



The Use of Niacin in the Hubbard Detoxification Program

Background

Over the last three decades, the detoxification program developed by L. Ron Hubbard has been employed to help thousands of individuals find relief from symptoms associated with occupational and environmental chemical exposures. It has also been widely used as a component of drug rehabilitation.

The Hubbard regimen, which is followed on a daily basis for a period averaging 3-4 weeks, combines 20 to 30 minutes of aerobic exercise, two to four hours of sweating in a low-heat sauna (punctuated by regular breaks for cool-down and fluid replacement), electrolytes, polyunsaturated oils and gradually increasing doses of niacin, multivitamins and minerals to support the process itself and for biochemical repair¹. It is designed to gradually increase the rates at which the body can both mobilize and excrete accumulated chemical and drug contaminants.

First developed in 1978, this approach to the problem of chemical

body burden remains unique. Its aim to address the lingering effects from abuse of prescription and street drugs, as well as those from environmental chemicals, mirrors a view that has emerged in recent years among environmental health specialists: the possibility that addictive drugs and environmental pollutants initiate an identical disease process².

Program Safety

Published reports and clinical experience in thousands of cases have established the safety of the Hubbard regimen. Nearly three decades ago, one study³ examined the health status of one hundred and three individuals who volunteered for additional testing concomitant with the detoxification program. Minor temporary complications were noted in less than 3 percent of participants.

A later safety study reviewed the medical status of 24 individuals who had completed detoxification to address exposures sustained during the Chernobyl disaster⁴. "There is

evidence suggesting that the program revitalizes the immune system and improves the general physical condition of the participant," the researchers concluded. "In spite of its robust regimen, there is an absence of negative health effects." A summary of observations from the director of an occupational medicine clinic echoed this, noting that no serious side effects had been seen in 3,500 cases that completed the Hubbard program⁵.

The most comprehensive long-term review to date involved a ten-year follow up with a group of 36 individuals who completed the Hubbard program in the former Soviet Union during the 1990s⁶. A wide range of physical and psychological responses and complete laboratory panels were monitored; in all, around 370 parameters per patient were quantitatively evaluated. The health status of these individuals was compared to that of two matched groups that did not undergo detoxification, with the same parameters measured for control

group members (matched in age, gender, and location of residence).

Detoxification was found to have markedly improved the general physical and psychological conditions of the participants. There was an absence of negative health effects and, in fact, those who had completed the program were found to have better liver function than those who did not – a finding with significance in regard to the use of niacin in the program.

The Role of Niacin

The elements of the Hubbard program operate in concert, with the aim of triggering the gradual release of chemical by-products that have accumulated in fat and other tissues, promoting elimination, particularly through the skin, by inducing heavy sweating.

Research regarding the role that niacin plays in this process is incomplete. It may correct localized tissue deficiencies of niacin; it does cause a dramatic increase in free fatty acid (FFA) release from adipose tissue for over 24 hours⁷⁻⁹. Release of free fatty acids has been shown to be accompanied by a release of fat-stored toxins in animal studies^{10,11} as well as in human studies of serum PCBs after weight loss¹². The vasodilation resulting from niacin intake^{13,14} (experienced as a “flush”) may increase movement of chemical residues from deeper circulation through the dermal tissues for redistribution into sebum and/or into sweat glands. This

normally relatively minor excretory route is enhanced by increased sweating during the exercise and sauna portions of the daily regimen.

Researchers have highlighted additional benefits from niacin intake that may have relevance to the improvements seen among program completions, from reducing vascular inflammation¹⁵ to maintaining enzymes necessary for biotransformation and elimination of xenobiotics (chemicals foreign to the body)¹⁶⁻¹⁸ but a detailed discussion of these findings is beyond the scope of this paper.

Considerations Regarding Niacin Use

The form of niacin that is used in the Hubbard program is crystalline niacin, also known as “immediate release” (IR) niacin. High doses of crystalline niacin have been used effectively as a therapy to prevent heart disease for more than 50 years¹⁹⁻²¹.

“Overall, the perception of niacin side effects is often greater than the reality,” observes a recent review of safety considerations regarding the nutrient. Rare side effects, which resolve when niacin use ceases, can include reversible blurred vision, nausea and vomiting, and the exacerbation of peptic ulcers. Clinically unimportant and small (<10%) laboratory abnormalities can include slower blood clotting, increased uric acid, and decreases in platelet count

and serum phosphorus²².

The “niacin flush” is the most bothersome side effect associated with this supplement. However, “with its transient and non-pathological effects, the flushing reaction in response to supplemental nicotinic acid deserves to be characterized as a nuisance, but not as a hazard”²³. The Coronary Drug Project tested safety and effectiveness of treatment with 1-3g/day for high cholesterol and coronary outcome. At a 15-year follow-up, mortality was reduced in the treated group²⁴.

In an effort to eliminate the “flush” response, “sustained release” (SR) forms of niacin were developed in the 1960s with, as one researcher characterizes it, “problematic results”²⁵. The primary concern with SR niacin has been a potential for chemically-driven liver damage (hepatotoxicity)²⁶.

When the term “niacin” is evoked in the context of detoxification, the distinction between the two forms is critical to addressing questions regarding program safety. As numerous studies have revealed, the health effects associated with the two forms are quite different.

These differences were demonstrated in a clinical trial comparing the effects of administering increasing levels of both crystalline (IR) and slow release niacin²⁷. The trial involved 46 adults. Half were given IR niacin, and half SR niacin. After an initial treatment period, doses were sequentially

increased and participants received 1000, 1500, 2000 and 3000 mg/d of niacin, maintaining each dosage level for 6 weeks. None of the patients taking IR niacin developed hepatotoxic effects, while 12 (52%) of the 23 patients taking SR niacin did. Citing these findings, as well as case reports of adverse liver effects associated with the use of SR niacin²⁸⁻⁴² the researchers recommended that its use be restricted.

A reason has been discovered for the difference in effect from administration of the two forms of niacin⁴³, in essence that the differing absorption rates of the two different forms result in their entering different metabolic pathways^{26,44}. (A 1-g dose of IR niacin would be absorbed and metabolized within 2 hours, while absorption of a 1-g dose of SR niacin would take >20 hours⁴⁵.)

On one pathway, metabolizing of the IR form produces molecules that cause the “flushing” reaction. When niacin is absorbed slowly, as with SR formulations, a second pathway produces metabolites associated with hepatotoxicity^{46,47}. This phenomenon is reflected in case histories of individuals who developed liver abnormalities while taking SR niacin, and whose liver tests returned to normal after switching to IR niacin³⁰.

The issue of absorption has some relevance in considering the dosages of IR niacin used in the Hubbard program. These progress in stages, in balance with other nutrients, beginning at 100 mg/day and poten-

tially reaching as much as 5 g/day¹. It is important to note that many participants do not reach the highest dosages. Program completion is generally accomplished in 30 days or less; consequently, the time spent at any given dose would be a matter of days.

While these dosages are above those recommended for dietary supplementation, they are consistent with those seen in therapeutic administration of niacin for management of elevated blood lipids. The monograph for Niacor (an FDA approved commercial niacin product) notes that the maximum adult dosage is 6 g per day. Doses of IR niacin as high as 6 g have been maintained for as long as 6 months with minimal effect and no impact on liver function³⁰. Participants in the IR/SR comparison study mentioned above²⁸ received 3 g per day for six weeks with no hepatotoxic effects observed. Adverse effects associated with IR niacin intake are likewise absent in clinical delivery of detoxification to thousands of cases⁵.

In 1998, the FDA approved an “extended release” (ER) form of niacin, with the trademark Niaspan. The ER form was developed in an effort to avoid both the flushing response to IR niacin and the hepatotoxicity associated with SR forms⁴².

It is worth noting that drug users who inject drugs are at increased risk for Hepatitis C. In these cases, or where there is a history of liver disease, a physician must determine whether the individual is a candidate

for the detoxification program. A recent literature review suggests that IR niacin can be used safely in some patients with chronic liver disease with appropriate monitoring⁴⁷.

Summary

Over decades of application, involving more than 10,000 individuals with a wide range of occupational and environmental exposures or drug history, the Hubbard program has been demonstrated to be a safe, non-invasive intervention that can effectively address symptoms associated with both chemical injury and drug abuse. The scientific literature regarding therapeutic use of niacin, as well as clinical experience, offer little to suggest that the short-term use of this nutrient in the regimen presents significant or unacceptable risk to participants. Given the shortage of therapies for addressing chemical body burden and its impact, this regimen is most correctly viewed as a welcome addition to the tools available to healthcare providers.

Authors:

Kathleen Kerr, MD, FASE Senior Research Associate

Carl Smith, FASE Editor in Chief

Address correspondence to
carl.smith@fasenet.org

References:

- Hubbard LR. *The Technical Bulletins*. Vol XII: Bridge Publications, 1978.
- Miller CS. Toxicant-induced loss of tolerance. *Addiction*. 96(1):115-139, 2000.
- Schnare DW, Denk G, Shields M, Brunton S. Evaluation of a detoxification regimen for fat stored xenobiotics. *Med Hypotheses*. 1982;9(3):265-282.
- Tsyb AS, Parshkov EM, Barnes J, Yarzutkin VV, Vorontsov NV, Dedov VI. Rehabilitation of a Chernobyl affected population using a detoxification method. *Proceedings of the International Radiological Post-Emergency Response Issues Conference*. US EPA, 1998.
- Communication from David E. Root, MD, MPH, FACOEM, who was medical director of the clinic and oversaw each of the 3,500 cases on which these observations are based. Prior to entering private practice, Dr. Root was a career officer in the Air Force, serving as a flight surgeon and retiring as a Colonel. He has participated as a co-investigator in several studies relating to the program, including projects in the former Yugoslavia, Russia and Kazakhstan.
- Parshkov E, Sokolov V, Proshin A, Doroshchenko V, Barnes J, Gaiman S. Dynamics of the main systems of the body in the course and after the detoxification program (pooled analysis of three detoxification trials in Russia). Third International Conference on Chemical Contamination and Human Detoxification. New York: Hunter College, 2005.
- Carlson LA, Oro L. Effect of nicotinic acid on plasma free fatty acids - demonstration of a metabolic type of sympathicolysis. *Acta Medica Scandinavica*. 1962;172(6):641-&.
- Kamanna VS, Ganji SH, Kashyap ML. Niacin: An Old Drug Rejuvenated. *Current Atherosclerosis Reports*. Jan 2009;11(1):45-51.
- Meyers CD, Kashyap ML. Management of the metabolic syndrome-nicotinic acid. *Endocrinol Metab Clin North Am*. 2004;33(3):557-575, vii.
- Findlay GM, DeFreitas AS. DDT movement from adipocyte to muscle cell during lipid utilization. *Nature*. 1971 Jan 1 1971;229(5279):63-65.
- Mitjavila S, Carrera G, Fernandez Y. Evaluation of the toxic risk of accumulated DDT in the rat: during fat mobilization. *Arch Environ Contam Toxicol*. Jul 1981;10(4):471-481.
- Imbeault P, Chevrier J, Dewailly E, et al. Increase in plasma pollutant levels in response to weight loss is associated with the reduction of fasting insulin levels in men but not in women. *Metabolism-Clinical and Experimental*. Apr 2002;51(4):482-486.
- Messamore E. Relationship between the niacin skin flush response and essential fatty acids in schizophrenia. *Prostaglandins Leukot Essent Fatty Acids*. 2003 Dec 2003;69(6):413-419.
- Karpe F, Frayn KN. The nicotinic acid receptor--a new mechanism for an old drug. *Lancet*. 2004 Jun 5 2004;363(9424):1892-1894.
- Ganji SH, Qin SC, Zhang LH, Kamanna VS, Kashyap ML. Niacin inhibits vascular oxidative stress, redox-sensitive genes, and monocyte adhesion to human aortic endothelial cells. *Atherosclerosis*. Jan 2009;202(1):68-75.
- Berger F, Ramirez-Hernandez MH, Ziegler M. The new life of a centenarian: signaling functions of NAD(P). *Trends Biochem Sci*. Mar 2004;29(3):111-118.
- Majamaa K, Rusanen H, Remes AM, Pyhtinen J, Hassinen IE. Increase of Blood NAD⁺ and Attenuation of Lactacidemia During Nicotinamide Treatment of a Patient with the MELAS Syndrome. *Life Sciences*. 1996;58(8):691-699.
- Pastore A, Federici G, Bertini E, Piemonte F. Analysis of glutathione: implication in redox and detoxification. *Clin Chim Acta*. 2003 Jul 1 2003;333(1):19-39.
- Altschul R, Hoffer A, Stephen JD. Influence of nicotinic acid on cholesterol in man. *Arch Biochem Biophys* 1955;54:558-559.
- Charman RC, Matthews LB, Braeuler C. Nicotinic acid in the treatment of hypercholesterolemia. *Angiology* 1972;23:29-35.
- DiPalma JR, Thayer WS. Use of niacin as a drug. *Annu Rev Nutr* 1991;11:169-187.
- Guyton J, Bays H. Safety considerations regarding niacin therapy. *Am J Cardiol* 2007;99[suppl]:22C-31C.
- Hathcock JN. Vitamins and minerals: efficacy and safety. *Am J Clin Nutr*. 66:427-437, 1997.
- Berge KG, Canner PL. *Coronary Drug Project - Experience with Niacin*. Sep 21-22 1989:S49-S51.
- Knopp R. Evaluating niacin in its various forms. *Am J Cardiol* 2000;86(suppl):51L-56L.
- Pieper JA. Understanding niacin formulations. *Am J Manag Care*. 2002; 8(12), Sup: S308-31.
- McKenney JM, Proctor JD, Harris S, Chinchili VM. A comparison of the efficacy and toxic effects of sustained- vs immediate-release niacin in hypercholesterolemic patients. *JAMA*. 1994 Mar 2 1994;271(9):672-677.
- Knapp TRF, Middleton RK. Adverse effects of sustained-release niacin. *Ann Pharmacotherapy*. 1991;25:253-254.
- Clementz GL, Holmes AW. Nicotinic acid-induced fulminant hepatic failure. *J Clin Gastroenterol*. 1987;9:582-584.
- Henkin Y, Johnson KC, Segrest JP. Rechallenge with crystalline niacin after drug-induced hepatitis from sustained-release niacin. *JAMA*. 1990; 264:241-243.
- Palumbo PJ. Rediscovery of crystalline niacin. *Mayo Clin Proc*. 1991;66:112-113.
- Frost PH. All niacin is not the same. *Ann Intern Med*. 1991;114:1065.
- Etchason JA, Miller TD, Squires RW, et al. Niacin-induced hepatitis: a potential side effect with low-dose time-release niacin. *Mayo Clin Proc*. 1991; 66:23-28.
- Mullin GE, Greenson JK, Mitchel MC. Fulminant hepatic failure after ingestion of sustained release nicotinic acid. *Ann Intern Med*. 1989;111: 253-255.
- Knopp RH. Niacin and hepatic failure. *Ann Intern Med*. 1991;111:769.
- Goldstein MR. Potential problems with the widespread use of niacin. *Am J Med*. 1988;85:881.
- Ferenchick G, Rovner D. Case report: hepatitis and hematemesis complicating nicotinic acid use. *Am J Med Sci*. 1989;298:191-193.
- Hodis HN. Acute hepatic failure associated with the use of low-dose sustained-release niacin. *JAMA*. 1990;264:181.
- Patterson DJ, Dew EW, Gyorkey R, Graham GY. Niacin hepatitis. *South Med J*. 1983;76:239-241.
- Einstein N, Baker A, Galper J, Wolfe H. Jaundice due to nicotinic acid therapy. *Dig Dis*. 1975; 20:282-286.
- Dalton TA, Berry RS. Hepatotoxicity associated with sustained-release niacin. *Am J Med*. 1992; 93:102-104.
- Morgan JM, Capuzzi D, Guyton J. A new extended-release niacin (niapan): efficacy, tolerability, and safety in hypercholesterolemic patients. *Am J Cardiol*. 1998; 82(12A):29U-34U.
- Piepho RW. The pharmacokinetics and pharmacodynamics of agents proven to raise high-density lipoprotein cholesterol. *Am J Cardiol*. Dec 21 2000;86(12A):35L-40L.
- McKenney J. New perspectives on the use of niacin in the treatment of lipid disorders. *Arch Intern Med*. Apr 12 2004;164(7):697-705.
- McKenney JM, Proctor JD, Harris S, Chinchili VM. A comparison of the efficacy and toxic effects of sustained- vs immediate-release niacin in hypercholesterolemic patients. *JAMA*. 1994 Mar 2 1994;271(9):672-677.
- McKenney J. Niacin for dyslipidemia: considerations in product selection. *Am J Health Syst Pharm*. May 15 2003;60(10):995-1005.
- Gupta NK, Lewis JH. Review article: the use of potentially hepatotoxic drugs in patients with liver disease. *Aliment Pharmacol Ther* 2008; 28, 1021-1041.