

The Role of Heavy Metal Detoxification in Heart Disease and Cancers : A Pilot Study in Detoxification of Heavy Metals

**Daniel Dugi, M.D.
Sir Arnold Takemoto**

Presented at the
WESCON Biomedicine and Bioengineering Conference
Anaheim Convention Center
The Future of Medicine Afternoon Session

September 24, 2002 Anaheim, California USA

ABSTRACT

Heavy metal detoxification has been shown to decrease cancer mortality by 90% in a 18-year controlled clinical study by Blumer and Cranton. Frustachi et. al. has shown a very strong correlation between cardiomyopathy (heart disease) and heavy metals accumulation in the coronary arteries and heart muscle. The role of heavy metal detoxification in the prevention and/or treatment of cancers and heart disease is paramount for optimum healing or prevention. Heavy metal accumulation can cause suppression of the immune system, bind receptor sites, inhibit proper enzyme systems, and lead to undesirable free-radical and oxidative functions.

A pilot study utilizing a unique oral detoxifying concentrate, DeTox Max, containing true disodium EDTA, microencapsulated in essential phospholipids microspheres was utilized as a provocation, detoxifying agent for a 16 patient pilot study. Significant quantities of heavy metals were excreted in a 48-hour collection versus each patient's baseline 24 hour collection.

The pilot study results confirmed substantial excretion of heavy metals. A surprising outcome of the study was the remarkable clinical healing and significant increase in brain acuity in patients that occurred within 2 weeks after only one vial was utilized, compared to previous pre - provocation.

Detox MAX Clinical Study Proposal

- **To assess the heavy metal detoxifying ability of Detox MAX, an oral detoxification agent containing 22 grams of essential phospholipids (EPL's); micro-encapsulating 1 gram of sodium endetate.**
- **To ascertain the most efficacious dose regimen of Detox MAX**

Heavy Metal Accumulation

- **Linked to increased free-radical activity and oxidation processes**
- **Can contribute to premature aging and aging diseases**
- **Can cause suppression and/or deregulation of the immune system, leading to a ten fold increase in cancer mortality** (Blumer, W. and Cranton, E.)
- **Suspected to contribute to learning disorders and neuro-degenerative diseases**
- **Increases risk of cardiomyopathy**
(Frustachi et. al.)

Ninety Percent Reduction in Cancer Mortality after Chelation Therapy With EDTA

Walter Blumer, M.D. and Elmer Cranton, M.D.

ABSTRACT: Mortality from cancer was reduced 90% during an 18-year follow up of 59 patients treated with Calcium-EDTA. Only one of 59 treated patients (1.7%) died of cancer while 30 of 172 non-treated control subjects (17.6%) died of cancer ($P=0.002$). Death from arteriosclerosis was also reduced. Treated patients had no evidence of cancer at the time of entry into this study.

Observations relate only to long-term prevention of death from malignant disease, if chelation therapy is begun before clinical evidence of cancer occurs. Control and treated patients lived in the same neighborhood, adjacent to a heavily traveled highway in a small Swiss city. Both groups were exposed to the same amount of lead from automobile exhaust, industrial pollution and other carcinogens. Exposure to carcinogens was no greater for the studied population than exists in most other metropolitan areas throughout the world. Statistical analysis showed EDTA chelation therapy to be the only significant difference between controls and treated patients to explain the marked reduction in cancer mortality.

Marked elevation of myocardial trace elements in idiopathic dilated cardiomyopathy compared with secondary cardiac dysfunction

Andrea Frustaci MD, Nicola Magnavita MD, Cristina Chimenti MD, Marina Caldarulo MD, Enrico Sabbioni PhD, Romano Pietra PhD, Carlo Cellini MD, Gian Federico Possati MD and Attilio Maseri MD

Received: 6/8/1998. Revised: 1/11/1999. Accepted: 1/21/1999.

Abstract

OBJECTIVES

We sought to investigate the possible pathogenetic role of myocardial trace elements (TE) in patients with various forms of cardiac failure.

BACKGROUND

Both myocardial TE accumulation and deficiency have been associated with the development of heart failure indistinguishable from an idiopathic dilated cardiomyopathy.

METHODS

Myocardial and muscular content of 32 TE has been assessed in biopsy samples of 13 patients (pts) with clinical, hemodynamic and histologic diagnosis of idiopathic dilated cardiomyopathy (IDCM), all without past or current exposure to TE. One muscular and one left ventricular (LV) endomyocardial specimen from each patient, drawn with metal contamination-free technique, were analyzed by neutron activation analysis and compared with 1) similar surgical samples from patients with valvular (12 pts) and ischemic (13 pts) heart disease comparable for age and degree of LV dysfunction; 2) papillary and skeletal muscle surgical biopsies from 10 pts with mitral stenosis and normal LV function, and 3) LV endomyocardial biopsies from four normal subjects.

RESULTS

A large increase (>10,000 times for mercury and antimony) of TE concentration has been observed in myocardial but not in muscular samples in all pts with IDCM. Patients with secondary cardiac dysfunction had mild increase (5 times) of myocardial TE and normal muscular TE. In particular, in pts with IDCM mean mercury concentration was 22,000 times (178,400 ng/g vs. 8 ng/g), antimony 12,000 times (19,260 ng/g vs. 1.5 ng/g), gold 11 times (26 ng/g vs. 2.3 ng/g), chromium 13 times (2,300 ng/g vs. 177 ng/g) and cobalt 4 times (86,5 ng/g vs. 20 ng/g) higher than in control subjects.

CONCLUSIONS

A large, significant increase of myocardial TE is present in IDCM but not in secondary cardiac dysfunction. The increased concentration of TE in pts with IDCM may adversely affect mitochondrial activity and myocardial metabolism and worsen cellular function.

Affiliations:

Department of Cardiology, Catholic University, Rome, Italy. Department of Occupational Medicine, Catholic University, Rome, Italy. Department of Cardiac Surgery, Catholic University, Rome, Italy. CEC Environmental Institute Joint Research Center Ispra (VA), Rome, Italy.

Free-radicals and Oxidation Processes

- **Heavy metals are known to catalyze free-radical activity and increase the aging process**
- **Heavy metals are used in chemical synthesis as a catalyst to enhance free-radical propagation**
- **Heavy metals catalyze oxidative processes**

Premature Aging

- **Systemic heavy metal and vascular decalcification treatments** can lead to anti-aging processes and enhanced cardiovascular (❤️ Heart) functions

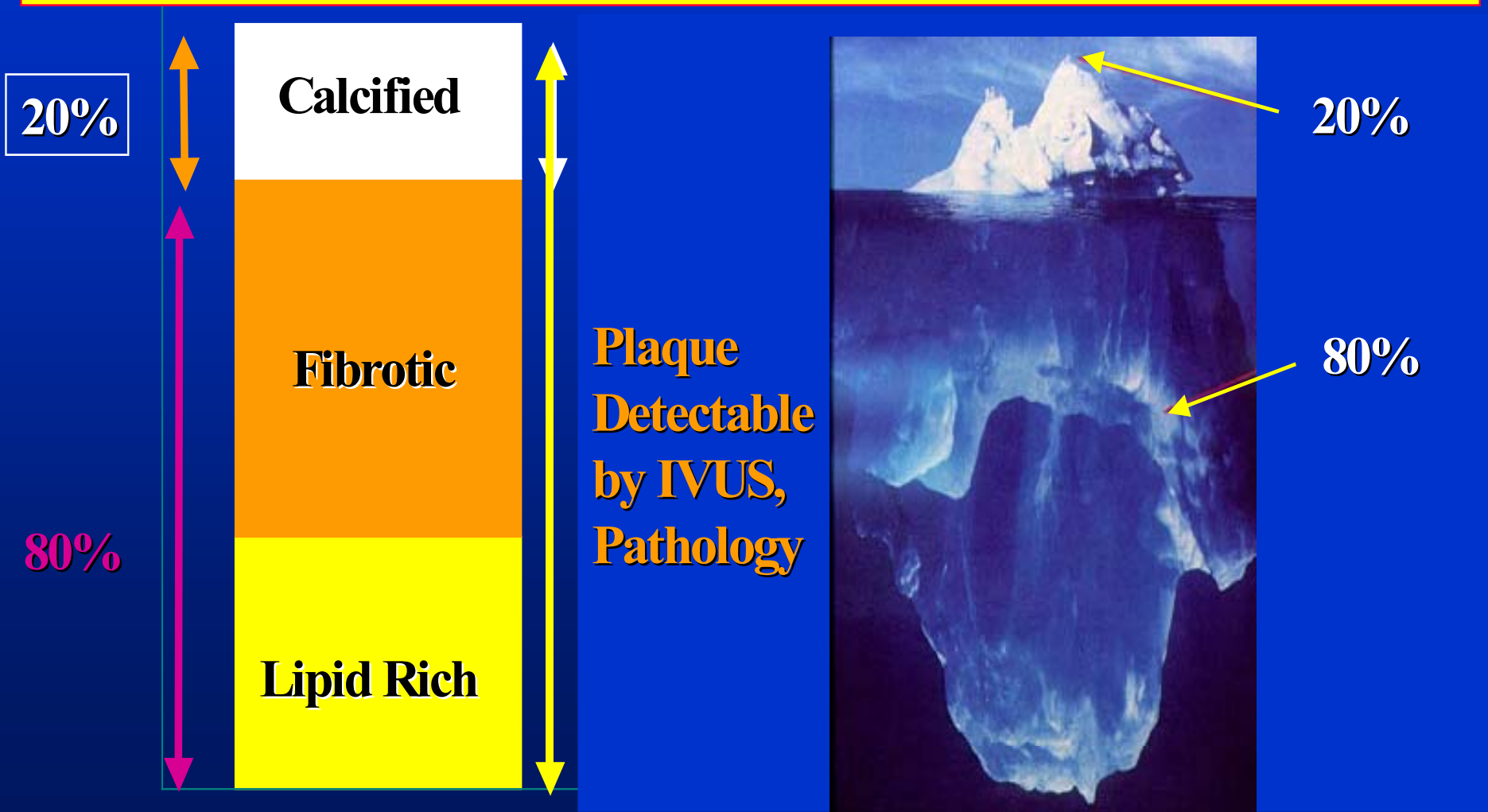
Immune System

- **Heavy metals can cause suppression and/or deregulation of the immune system leading to binding of receptor sites, inhibition of proper enzyme functions, decreased antibody response, improper hormone functions and undesirable up regulation of the immune system contributing to autoimmune disease**

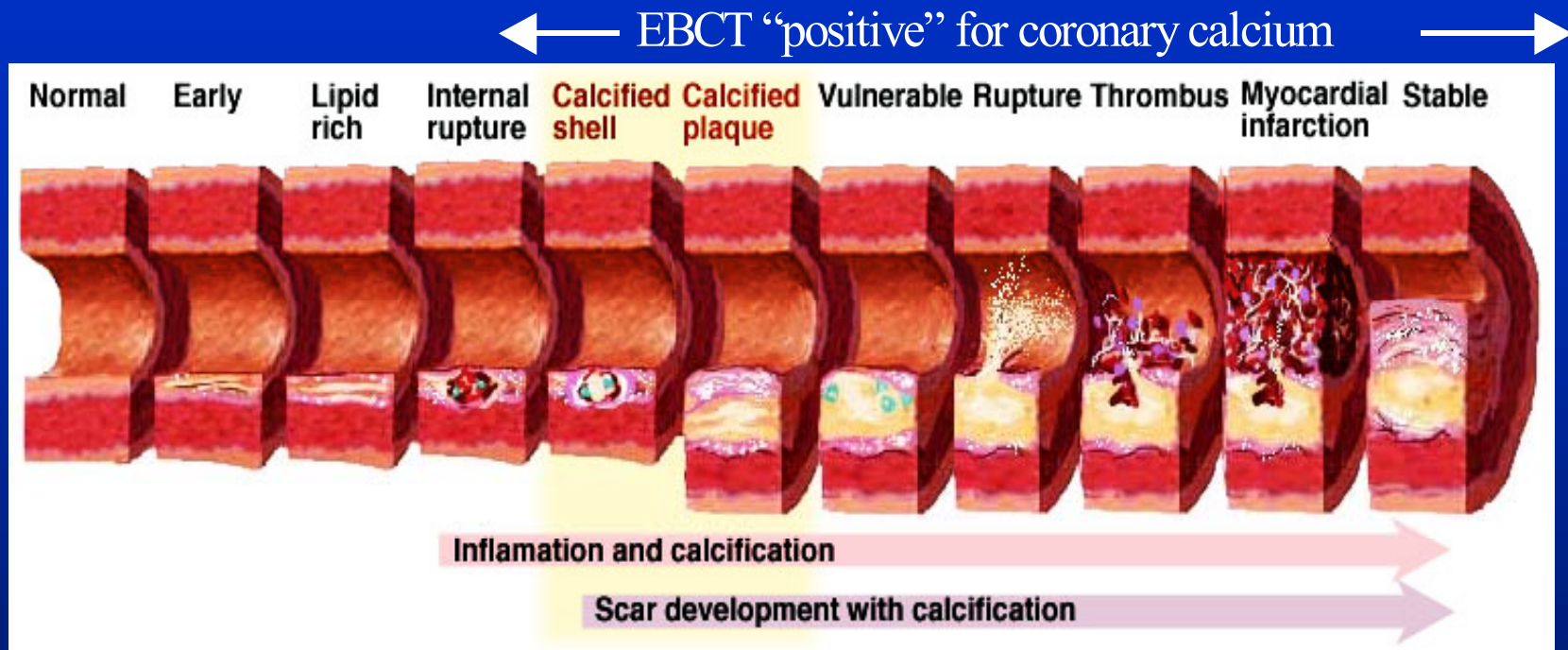
Learning Disorders and Neuro-Degenerative Diseases

- **Heavy metals, particularly aluminum and mercury can bind and accumulate in brain tissue**
- **Affects proper brain functions**
- **Has been found in high concentrations in degenerative neuro diseases such as Alzheimer's, Parkinson's, ALS (Lou Gehrig's disease) and Dementia**
- **Enhanced risk of Autism**

Total Coronary Artery Plaque and EBCT Coronary Calcium



Historical Development of a Coronary Artery Plaque



This process, in various stages of development, can be seen in many areas of the coronary artery system, consistent with the “diffuse” nature of coronary artery disease

CARDIOVASCULAR BENEFITS

(DeTox Max contains 22 grams of EPL's)

1 to 2 grams of Systemic Essential Phospholipids (EPL's)

Can promote (100+ clinical studies in Europe):

- **Decreased “Unstable” or Vulnerable Coronary Plaque**
- **Decreased mean total Cholesterol (-12% to -25%)**
- **Decreases in mean LDL (-34%)**
- **Increases in mean HDL (+50 to +100%)**
- **Reduction of serum triglycerides, Type II Diabetes (-37.7%)**

CARDIOVASCULAR BENEFITS cont.

- Significant lowering of cardiovascular risk ratio (-33+%)
- Decreased reactive platelet aggregation (-60%)
- Decreases of angina in Stage III & IV attacks or elimination
- Increased walking exercise tolerance with angina patients by as much as +900% before angina or nitro is needed

Essential Phospholipid Microsphere EDTA System

- Supplies microsphere encapsulated droplets of disodium EDTA in an essential phospholipid delivery system with no diarrhea symptoms and extremely high gastrointestinal assimilation to circulating bloodstream
- Slow release of EDTA systemically over a 48 hour + time duration, greatly decreasing possibility of overloading kidneys with heavy metal toxicity
- “True” plasma soluble ion exchange properties; thereby minimizing beneficial mineral excretion via unique delivery system

Clinical Protocol

- **Baseline 24 urine was collected for each of the 16 patients before provocation.**
- **Patients consumed 1 vial of Detox Max in 6-8 OZ. of fruit juice.**
- **Two successive 24 hr. urine collections.**

Protocol - Bedtime DetoxMAX

- **Empty bladder before taking DeTox MAX**
- **Take 1 vial of Detox MAX with 1 glass of cranberry or pineapple juice at bedtime (mixed thoroughly).**
- **Collect all urine after taking Detox MAX. Note the time of the 1st urine specimen and continue saving urine for the next 24 hrs. Put all urine in container labeled #1.**

Protocol - Bedtime DetoxMAX cont.

- **KEEP CONTAINER OF URINE ON ICE AT ALL TIMES!!!!**
- **After collecting urine for 24 hours, put container #1 on ice and begin collecting urine again in container #2 for the next 24 hours.**
- **Return container #1 to the clinic at 8:00 AM, keeping it on ice.**
- **Return container #2 to the clinic at 8:00 AM, keeping it on ice.**

Detox Max Study Results

	Increase Over Baseline 0-24 Hrs.	Increase Over Baseline 24-48 Hrs.	Maximum Increase Over Baseline Total 48 Hrs.
Aluminum	>100 %: 960 ug/24 Hrs	0 %	>>100 %: 960 ug/24 Hrs
Antimony	0 %	50 %	50 %
Arsenic	75 %	1483.30 %	1558.30 %
Beryllium	0 %	0 %	0 %
Bismuth	4967.70 %	1833.30 %	6801 %
Cadmium	133.30 %	100 %	233.30 %
Lead	2033.30 %	2300 %	4333.30 %
Mercury	0 %	312.50 %	312.50 %
Nickel	977.80 %	1011.10 %	1988.90 %
Platinum	>100 %: 0.8 ug/24 Hrs.	0 %	>100 %: 0.8 ug/24 Hrs
Thallium	100 %	200 %	300 %
Thorium	>100 %: 0.05 ug/24 Hrs	0.00 %	>100 %: 0.05 ug/24 Hrs
Tin	1380 %	600 %	1980 %
Tungsten	50 %	100 %	150 %
Uranium	>100 %: 0.09 ug/24 Hrs	0 %	>100 %: 0.09 ug/24 Hrs

Preliminary Patient Outcomes 1 vial of DeTox MAX

Patient # 3

**52 year old male with
pancytopenia/myelodysplasia**

- **Platelets ~35,000
before 1 vial of DeTox MAX**
- **Platelets ~54,000 1/21/2002
after 1 vial of DeTox Max**

Preliminary Patient Outcomes 1 vial of DeTox MAX

Patient #6

59 year old male with Cardiomyopathy, Diabetes Mellitus, long term Erectile Dysfunction, previous Myocardial Infarction and family history of Alzheimer's

1 week post treatment, patient's family notices significant memory and energy level increase with erectile dysfunction no longer a problem

Preliminary Patient Outcomes 1 vial of DeTox MAX

Patient #8

46 year old female with type II Diabetes, 4 Coronary Artery Bypass failure, Depression, **Virtually No Exercise Tolerance (20 feet) and daily substantial angina chest pains upon any exertion**

1 week post 1 vial treatment, mental capacity has increased and **patient is able to clean entire house without angina chest pains**

Preliminary Patient Outcomes 1 vial of DeTox MAX

Patient #16

75 year old male with Prostate Cancer, Hypertension, Coronary Artery Disease and Renal Insufficiency approaching a need for Kidney Dialysis Treatments

2 weeks post 1 vial treatment, he notices significant increase in daily energy, exercise tolerance, mental capacity and is sleeping better and his kidney functions have dramatically increased!

REFERENCES

1. Trowbridge, J.P., and Walker, M. *The Healing Powers of Chelation Therapy*, Stamford, Conn: New Way of Life, 1988.
2. Frackelton J. and Christensen, L., Mercury poisoning and its potential impact on hormone regulation and aging: preliminary clinical observations using a new therapeutic approach, *J. of Advancement in Medicine*, V. 11, No. 1 (spring 1980) p. 9-23.
3. Blumenthal, H.T.; Lansing, A.I.; and Wheeler, P.A. "Calcification of the media of the human aorta and its relation to intimal arteriosclerosis, aging and disease." *Amer. J. Path.* 20 (1944), 665.
4. Boyle, A.J.; Mosher, R.E.; and McCann, D.S. "Some vivo effects of chelation. 1: Rheumatoid arthritis." *J. Chronic Dis.* 16 (1963), 325-328.
5. Leipzig, L.H.; Boyle, A.J.; and McCann, D.S. "Case histories of rheumatoid arthritis treated with sodium or magnesium EDTA." *J. Chronic Dis.* 22 (1970), 553-563.
6. Schroeder, H.A. *The Poisons Around Us*. Bloomington, Ind.: Indiana University Press, 1974
7. Crapper-McLaghlan, D.R. "Aluminum toxicity in senile dementia-implications for treatment." Presented to the Fall 1981 meeting of AAMP.
8. Casdorff, H.R., and Walker, M. *Toxic Metal Syndrome: How Metal Poisoning Can Affect Your Brain*. Garden City Park, N.Y., Avery Publishing Group, 1995, p. 267.
9. Frustachi A. et. al., Marked elevation of myocardial trace elements in idiopathic dilated cardiomyopathy compared with secondary cardiac dysfunction, *J. Amer. College of Cardiology*, V. 33, 6 (May 1999), 1578-1583
10. Blumer, W., and Cranton, E., "Ninety Percent Reduction in Cancer Mortality after Chelation Therapy With EDTA", *J. of Advancement in Medicine*, Vol 2, No, 1/2, Spring/Summer 1989.
11. Schwartz G. and Reis L, Is Cadmium a cause of human pancreatic cancer?, *Cancer Epidemiology, Biomarkers & Prevention* 9, (Feb. 2000) p. 139-145
12. Hultber B. et. al., Alterations of thiol metabolism in human cell lines induced by low amounts of copper, mercury or cadmium ions, *Toxicology*, V. 126, 3 (April 3, 1998), 203-212.

REFERENCES Cont.

13. Bates MN et. al., Cancer incidence, morbidity and geothermal air pollution in Rotoru, *New Zealand, Int. J. Epidemiol.* V. 27, No. 1 (Feb 1998) p. 10-14.
14. Malmstron, B.G. "Role of metal binding in enzymic reactions." *Fed. Proc.* (Suppl. 10, 1961), 60-69.
15. Cave S., Autism and Mercury, Testimony presented before the Committee on Government Reform, U.S. House of Representatives, July 18, 2000.
16. Lamar, C.P. "Calcium chelation of atherosclerosis-nine years; clinical experience." Fourteenth Annual Meeting, American College of Angiology, 1968.
17. Brucknerova, O., and Tulacek, J. "Chelates in the treatment of occlusive atherosclerosis," *Vnitř. Lek.* 18 (1972), 729
18. Ohno, T. "Clinical and experimental studies of arteriosclerosis." *Excerpta Medica: Intern. Med.* 17 (1963), 8
19. Seven, M.J. (de.). *Metal Binding in Medicine*. Philadelphia: J.B. Lippincott Co., 1960.
20. Soffer, A. *Chelation Therapy*. Springfield, Ill.: Charles C. Thomas, Publisher, 1964.
21. Spencer, H. "Studies on the effects of chelating agents in man." *Ann. N.Y. Acad. Sci* 88 (1960), 435.
22. Clarke, N.E.; Clarke, C.N.; and Mosher, R.E. "Treatment of agina pectoris with disodium ethylene diamine tetraacetic acid." *Am. J. Med. Sc.* 232 (1956), 654-666.
23. Kitchell, J.R., et.al. "The treatment of coronary artery disease with disodium EDTA-A reappraisal." *Am. J. Cardiol.* 11 (1963), 501-506.
24. Lamar, C.P. "Calcium chelation of atherosclerosis-nine years' clinical experience." Fourteenth Annual Meeting. American College of Angiology, 1968.
25. Hancke C. and Flytine, K., "Benefits of EDTA Chelation Therapy in Arteriosclerosis: A Retrospective Study of 470 Patients" *J. of Advancement in Medicine*, Vol 6., No 3, Fall 1993.

REFERENCES Cont.

28. Nikitina, E.K., et. al. "Treatment of atherosclerosis with trilon B. (EDTA)." *Kardiologija* 12 (1972), 137.
29. Zapadnick, V.I., et. al. "Pharmacological activity of Unithiol and its use in clinical practice." *Vrach. Delo.* 8 (1973), 122.
30. Soffer, A. "Chelation therapy for arteriosclerosis." *JAMA* 233 (1975).
31. Meltzer, L.E.; Urol, M.E.; and Kitchell, J.R. "The treatment of coronary artery disease with disodium EDTA." In M.J. Seven, ed. *Metal Binding in Medicine*. Philadelphia: J.B. Lippincott Co., 1960, pp. 132-136.
32. Dugi, D. and Takemoto, A., Paper presented at the Third Annual BioImmune Physicians Clinical Protocol Workshop January 18, 2002, Scottsdale, Arizona USA.
33. Casellas Bernat, G. et al., *Clin. Med.* 15 (1975) 90-95.
34. Skorepa, J. et al., *Casopis Lekarů Ceských* 113 (1974) 784-786.
35. Tomasevic, M., Unpublished report no. 842 746.
36. Yasugi, T., *Japan J. New Rem. Clin.* 22 (1973) 691-693.
37. Horsch, A.K., *VASA* 15 (1986) 275-279.
38. Belousova, S.S. et al., *Kardiologija* 25 (1985) 112-115.
39. Izumi, H. et al., 11th Proceed. Japanese Atheroscl. Soc., Tokyo 1979.
40. Nakamura, H. et. al., *Japan J. New Rem. Clin.* 22 (1973) 1565-1574.
41. Stankovic, D. et. al., *Medicinski Arhiv* 28 (1974) 287-296.
42. Thurnherr, A., *Therapiewoche* 7 (1956) 116.

REFERENCES Cont.

44. Kirsten R. et. al., *Int. J. Clin. Pharm. Therap. Tox.* 27 (1989) 129-134.
45. Goto, Y., Nakamura, H., *Praxis* 55 (1966) 1235-1237.
46. Andreis, J., Report no. 842-669 of 1971.
47. Almazov, V.A. et. al., Lipostabil Symposium, Moscow, Nov. 1984.
48. Salvioli, G. et al., *Il Fegato* 21 (1975) 5-25 and 4th Int. Sympos. Atheroscl., Tokyo 1976 and Diab. Obes. Hyperlipoprot., Cupaldi V. et. al.(eds.) Academic Press: New York 1987.
49. Salvioli, G. et. al., *Gut* 19(1978) 844-850.
50. Almazov, V.A., Lipostabil Symposium Moscoe, Nov. 1984.
51. Almazov, V.A. et. al., Lipostabil Symposium Moscoe.