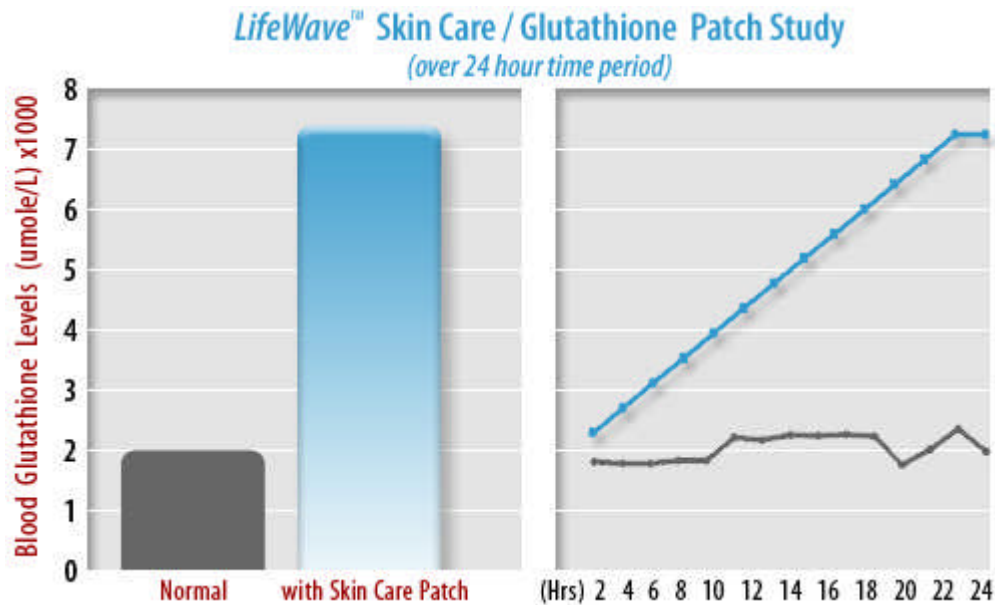


Have you seen the report on our Skin patch yet? Well here it is! September 2006



A double blind placebo controlled study was performed to independently examine the effectiveness of the LifeWave Skin Care/Glutathione patch in being able to elevate blood levels of Glutathione over a period of 24 hours and several days. Baseline data was collected for all subjects; the average Blood Glutathione levels for all individuals was an average of 2020 umole/L as displayed by the graph on the left ("Normal" value). After 24 hours of using the LifeWave Skin Care/Glutathione patch, the average blood Glutathione levels of all individuals was 7326 umole/L. This means that within 24 hours of using the LifeWave Skin Care/Glutathione patch the average increase in Glutathione is over 3 TIMES HIGHER than normal. The graph on the right shows how over a period of 24 hours blood Glutathione levels increase dramatically for LifeWave patch users versus not using the LifeWave Skin Care patch.

Blood Glutathione levels have been identified as being one of the most important indicators of our overall health. As the master antioxidant in the body Glutathione has a range of diverse metabolic functions including acting as a free radical scavenger, "recharging" depleted antioxidants back into their active state (Vitamin C, Vitamin E, Vitamin A, etc.), maintaining the immune system, supporting protein structures and removing heavy metals such as mercury through the liver.

Present methods that attempt to elevate Glutathione levels are not very effective. Oral supplements of Glutathione are destroyed by stomach acids. Glutathione is a tri-peptide composed of three amino acids and adding these amino acids to the diet does not guarantee elevated levels of Glutathione. Injections of Glutathione does translate into elevated levels of Glutathione in the blood however this method is undesirable on a daily basis, is expensive and inconvenient.

The LifeWave Skin Care/Glutathione patch is a new way to dramatically increase Glutathione levels in the body on a daily basis. Our clinical studies show that the AVERAGE increase in blood Glutathione levels over a 24 hour period is a remarkable 3 TIMES HIGHER than normal. Our patch technology makes elevating Glutathione levels convenient, effective and safe.

Glutathione in disease.

Review Article

Current Opinion in Clinical Nutrition & Metabolic Care. 4(1):65-71, January 2001.

Reid, Marvin a,b; Jahoor, Farook a

Abstract:

Altered glutathione metabolism in association with increased oxidative stress has been implicated in the pathogenesis of many diseases. However, whether strategies aimed at restoring glutathione concentration and homeostasis are effective in ameliorating or modifying the natural history of these states is unknown. In this review we discuss the pathogenic role for altered glutathione metabolism in such diseases as protein energy malnutrition, seizures, Alzheimer's disease, Parkinson's disease, sickle cell anaemia, chronic diseases associated with ageing and the infected state. In addition, we discuss the efficacy of glutathione precursors in restoring glutathione homeostasis both in vitro and in vivo.

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1: [Neurosci Lett](#). 1982 Dec 13;33(3):305-10.

[Related Articles](#). [Links](#)

Parkinson's disease: a disorder due to nigral glutathione deficiency?

[Perry TL](#), [Godin DV](#), [Hansen S](#).

Amino acid analysis of autopsied human brain showed reduced glutathione (GSH) content significantly lower in the substantia nigra than in other brain regions. GSH was virtually absent in the nigra of patients with Parkinson's disease. Oxidative degradation of L-DOPA and dopamine in vivo may generate reactive oxygen species (hydrogen peroxide, superoxide, hydroxyl radical, or singlet oxygen) which can damage membranes and other cellular components. Since GSH is an important natural antioxidant, a deficiency of GSH in the substantia nigra could make this region vulnerable to oxidative injury. If confirmed, the hypothesis that loss of nigrostriatal dopaminergic neurons results from a regional GSH deficiency could have important therapeutic implications for the management and prevention of Parkinson's disease.

Glutathione deficiency is associated with impaired survival in HIV disease

(AIDS / N-acetylcysteine / redox)

Leonore A. Herzenberg^{*}, Stephen C. De Rosa^{*}, J. Gregson Dubs^{*}, Mario Roederer^{*}, Michael T. Anderson^{*}, Stephen W. Ela^{*}, Stanley C. Deresinski[‡], and Leonard A. Herzenberg^{*}

Departments of ^{*} Genetics and [‡] Medicine, Stanford University Medical School, Stanford, CA 94305-5125

Contributed by Leonard A. Herzenberg, December 30, 1996

Glutathione (GSH), a cysteine-containing tripeptide, is essential for the viability **and** function of virtually all cells. *In vitro* studies showing that low GSH levels both promote HIV expression **and** impair T cell function suggested a link between GSH depletion **and** HIV **disease** progression. Clinical studies presented here directly demonstrate that low GSH levels predict poor survival in otherwise indistinguishable HIV-infected subjects. Specifically, we show that GSH deficiency in CD4 T cells from such subjects is associated with markedly decreased survival 2-3 years after baseline data collection (Kaplan-Meier **and** logistic regression analyses, $P < 0.0001$ for both analyses). This finding, supported by evidence demonstrating that oral administration of the GSH prodrug *N*-acetylcysteine replenishes GSH in these subjects **and** suggesting that *N*-acetylcysteine administration can improve their survival, establishes GSH deficiency as a key determinant of survival in HIV **disease**. Further, it argues strongly that the unnecessary or excessive use of acetaminophen, alcohol, or other drugs known to deplete GSH should be avoided by HIV-infected individuals.

Lung glutathione and oxidative stress: implications in cigarette smoke-induced airway disease Irfan Rahman and William MacNee

**Department of Respiratory Medicine, Medical School,
University of Edinburgh, Edinburgh EH8 9AG, United Kingdom**

Glutathione (GSH), a ubiquitous tripeptide thiol, is a vital intra- and extracellular protective antioxidant in the lungs. The rate-limiting enzyme in GSH synthesis is γ -glutamylcysteine synthetase (γ -GCS). The promoter (5'-flanking) region of the human γ -GCS heavy and light subunits are regulated by activator protein-1 and antioxidant response elements. Both GSH and γ -GCS expression are modulated by oxidants, phenolic antioxidants, and inflammatory and anti-inflammatory agents in lung cells. γ -GCS is regulated at both the transcriptional and posttranscriptional levels. GSH plays a key role in maintaining oxidant-induced lung epithelial cell function and also in the control of proinflammatory processes. Alterations in alveolar and lung GSH metabolism are widely recognized as a central feature of many inflammatory lung diseases including chronic obstructive pulmonary disease (COPD). Cigarette smoking, the major factor in the pathogenesis of COPD, increases GSH in the lung epithelial lining fluid of chronic smokers, whereas in acute smoking, the levels are depleted. These changes in GSH may result from altered gene expression of γ -GCS in the lungs. The mechanism of regulation of GSH in the epithelial lining fluid in the lungs of smokers and patients with COPD is not known. Knowledge of the mechanisms of GSH regulation in the lungs could lead to the development of novel therapies based on the pharmacological or genetic manipulation of the production of this important antioxidant in lung inflammation and injury. This review outlines 1) the regulation of cellular GSH levels and γ -GCS expression under oxidative stress and 2) the evidence for lung oxidant stress and the potential role of GSH in the pathogenesis of COPD.

γ -glutamylcysteine synthetase; oxidants; antioxidants; activator protein-1; antioxidant response element; smokers; lungs; chronic obstructive pulmonary disease; airway epithelium

1: [Brain Res Brain Res Rev.](#) 1997 Dec;25(3):335-58.

[Related Articles](#), [Links](#)

ELSEVIER
FULL-TEXT ARTICLE

Neurodegenerative disorders in humans: the role of glutathione in oxidative stress-mediated neuronal death.

[Bains JS](#), [Shaw CA](#).

Department of Ophthalmology, The University of British Columbia, Vancouver, Canada. jbains@unixg.ubc.ca

Oxidative stress has been implicated in both normal aging and in various neurodegenerative disorders and may be a common mechanism underlying various forms of cell death including necrosis, apoptosis, and excitotoxicity. In this review, we develop the hypothesis that oxidative stress-mediated neuronal loss may be initiated by a decline in the antioxidant molecule glutathione (GSH). GSH plays multiple roles in the nervous system including free radical scavenger, redox modulator of ionotropic receptor activity, and possible neurotransmitter. GSH depletion can enhance oxidative stress and may also increase the levels of excitotoxic molecules; both types of action can initiate cell death in distinct neuronal populations. Evidence for a role of oxidative stress and diminished GSH status is presented for Lou Gehrig's disease (ALS), Parkinson's disease, and Alzheimer's disease. Potential links to the Guamanian variant of these diseases (ALS-PD complex) are discussed. In context to the above, we provide a GSH-depletion model of neurodegenerative disorders, suggest experimental verifications of this model, and propose potential therapeutic approaches for preventing or halting these diseases.

1: [Hepatology](#). 1992 Dec;16(6):1423-7.

[Related Articles](#), [Links](#)

Hepatic mitochondrial glutathione depletion and progression of experimental alcoholic liver disease in rats.

[Hirano T](#), [Kaplowitz N](#), [Tsukamoto H](#), [Kamimura S](#), [Fernandez-Checa JC](#).

University of Southern California School of Medicine Department of Veterans Affairs Outpatient Clinic, Los Angeles 90033.

Long-term ethanol feeding has been shown to selectively reduce hepatic mitochondrial glutathione content by impairing mitochondrial uptake of this thiol. In this study, we assessed the role of this defect in evolution of alcoholic liver disease by examining the mitochondrial glutathione pool and lipid peroxidation during progression of experimental alcoholic liver disease to centrilobular liver necrosis and fibrosis. Male Wistar rats were intragastrically infused with a high-fat diet plus ethanol for 3, 6 or 16 wk (the duration that resulted in induction of liver steatosis, necrosis and fibrosis, respectively). During this feeding period, the cytosolic pool of glutathione remained unchanged in the ethanol-fed animals compared with that in pair-fed controls. In contrast, the mitochondrial pool of glutathione selectively and progressively decreased in rats infused with ethanol for 3, 6 or 16 wk, by 39%, 61% and 85%, respectively. Renal mitochondrial glutathione level remained unaffected throughout the experiment. Serum ALT levels increased significantly in the ethanol-fed rats at 6 wk and remained elevated at 16 wk. In the mitochondria with severely depleted glutathione levels at 16 wk, enhanced lipid peroxidation was evidenced by increased malondialdehyde levels. Thus a progressive and selective depletion of mitochondrial glutathione is demonstrated in the liver in this experimental model of alcoholic liver disease and associated with mitochondrial lipid peroxidation and progression of liver damage.

Decreased glutathione transferase activity in brain and ventricular fluid in Alzheimer's disease

MA Lovell, C Xie and WR Markesbery

Department of Chemistry, Sanders-Brown Center on Aging, University of Kentucky, Lexington 40536-0230, USA.

OBJECTIVE: To investigate the levels of glutathione transferase (GST), a protective enzyme against aldehydes, and especially 4-hydroxynonenal (HNE) in the brain and ventricular CSF of autopsied AD and normal control subjects. **BACKGROUND:** Studies have implicated increased levels of oxidative stress in the brain in the pathogenesis of AD. Decreased levels of polyunsaturated fatty acids and increased levels of markers of lipid peroxidation have been reported in the brain in AD, particularly in areas severely affected in the disease. HNE, one marker of lipid peroxidation, is neurotoxic in neuronal culture and in vivo and is elevated in AD brain and CSF. **METHODS:** We measured levels of GST activity and protein in multiple brain regions and ventricular CSF in short-postmortem-interval AD patients and age-matched prospectively evaluated control subjects. **RESULTS:** A decrease in GST activity in all brain areas was observed in AD compared with controls with significant decreases in the amygdala, hippocampus and parahippocampal gyrus, inferior parietal lobule, and nucleus basalis of Meynert. Levels of GST protein also were depleted in most brain regions in AD. A significant decrease in GST activity and protein levels was also found in ventricular CSF in AD. **CONCLUSION:** Reduced levels of GST, a protective mechanism against HNE, may have a role in the pathogenesis of neuron degeneration in AD.

Impairment of intestinal glutathione synthesis in patients with inflammatory bowel disease

B Sido,^a V Hack,^b A Hochlehnert,^a H Lipps,^b C Herfarth,^a W Dröge^b

^a Department of Surgery, University of Heidelberg, Heidelberg, Germany,

^b Department of Immunochemistry, German Cancer Research Center, Heidelberg, Germany

Correspondence to: Dr B Sido, Department of Surgery, University of Heidelberg, Im Neuenheimer Feld 110, 69120 Heidelberg, Germany.

Accepted for publication 31 October 1997

Background—Reactive oxygen species contribute to tissue injury in inflammatory bowel disease (IBD). The tripeptide glutathione (GSH) is the most important intracellular antioxidant.

Aims—To investigate constituent amino acid plasma levels and the GSH redox status in different compartments in IBD with emphasis on intestinal GSH synthesis in Crohn's disease.

Methods—Precursor amino acid levels were analysed in plasma and intestinal mucosa. Reduced (rGSH) and oxidised glutathione (GSSG) were determined enzymatically in peripheral blood mononuclear cells (PBMC), red blood cells (RBC), muscle, and in non-inflamed and inflamed ileum mucosa. Mucosal enzyme activity of γ -glutamylcysteine synthetase (γ GCS) and γ -glutamyl transferase (γ GT) was

analysed. Blood of healthy subjects and normal mucosa from a bowel segment resected for tumour growth were used as controls.

Results—Abnormally low plasma cysteine and cystine levels were associated with inflammation in IBD ($p < 10^{-4}$). Decreased rGSH levels were demonstrated in non-inflamed mucosa ($p < 0.01$) and inflamed mucosa ($p = 10^{-6}$) in patients with IBD, while GSSG increased with inflammation ($p = 0.007$) compared with controls. Enzyme activity of γ GCS was reduced in non-inflamed mucosa ($p < 0.01$) and, along with γ GT, in inflamed mucosa ($p < 10^{-4}$). The GSH content was unchanged in PBMC, RBC, and muscle.

Conclusions—Decreased activity of key enzymes involved in GSH synthesis accompanied by a decreased availability of cyst(e)ine for GSH synthesis contribute to mucosal GSH deficiency in IBD. As the impaired mucosal antioxidative capacity may further promote oxidative damage, GSH deficiency might be a target for therapeutic intervention in IBD.

(*GUT* 1998;**42**:485-492)

Keywords: Crohn's disease; ulcerative colitis; glutathione; amino acids; γ -glutamylcysteine synthetase; mucosa

Significance of glutathione in lung disease and implications for therapy.

Morris PE, Bernard GR.

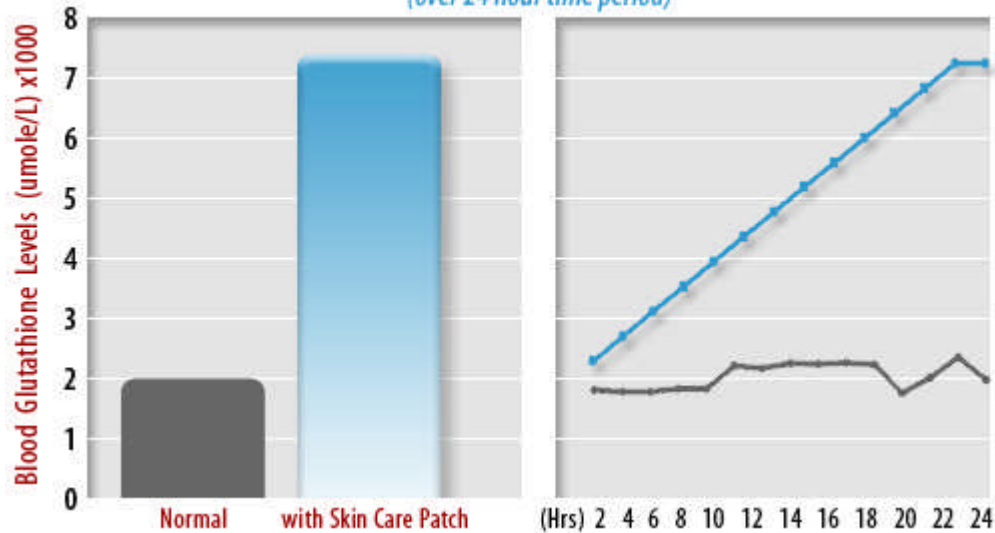
Center for Lung Research, Vanderbilt University, Nashville, Tennessee.

Glutathione is a tripeptide that contains an important thiol (sulfhydryl) group within the central cysteine amino acid. Glutathione is involved in numerous vital processes where the reducing potential of the thiol is used. Several lung disorders are believed to be characterized by an increase in alveolar oxidant burden, potentially depleting alveolar and lung glutathione. Low glutathione has been linked to abnormalities in the lung surfactant system and the interaction between glutathione and antiproteases in the epithelial lining fluid of patients. Normal levels of intracellular glutathione may exert a critical negative control on the elaboration of proinflammatory cytokines. The increase of intracellular reactive oxygen species is believed to correlate with the activation of NF-kappa B, a transcription activator linked to the elaboration of several cytokines. There is now sufficient data to strongly implicate free radical injury in the genesis and maintenance of several lung disorders in humans. This information is substantial and will help the development of clinical studies examining a variety of inflammatory lung disorders.

NOW, WHAT IS GOING TO HAPPEN? ONLY TIME WILL TELL. BUT WHAT DO YOU THINK?

Have you seen the report on our Skin patch yet ??? Well here it is

LifeWave™ Skin Care / Glutathione Patch Study (over 24 hour time period)



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