance and the resultant Type 2 diabetes can be prevented.

"The UCSD research team showed that the macrophage is the cause of this cascade of events by knocking out a key component of the inflammatory path way in the macrophage."

The findings of the research team, led by principle investigators Michael Karin, Ph.D., Professor of Pharmacology in UCSD's Laboratory of Gene Regulation and Signal Transduction, and Jerrold Olefsky, Distinguished Professor of Medicine and Associate Dean for Scientific Affairs, will be published as the feature article of the November 7 issue of *Cell Metabolism.*

"Our research shows that insulin resistance can be disassociated from the increase in body fat associated with obesity," said Olefsky.

Macrophages, found in white blood cells in the bone marrow, are key players in the immune response. When these immune cells get into tissues, such as adipose (fat) or liver tissue, they release cytokines, which are chemical messenger molecules used by immune and nerve cells to communicate. These cytokines cause the neighboring liver, muscle or fat cells to become insulin resistant, which in turn can lead to Type 2 diabetes.

The UCSD research team showed that the macrophage is the cause of this cascade of events by knocking out a key component of the inflammatory pathway in the macrophage, JNK1, in a mouse model. This was done through a procedure called adoptive bone marrow transfer, which resulted in the knockout of JNK1 in cells derived from the bone marrow, including macrophages.

With this procedure, bone marrow was transplanted from a global JNK1 knockout mouse (lacking JNK1 in all cell types) into a normal mouse that had been irradiated to kill off its endogenous bone marrow. This resulted in a chimeric mouse in which all tissues were normal except the bone marrow, which is where macrophages originate. As a control, the scientists used normal, wild-type mice as well as mice lacking JNK1 in all cell types. These control mice were also subjected to irradiation and bone marrow transfer.

The mice were all fed a high-fat diet. In regular, wild-type mice, this diet would normally result in obesity, leading to inflammation, insulin resistance and mild Type 2 diabetes. The chimeric mice, lacking JNK1 in bone marrow-derived cells, did become obese; however, they showed a striking absence of insulin resistance – a pre-condition that can lead to development of Type 2 diabetes.

"If we can block or disarm this macrophage inflammatory pathway in humans, we could interrupt the cascade that leads to insulin resistance and diabetes," said Olefsky. "A small molecule compound to block JNK1 could prove a potent insulin-sensitizing, anti-diabetic agent."

The research also proved that obesity without inflammation does not result in insulin resistance. Olefsky explained that when an animal or a human being becomes obese, they develop steatosis, or increased fat in the liver. The steatosis leads to liver inflammation and hepatic insulin resistance.

The chimeric mice did develop fatty livers, but not inflammation. "Their livers remained normal in terms of insulin sensitivity," said Olefsky, adding that this shows that insulin resistance can also be disassociated from fatty liver.

"We aren't suggesting that obesity is healthy, but indications are promising that, by blocking the macrophage pathway, scientists may find a way to prevent the Type 2 diabetes now linked to obesity and fatty livers," Olefsky said.-University of California - San Diego

Jingle all the way. . .
Ha Ha Ha

The Inflammatory Response

Research Communication

Exhaled Nitric Oxide is Decreased by Exposure to the Hyperbaric Oxygen Therapy Environment

Mediators of Inflammation
Volume 2006 (2006), Article ID 72620, 6 pages
doi:10.1155/MI/2006/72620

Zudin A. Puthucheary,[1] Jia Liu,1 Michael Bennett,2 Barbara Trytko,[2] Sharron Chow,[1] and Paul S. Thomas[1]

[1]UNSW and Department of Respiratory Medicine, Faculty of Medicine, Prince of Wales Hospital, Randwick 2031, NSW, Australia

[2]Hyperbaric Unit, Prince of Wales Hospital, Randwick 2031, NSW, Australia

Received 30 May 2006; Revised 24 August 2006; Accepted 25 August 2006

ABSTRACT

Exhaled nitric oxide (eNO) detects airway inflammation. Hyperbaric oxygen therapy (HBOT) is used for tissue hypoxia, but can cause lung damage. We measured eNO following inhalation of oxygen at different tensions and pressures.

Methods. *Part 1*, eNO was measured before and after HBOT. All patients and attendants provided an expired NO sample prior to compression, the patients then underwent a standard 90-minute HBOT session at 2.4 atmospheres absolute (ATA), breathing 100% O2. These sessions involve a five to ten minute compression phase breathing air, 90 minutes breathing 100% oxygen at 2.4 ATA by demand valve mask or hood, and a further 20 minutes slow decompression while continuing to breathe oxygen. The total time in the chamber for each session is therefore approximately two hours.

Immediately on exiting the hyperbaric chamber, a further exhaled NO sample was taken from patients and staff.

Part 2, normal subjects breathed 40% oxygen. A separate section studied normal subjects breathing 40% oxygen for 90 minutes via a face mask at 1 ATA. Exhaled NO samples were collected every 15 minutes.

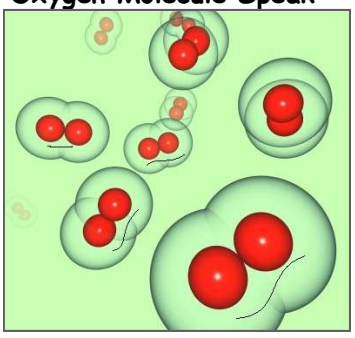
Results. Baseline eNO levels in patients prior to HBOT exposure were significantly higher than in normal subjects ($P < .05$).

After HBOT, eNO significantly decreased in patients (15.4 ± 2.0 versus 4.4 ± 0.5 ppb, $P < .001$), but not in normal subjects, after either 100% O2 at increased pressure or 40% oxygen, 1 ATA. In an in vitro study, nitrate/nitrite release decreased after 90 minutes HBOT in airway epithelial (A549) cells.

Conclusion. HBO exposure causes a fall in eNO. Inducible nitric oxide synthase (iNOS) may cause elevated eNO in patients secondary to inflammation, and inhibition of iNOS may be the mechanism of the reduction of eNO seen with HBOT.

This might have beneficial effects on inflammation, a question which could to be further investigated by measurement of iNOS expression in a randomized controlled study in response to hyperoxia.

Oxygen Molecule Speak



"With hyperbarics we're under so much pressure to perform."

Archives: Brainres.2006

Hyperbaric oxygen treatment decreases inflammation and mechanical hypersensitivity in an animal model of inflammatory pain

Hilary D. Wilson[a], Judy R. Wilson[b] and Perry N. Fuchs[c],

[a]University of Texas at Arlington, Department of Psychology, 501 S. Nedderman Drive, Arlington, TX 76019, USA

[b]Department of Kinesiology, University of Texas at Arlington, TX 76019, USA

[c]Departments of Psychology, Biology, and Converging Biotechnology Center, University of Texas at Arlington, 501 S. Nedderman Drive, Arlington, TX 76019, USA

Revised 26 April 2006. Available online 5 June 2006.

Abstract

Hyperbaric oxygen therapy has been used to treat a variety of ailments from carbon monoxide poisoning to fibromyalgia.

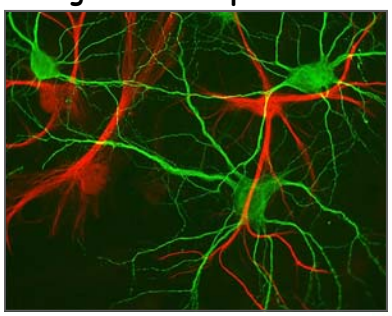
The purpose of this experiment was to explore the effect of hyperbaric oxygen treatment on carrageenan-induced inflammation and pain in rats.

Hyperbaric oxygen treatment significantly decreased inflammation and pain following carrageenan injection.

Clinically hyperbaric oxygen may be used in situations where NSAIDS are contraindicated or in persistent cases of inflammation.

Keywords: Analgesia; Antinociception; Carrageenan; Inflammation; Nociception

Idling Neurons Speak



"Oh synapse! I did it again!"

THE INTERNATIONAL HYPERBARICS ASSOCIATION, INC.

"Mundo vitam dare"

15810 East Gale Avenue #178
Hacienda Heights, CA 91745

Toll free: 877.IHA..USA1
(877.442.8721)

pressurepoint.newsletter@ihausa.org
hyperbarics@ihausa.org

The International Hyperbarics Association, Inc., is a coalition of doctors, parents, patients, corporate chamber-industry professionals, hyperbaric center owners, and above all members who are committed to the cause of medical hyperbarics.

Our members come to us from all geographical areas with one common goal— to share their knowledge regarding the latest hyperbaric news. Our driving force is our members, who are committed to do all we can:

"to give life to the world."
— "Mundo vitam dare"

Gastro News (cont)

index (CTSI) score of 9.

Administration of 100% oxygen was commenced within 72 h of presentation at a pressure of 2.5 atmospheres for 90 min and given twice daily for a total of 5 days. Therapy was well tolerated with improvements in APACHE II and CTSI grading scores.

HBO therapy for severe acute pancreatitis appeared to be safe and may have a role in improving treatment outcomes. Further study is required.

Journal of Gastrointestinal Surgery vol11: no 8
1091-255X (Print) 1873-4626 (Online); 08 2007
DOI10.1007/s11605-007-0175-2; Pp 1008-1015

Hyperbaric Oxygen Therapy Reduces Severity and Improves Survival in Severe Acute Pancreatitis

Mehrdad Nikfarjam[1], Christine M Cuthbertson[1], Caterina Malcontenti-Wilson[1], Vijayaragavan Muralidharan[1], Ian Millar[2], and Christopher Christophi[1]

[1]Department of Surgery, University of Melbourne, Austin Hospital, Lance Townsend Building Level 8, Studley Rd, Heidelberg, Melbourne, Victoria, 3084, Australia

[2]Hyperbaric Unit, Alfred Hospital, Melbourne, Victoria, AustraliaPublished online: 22 May 2007

Abstract Severe acute pancreatitis is characterized by pancreatic necrosis, resulting in local and systemic inflammation. Hyperbaric oxygen (HBO) therapy modulates inflammation, but has not been extensively studied in pancreatitis. This study investigates the effects of HBO in a rat model of severe acute pancreatitis.

Methods: Sixty-four rats were induced with severe pancreatitis using 4% sodium taurocholate and randomized to HBO treatment or control. HBO was commenced 6 h after induction (100% oxygen at 2.5 atmospheres for 90 min) and continued every 12 h for a maximum of eight treatment episodes.

Surviving animals were killed at 7 days. Severity of pancreatitis was graded macroscopically and microscopically. Lung edema was calculated using wet and dry lung weights. Macroscopic and microscopic severity scores (mean ± SE) of HBO-treated animals with pancreatitis (8.3 ± 0.7; 9.6 ± 0.4) were lower than those of controls (10.5 ± 0.5; 11.1 ± 0.4) ($p = 0.02$ and $p = 0.03$, respectively).

The HBO-treated group had reduced pancreatic necrosis compared to controls ($40 \pm 4\%$ vs. $54 \pm 4\%$; $p = 0.003$). There was no difference in pulmonary edema between the groups. Median survival in the HBO-treatment group was 51 h, compared to 26 h in controls. Day-7 survival was significantly improved in the HBO-treated animals compared to controls (40% vs. 27%; $p = 0.04$).

Conclusion: HBO therapy reduces overall severity, decreases the extent of necrosis, and improves survival in severe acute pancreatitis.

Keywords *Severe acute pancreatitis - Necrosis - Hyperbaric oxygen therapy*

Global Toy Concern

Chinese Santa

legal fees, chemical processing fees, disposal fees, business taxes, and inspection costs on every level of manufacture and many more infinitesimal costs on 'possible' risks. Quite simply, we have put many companies out of business with sets of rules intending to rule out any and all possible harm to children.

In theory, these *precautions* sound like an awesome idea. In practice, we wind up with *China Toxicity Blues.* American toy manufacturers (many out of business now) are left with the burden of proving any number of "possibilities" of harm, the overwhelming amount of which haven't the faintest chance of even occurring. Yet, the tests—the rules—must be followed. We are so concerned with the effects on the environment (the trees), that we overlook the forest (our children).

On the micro-level, testing every single possibility has made manufacturers paranoid about violating "rules" here in America. Instead, China has become an appealing solution.

We know that if is American-made, costs will limit quality—in the aesthetic sense, especially—and demand. But why? As consumers, we need to make hard choices. Do we continue to purchase toxic goods from China, or do we insist from legislators that business be allowed to manufacture goods within reasonable parameters.

This is just a theory, but why are our children being poisoned by our lack of information? We must empower ourselves to be savvy consumers. If we are going to engage in the game, then we must know all the players, the rules and how to stay in the game—it's childish not to.

We buy without thinking of the free market process and even fall into the pitfalls of "boycotting" companies, countries, without seeing the real problem—we're putting ourselves out of business!