



HMP Heavy Metal Protect

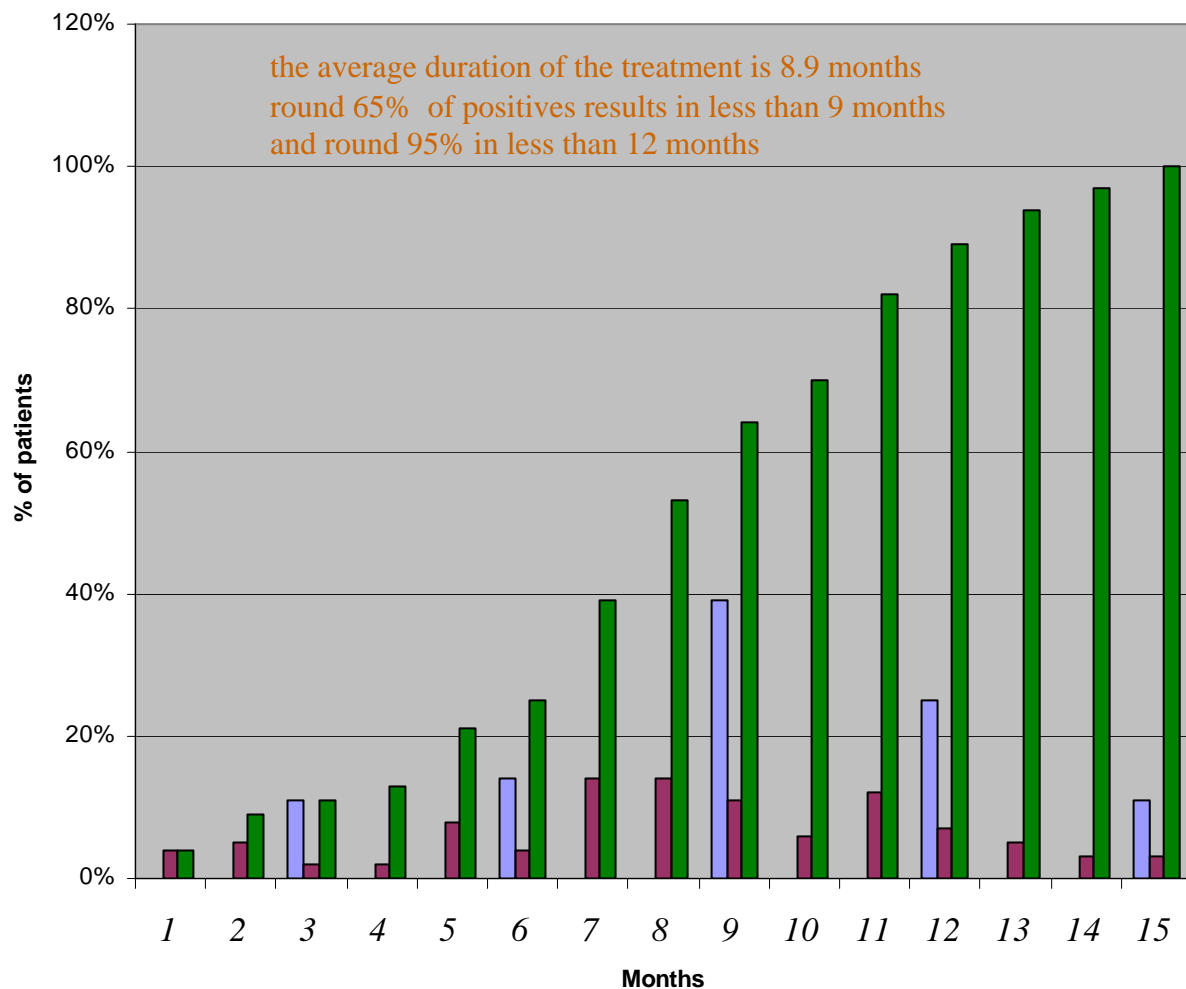
GCF (Gluten, Lactose and Casein Free)

◆ **Results**

Results with 100 patients

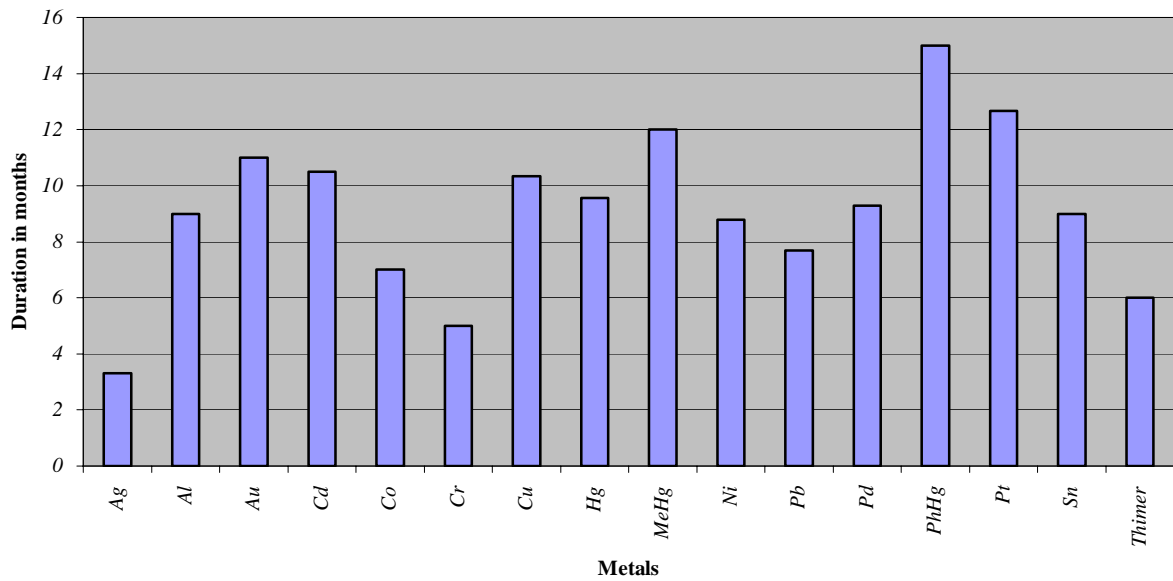
Melisa negatvation

■ *Quarterly* ■ *Monthly* ■ *General*

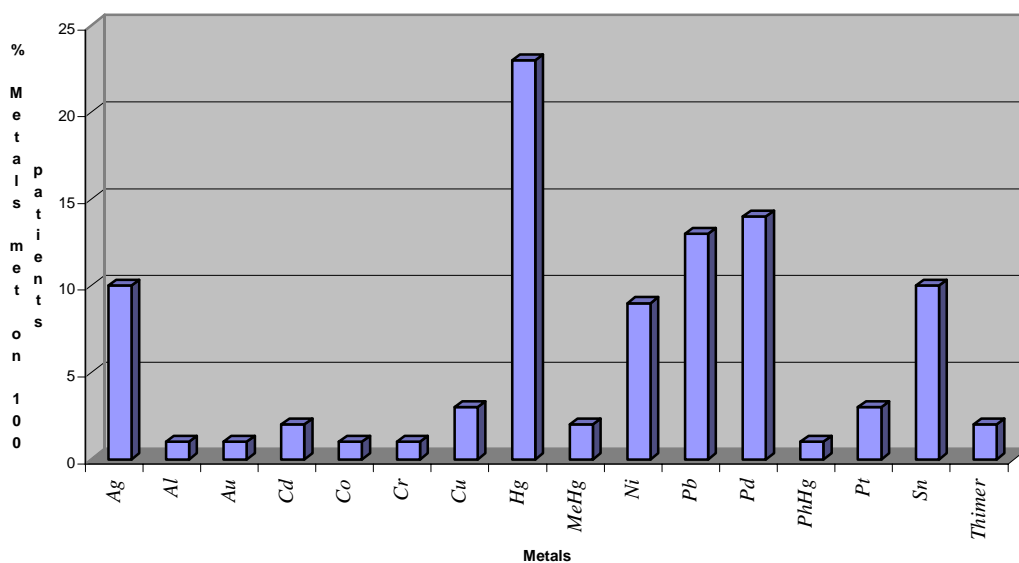




Duration of treatment for each metal



Récurrance of the metals met, in %





◆ **Composition**

GSH (reduced glutathione), Lipoic acid (tiotic), SOD (50U/g), Selenomethionine (Se 40%), Vitamin E (DL-alpha-tocopherol), Piconogenol (OPC extract of grape seed), Vitamin B2 (Riboflavin), Mycelium shitake (atomized), willow extract

◆ **How the various components work**

α -lipoic acid :

This is an octanoic acid, i.e. a fatty acid, whose carbon 6 and carbon 8 hydrogen atoms are replaced by a disulphuric bridge (-S – S-). It is part of the vitamin B group and its role is vital in the energy metabolism. It is a co-factor in pyruvate dehydrogenase and is found in reduced form with two –SH after carrying out its catalytic role at the Krebs cycle level.

Lipoic acid is a very powerful anti-oxidant but can also be used to detect toxic metals because of its two thiol groups.

GSH :

Glutathione^B is a tripeptid – γ glutaminacysteinilglycine – co-factor in the destruction of free radicals of oxygen (OFR).

In cases of poisoning by metals its activity is blocked.

In addition, toxic metals inhibit the metabolism of the mitochondria whose role is to reduce the content of oxygen in water.

The inhibition of the metabolism of the mitochondria leads to the formation of OFR.

The addition of reduced glutathione (GSH) keeps the OFR at a normal level.

It should also be noted that GSH is involved in the synthesis of prostaglandin H₂, thus regulating inflammation.



Selenomethionine and Vit.E

Where hydrogen peroxide accumulates, selenium, a co-factor in membrane and cytoplasm glutathione peroxidase, is indispensable for the formation of a reservoir of glutathione in the form of glutathione disulfide (GSSG).

The cell can once more be fed with GSH via the action of glutathione reductase.

Selenium allows the formation of a « GSH-heavy metal » complex such as GS-Hg-SG which blocks the vital activity of GSH.

The choice of selenium associated with methionine is motivated by the fact that methionine is an essential precursor amino acid for cysteine, another indispensable amino acid for the proper structure of proteins. In the presence of toxic metals, cysteine becomes unavailable because it is an amino acid with a thiol function (-SH) very similar to that of metals.

The presence of vitamin E, a liposoluble vitamin, prevents the oxidation of fatty membrane acids.

SOD

This enzyme is found in the mitochondria and in the cytoplasm. Its role is to destroy superoxide anions.

The cofactors of SOD are the oligo-elements Mn-Zn and Cu. Toxic metals such as Cd and Hg displace these oligo-elements and inhibit the activity of the superoxide dismutase.

Pycnogenol

Pycnogenol is a mixture of flavonoids rich in polyphenols and proantocyanidines.

Pycnogenol has numerous interesting functions from the biological point of view: its components are anti-radicals and have a much longer active half-life than vitamins C and E, and also can be used to treat inflammation.

The hydrosoluble active principles of pycnogenol cross the cephalo-meningeal barrier protecting the central nervous system from the effects of toxic metals involved in the etiology of some degenerative nervous diseases.

The addition of pycnogenol also enables regeneration of the ascorbyl group (vit C)^C and protects GSH against oxidant stress.



Vitamin B2

Riboflavin, in the form of FAD, is the vitaminic co-factor in cytochrome-C-oxidase.

This enzyme located in the mitochondria at the end of the respiratory chain eliminates any remaining free radicals not destroyed upstream.

Mycelium shitake:

allows passage through the cell wall, has a nutritional and revitalising action, breaks metallic bridges existing at enzyme and protein levels; some Japanese studies have shown an important immuno-stimulatory function.

Willow (Salix alba)

Has anti-inflammatory and dechelatory properties.

- ◆ A box of 60 capsules.
- ◆ Recommendations for administration: 1 capsule twice a day during meals, for a period of at least 3 months

^B **GSH**

acidity of the stomach induces hydrolysis of the amino components. Taken during meals, with an alimentary bolus, around 80% is absorbed in the small intestine and the colon.

^C **Vitamin C or ascorbic acid**

Vitamin C is known to have anti-oxidation properties. However, in certain cases, vitamin C has an oxidating action. A paradox?

The paradox depends on the concentration of the vitamin and the power of the ascorbic acid to reduce transition metals.

In the presence of ferric iron, vitamin C exercises a pro-oxidation action leading to an increase in free fatty acids and triglycerides; similarly a strong increase in the activity of β -N-acetyl-D-glucosaminidase reflecting lysis of the lysosomal membrane freeing proteolytic enzymes in the cytoplasm and retarded growth.¹

It should also be noted that vitamin C increases the production of intra-erythrocytic hydrogen peroxide (H_2O_2) in a dose-dependent fashion.²

In this specific case, intra-globular toxic production derives from an excess of vitamin C and is associated with the inhibition of erythrocytic catalase activity by Fe^{3+} ions in the extracellular compartments.

Fe^{3+} and peroxides oxidise ascorbic acid which thereby becomes a pro-oxidation agent.

This combined action of iron and vitamin C is normally slowed by vitamin E and glutathione provided that the latter remains in the reduced GSH form.

It is known that toxic metals block glutathione activity.

It is nonetheless true that whilst the presence of ascorbic acid reduces the risk of oxidation, in some circumstances vitamin C can promote free radicals and lipidic peroxidation.

It has been demonstrated, for example, that anti-oxidants, including vitamin C, are active in the preventive phase but speed up the process of oxidation of LDLs once this process has begun.³

¹ Chen K. et al. *Am J Physiol Endocrinol Metab* 2000 **279** : 1406-1412

² Shalu Mendiratta et al. *Biochimica et Biophysica Acta* 1998 **1380** : 389-395

³ Otero P. et al. *Free Radical Research* 1997 **27** : 619-626