



The Lead Education and Abatement Design Group
Working to eliminate lead poisoning globally and to protect the
environment from lead in all its uses: past, current and new uses
ABN 25 819 463 114

Alcohol's link to higher lead and iron levels

By Robert Taylor, Researcher, The LEAD Group Inc.

Since the 1980s it has been clear there were links between higher blood lead levels and alcohol consumption (Shaper et al 1982, Hense et al 1992, Pizent et al 2001). In the USA women who were heavy alcohol consumers were 5.6 times more likely to be in the highest 10% of the population for blood lead than teetotallers [consumers of no alcohol] (Lee et al 2005). Much effort was expended in the 1990's, tracking possible sources of lead contamination in alcohol, notably lead seals on wine bottles and illicit liquor. But the overall correlations were not explainable by the degree of lead contamination (Shaper et al 1982 p301).

A clue, however, appeared in an apparent contradiction – individuals who consumed significant alcohol were likely to have higher iron levels (Whitfield 2001). Two glasses of alcohol a day reduces the risk of being iron deficient (Whitfield et al 2001 p1041) and any alcohol consumption reduced the risk of iron deficiency anaemia by up to 40% (Ioannou et al 2004). Again much research effort was spent identifying sources of contamination, notably in Africa, where beer was brewed in iron vats (Choma et al 2007). But the trend was apparent outside Africa (Whitfield et al 2001) and was the result of increased rates of iron absorption from the intestine (Duane et al 1992, Kohgo et al 2007 p4701).

The reason why higher levels of iron and lead are contradictory is that lead and iron compete for absorption. Indeed lead is only readily absorbed and distributed because body systems are unable to easily identify lead from iron, calcium and, in some cases, zinc and lead replaces these essential minerals in many key functions. Iron and lead share a common transporter within the body, DMT1, which is used for both intestinal absorption of iron (or lead) and their transfer within the body via the bloodstream. DMT1 levels are increased by iron deficiency. There is clear evidence that iron deficiency results in higher lead absorption. (Taylor 2009). This should mean that if adequate amounts of iron are absorbed, lead absorption should be restricted to some degree. For individuals who consume significant alcohol, the reverse seems true – they absorb more lead as they absorb more iron.

An explanation was not possible until the first decade of the twenty first century. In 2003-2004 the molecule that predominantly regulates iron metabolism was identified: hepcidin. The higher the iron storage levels within the body are, the more hepcidin is produced by the liver and the less iron can be absorbed (Harrison-Findik 2009). One mechanism for this is the suppression of DMT1 production which thus reduces intestinal absorption of lead (Mena et al 2008). Iron storage levels within the body have more impact on the absorption of iron than either the quantity of iron in foods consumed or the food iron's bioavailability (capacity to be absorbed) (Hunt 2001).

The LEAD Group Inc.

PO Box 161 Summer Hill NSW 2130 Australia

Ph: (02) 9716 0014, Email www.lead.org.au/cu.html Web: www.lead.org.au/

It is clear that alcohol alters the rate of hepcidin production, at least partly through oxidative stress from free radicals in the body; though the range of methods by which it alters hepcidin levels is yet to be fully understood (Harrison-Findik 2009). Direct genetic impacts also occur. An intriguing study was done on the impact of ethanol (the active ingredient in alcohol) on the genes that govern hepcidin production. Ethanol reverses the impact of iron storage levels on most of these genes, so the alcohol-influenced body reacts to adequate iron as if it were iron deficient – reducing hepcidin production to increase iron absorption (Crist et al 2007). Given that iron deficiency increases lead absorption the predictable outcome should be an increase in lead absorption, which is what is found, despite the absence of actual iron deficiency.

Thus the theoretical impact reflects real world observations, but it must be emphasised far more work is needed to confirm the mechanisms by which alcohol increases both iron and lead absorption. The impacts of different ethanol doses and frequency of ethanol consumption (alcohol intakes), the relative effects on iron and lead absorption and even the confirmation that alcohol consumption leads to ethanol affecting the genes governing hepcidin, all need to be elucidated. But on present data, it would seem that alcohol greatly increases lead absorption by damaging the body's ability to regulate the absorption of iron and a fair proportion of this impact is likely to be due to impacts at the cellular level on DNA and/or RNA.

There is significant data that indicates that alcohol may also increase the susceptibility of some organs, to lead toxicity, by depleting calcium, zinc and magnesium levels (Béchetoille et al 1983, Flora et al 1991, Gupta & Gill 2000a) and some evidence alcohol could possibly magnify the impact of lead (act synergistically with lead) on organs such as the brain (Gupta & Gill 2000b). In conjunction with lead, alcohol may also increase the risk of high blood pressure among women (Hense et al 1994, Pizent et al 2001) though it must be emphasised the size of the link is not large, there is much contradictory data on links between blood lead levels and blood pressure and lead may not be a direct cause of higher blood pressure (Nawrot et al 2002, Hond et al 2003). There have been studies that have shown links between blood pressure and blood lead among US African- Americans, particularly women, but not US whites (Vupputuri et al 2003) and among Taiwanese males but not females (Chu et al 1999) while in Germany (Hense et al 1994) there was increased blood pressure among heavy female alcohol consumers but only among rural (not urban) heavy male alcohol consumers. Far more work is needed on this subject before any definitive statements can be made.

References

A Béchetoille, P Allain, J M Ebran, Y Mauras (1983) **“Modifications in blood zinc and lead concentrations and ALA-dehydratase activity in optic neuropathy from alcohol and tobacco abuse”** *Journal français d'ophtalmologie*. 02/1983; 6(3):231-5

http://www.researchgate.net/publication/16324230_Modifications_in_blood_zinc_and_lead_concentrations_and_ALA-dehydratase_activity_in_optic_neuropathy_from_alcohol_and_tobacco_abuse

Callie Crist, Elizabeth Klein, John Gollan and Dee Harrison-Findik, Jonathan Frye **“The Interaction of Alcohol and Iron-Overload in the in-vivo Regulation of Iron Responsive Genes”** *Cantaurus*, Vol. 15, 2-6, May 2007 <http://www.mcpherson.edu/science/cantaurus/07-crist.pdf>

Solomon Simon Ramphai Choma, Marianne Alberts, Petter Urdal (2007) **“Effect of traditional beer consumption on the iron status of a rural South African population”** *SAJCN 2007 Vol 20 No 2* <http://www.sajcn.com/2007/effect20no2.pdf>

Nain-Feng Chu, Saou-Hsin Liou, Trong-Neng Wu, Po-Ya Chang (1999) **“Reappraisal of the relation between blood lead concentration and blood pressure among the general population in Taiwan”** *Occup Environ Med* 1999;56:30-33
<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1757651/pdf/v056p00030.pdf>

P. Duane, K. B. Raja, R. J. Simpson and T. J. Peters (1992) **“Intestinal Iron Absorption In Chronic Alcoholics”** *Alcohol and Alcoholism* (1992) 27 (5): 539-544.
<http://alcalc.oxfordjournals.org/content/27/5/539.abstract>

S. J. S. Flora , Deo Kumar, S. R. S. Sachan and S. Das Gupta (1991) **“Combined exposure to lead and ethanol on tissue concentration of essential metals and some biochemical indices in rat”** *Biological Trace Element Research Vol 28 No 2 February 1991*
<http://www.springerlink.com/content/e114062566104376/>

Duygu Dee Harrison-Findik (2009) **“Is the iron regulatory hormone hepcidin a risk factor for alcoholic liver disease?”** *World J Gastroenterol* 2009 March 14; 15(10): 1186-119
www.ncbi.nlm.nih.gov/pmc/articles/PMC2658862/pdf/WJG-15-1186.pdf

Vandana Gupta & Kiran Dip Gill (2000a) **“Influence of ethanol on lead distribution and biochemical changes in rats exposed to lead”** *Alcohol Vol. 20, Is. 1 , Pages 9-17, January 2000*
[http://www.alcoholjournal.org/article/S0741-8329\(99\)00046-4/abstract](http://www.alcoholjournal.org/article/S0741-8329(99)00046-4/abstract)

Vandana Gupta & Kiran Dip Gill (2000b) **“Lead and Ethanol Coexposure: Implications on the Dopaminergic System and Associated Behavioral Functions”** *Pharmacology Biochemistry and Behavior Vol. 66, Is. 3, July 2000, Pages 465-474*

Hense HW, Filipiak B, Novak L, Stoeppler M (1992) **“Nonoccupational determinants of blood lead concentrations in a general population.”** *Int J Epidemiol.* 1992 Aug;21(4):753-62.
<http://www.ncbi.nlm.nih.gov/pubmed/1521981>

Hense HW, Filipiak B & Keil U. (1994) **“Alcohol consumption as a modifier of the relation between blood lead and blood pressure.”** *Epidemiology.* 1994 Jan;5(1):120-3.
<http://www.ncbi.nlm.nih.gov/pubmed/8117770>

Elly Den Hond, Tim Nawrot and Jan A. Staessen (2003) **“Hypertension and Low-Level Lead Exposure: A Scientific Issue or a Matter of Faith?”** *Letters in Hypertension* 2003, 42:e9
<http://hyper.ahajournals.org/content/42/3/e9.full.pdf>

Janet R Hunt (2001) **“How important is dietary iron bioavailability?”** *The American Journal of Clinical Nutrition* 2001;73:3-4 Editorial www.ajcn.org/cgi/reprint/73/1/3

George N. Ioannou, Jason A. Dominitz, Noel S. Weiss, Patrick J. Heagerty and Kris V. Kowdley (2004) **“The effect of alcohol consumption on the prevalence of iron overload, iron deficiency, and iron deficiency anemia.”** *Gastroenterology* 2004 May;126(5):1293-301
<http://www.ncbi.nlm.nih.gov/pubmed/15131790>

- Yutaka Kohgo, Katsuya Ikuta, Takaaki Ohtake, Yoshihiro Torimoto, Junji Kato (2007) **“Iron overload and cofactors with special reference to alcohol, hepatitis C virus infection and steatosis/insulin resistance”** *World J Gastroenterol* 2007; 13(35): 4699-4706 <http://www.wjgnet.com/1007-9327/13/4699.pdf>
- Lee, Mi-Gyung Chun, Ock Kyoung Sung, Wan O. (2005) **“Determinants of the Blood Lead Level of US Women of Reproductive Age”** *J. Am. College of Nutrition*, Vol. 24, No. 1, 1-9 (2005) <http://www.jacn.org/cgi/reprint/24/1/1>
- Natalia P. Mena, Andre’s Esparza, Victoria Tapia, Pamela Valde’s, and Marco T. Nu’nez (2008) **“Hepcidin inhibits apical iron uptake in intestinal cells”** *Am J Physiol Gastrointest Liver Physiol* 294: G192-G198, 2008 <http://ajpgi.physiology.org/content/294/1/G192.full.pdf+html>
- Nawrot, L Thijs, E M Den Hond, H A Roels and J A Staessen (2002) **“An epidemiological re-appraisal of the association between blood pressure and blood lead: a meta-analysis”** *J. of Human Hypertension* (2002) 16, 123-131 <http://www.nature.com/jhh/journal/v16/n2/full/1001300a.html>
- Alica Pizent, Jasna Jurasovic, Spomenka Telisman (2001) **“Blood pressure in relation to dietary calcium intake, alcohol consumption, blood lead, and blood cadmium in female nonsmokers”** *J. of Trace Elements in Med. and Bio.* Vol. 15, Is. 2-3, 2001, p 123-130 <http://www.sciencedirect.com/science/article/pii/S0946672X01800559>
- A G Shaper, S J Pocock, M Walker, C J Wale, B Clayton, H T Delves, and L Hinks (1982) **“Effects of alcohol and smoking on blood lead in middle-aged British men.”** *Br Med J (Clin Res Ed)*. 1982 January 30; 284(6312): 299-302 <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1495866/>
- Taylor, Robert **“Iron Nutrition and Lead Toxicity”** *Lead Action News* Vol 9 no3 [http://www.lead.org.au/lanv9n3/LEAD Action News vol 9 no 3 Full.pdf](http://www.lead.org.au/lanv9n3/LEAD%20Action%20News%20vol%209%20no%203%20Full.pdf)
- Suma Vupputuri; Jiang He; Paul Muntner; Lydia A. Bazzano; Paul K. Whelton; Vecihi Batuman (2003) **“Blood Lead Level Is Associated With Elevated Blood Pressure in Blacks”** <http://hyper.ahajournals.org/cgi/content/full/41/3/463>
- J. B. Whitfield, G. Zhu, A. C. Heath, L. W. Powell, and N. G. Martin **“Effects of Alcohol Consumption on Indices of Iron Stores and of Iron Stores on Alcohol Intake Markers”** *Alcohol Clin Exp Res*, Vol 25, No 7, 2001: pp 1037-1045 <http://genepi.qimr.edu.au/contents/p/staff/CV301.pdf>