



Editorial Opinion

Is Modified Citrus Pectin an Effective Mobilizer of Heavy Metals in Humans?

Three small studies supporting the use of modified citrus pectin (MCP) for mobilization of heavy metals have recently been conducted by several closely linked authors. The most recent study, published in the July/August 2008 issue of *Alternative Therapies in Health and Medicine*, claims dramatic results in lead mobilization.¹ Given the far-reaching adverse effects of lead in humans, there is a great patient demand for safe, all-natural agents for effectively mobilizing lead. It is therefore incumbent on practitioners to closely scrutinize the recent study and the preceding two studies it references to assess the validity of the conclusion reached in all three studies; i.e., that modified citrus pectin is an effective mobilizer of heavy metals in humans.

The most recent study was conducted on children hospitalized for lead toxicity at the Children's Hospital of Zhejiang University, Hangzhou, China. Seven children were given 15 g MCP (Pectasol[®]) in three divided doses daily until discharge, which occurred after blood lead levels dropped below 20 mcg/L. MCP was administered until discharge to two children for two weeks, to three children for three weeks, and to two children for four weeks. A close analysis of this study raises a number of substantive concerns:

- ✦ Although the trial was conducted at a university hospital, there is no mention of the study's approval by an institutional review board (IRB).
- ✦ The study's criteria for inclusion and exclusion were not noted. Although the authors state the MCP product was used for other children not in the study, their results were not included because they did not fit the inclusion criteria.
- ✦ The study had no control/placebo group, although the article states the study was conducted at a hospital that works with lead-poisoned individuals where it is reasonable to assume a group control would be available.
- ✦ Aside from baseline blood levels, only discharge levels were reported. Presumably, weekly measurements were taken in order to monitor progress and determine when to discharge, but that data was not reported.

Several legitimate questions arise from the above-noted concerns. First, without a control group, there is no way to know if, in this population, the noted reductions of lead are attributable to the treatment protocol or to the average normal lead half-life in that population (the blood half-life of lead is an *average* of 30 days because different populations have different rates). Second, since the time-to-discharge ranged from 14-28 days, and the starting lead levels were similar, the question can be asked why did

the product not work more consistently? Third, if the authors usually treat lead-toxic individuals, why does the discussion not compare the effect of MCP to the normal reduction seen with simple avoidance of lead or to other lead treatment protocols? Fourth, as the current standard for human research requires IRB approval to ensure, among other things, participant safety, why was there no IRB approval?

Perhaps more troubling, however, is the undisclosed relationship of three of the study's seven authors to the company that makes the MCP product used in the study. Of the study's seven authors, only one is disclosed as being associated with the company that makes Pectasol. Two authors are listed as the president and the vice president of San Francisco-based Centrax International; however, a Google search was necessary to reveal that Centrax International (www.centraxinc.com) appears to be the distributor of Pectasol to the Chinese market. A fourth author is also the primary author of the two previous human studies on the use of MCP for heavy metal burden that are used as the support documentation for the current study. In none of these three studies, however, is it disclosed that this individual is also the president and founder of the company, EcoNugenics, that makes the MCP product tested in all three studies. Thus, while only one of the seven authors was disclosed as having a financial interest in EcoNugenics, the company that manufactures Pectasol, it appears that four of the seven actually do, thus raising the obvious issue of conflict of interest, particularly when such pertinent information is not disclosed.

Two of the three studies are published in the technical literature,^{2,3} while one is referenced in a book not available to this author.⁴ The first article,² published in *Phytotherapy Research* in 2006, is the only one to state it was approved by an IRB. It involved eight subjects who ingested two different dosages of Pectasol over a six-day period. The 2007 study, published in *Forschende Komplementarmedizin*,³ reports the cases of five individuals with heavy metal burden on whom a variety of pectin products (including Pectasol) and various standard chelating agents were tested.

For those interested, a more in-depth critique* of the 2006 and 2007 studies follows the References.

In this writer's opinion, what we are left with are three published studies that are replete with significant omissions and questionable math, and with no reliable evidence that modified citrus pectin is able to block the absorption of heavy metals or reduce heavy metal burden in humans. Unless and until such documentation is forthcoming, it may be to the benefit of your patients to use agents whose safety and efficacy in reducing human heavy metal burden is clearly, and more satisfactorily, documented.

References

1. Zhao ZY, Liang L, Fan X, et al. The role of modified citrus pectin as an effective chelator of lead in children hospitalized with toxic lead levels. *Altern Ther Health Med* 2008;14:34-38.
2. Eliaz I, Hotchkiss AT, Fishman ML, Rode D. The effect of modified citrus pectin on urinary excretion of toxic elements. *Phytother Res* 2006;20:859-864.
3. Eliaz I, Weil E, Wilk B. Integrative medicine and the role of modified citrus pectin/alginate in heavy metal chelation and detoxification – five case reports. *Forsch Komplementmed* 2007;14:358-364.
4. Eliaz I, Gaurdino J, Hughes K. The health benefits of modified citrus pectin. In: Patil BS, Turner ND, Miller EG, Brodbelt JS. eds. *Potential Health Benefits of Citrus*. ACS Symposium Series 936. Oxford, UK: Oxford University Press; 2006:199-210.

*In the 2006 study, eight subjects ingested two different levels of Pectasol over six days and had urinary levels of aluminum, antimony, arsenic, beryllium, bismuth, cadmium, calcium, copper, iron and lead measured. Three urine mineral tests were conducted. The first measurement was prior to starting the product, the second on day 1 of the protocol, and the final test on day 6. Quantification of the cases' baseline urinary heavy metals was reported clearly as the average of the heavy metal in mcg/24 hrs. The numbers for the second and third measurements were then reported as the numeric "difference from day zero." For example, the average baseline cadmium was 0.54 mcg/24 hrs. The day 1 level was noted as being 0.35 mcg/24 hrs higher than the average for baseline (meaning the average went from 0.54 to 0.89). The level listed for day 6 is only 0.16 mcg different from baseline (meaning the average on day 6 had dropped to 0.70 mcg/24 hr).

This method of reporting the difference from baseline instead of the actual average level makes for some confusion, which is compounded by the numbers in the body of the text not matching the data in the study abstract. The abstract states, "In the first 24 h of MCP administration, the urinary excretion of arsenic increased significantly (130%, $p < 0.05$)." The data presented, however, show that the average arsenic levels on day 1 were only 9.4 mcg/24 hrs greater than the baseline average of 26, a 36-percent increase, not a 130-percent increase. The abstract further states: "On day 6, urinary excretion was increased significantly for cadmium (150%, $p < 0.05$)." Yet, as noted above, the average level of cadmium on day 1 was 0.54 mcg/24 hr and the day 6 average was only 0.16 mcg/24 hr higher – an increase of 30 percent, not 150 percent as reported. The abstract also states, "Lead showed a dramatic increase in excretion (560%, $p < 0.08$)." However, the reported numbers show the average level of urinary lead on day 0 was 0.38, which increased by 0.82 by day 6 for a total of 1.20 mcg/24 hrs, an increase of 215 percent, not 560 percent as reported.

No dietary protocol was in place in this study to ensure that dietary sources of heavy metals were excluded during the test. Such exposures could readily account for increased levels of certain heavy metals in a 24-hour sample. There was also no mention of testing the MCP itself for heavy metals, which could contaminate the urine samples. And finally, there was no placebo group, or crossover administration of placebo to the study group for an equal amount of time, to determine if these findings were actually due to MCP or to something else.

The 2007 study has similar problems. This study reports the cases of five individuals with heavy metal burden on whom a variety of pectin products and standard chelating agents were tested. Such an expansive array of treatment protocols arguably minimizes the validity of the results.

The initial levels of heavy metals for three of the five cases were measured using proven heavy metal mobilizers. One individual had a post-CaEDTA urine toxic test and two had post-DMPS urine flush tests to determine the apparent level of heavy metals. None of the subjects had a random or non-provoked urine heavy metal test conducted before administration of CaEDTA or DMPS to ensure that the provoked lead and mercury levels were not due, at least in part, to recent exposures. Without a non-flushed baseline, one cannot know whether the person is dumping mercury because they have recently been eating swordfish or because their body burden is so great. Since CaEDTA is a remarkable chelator of lead from the bones, it is reasonable to assume that a portion of the post-flush burden is from historical body burden. A simple pre-flush test would have dispelled any doubts about the origin. The CaEDTA case had an initial post CaEDTA urine lead level of 92 mcg/g creatinine (at only 1,000 mg of CaEDTA), which was reduced to 47 mcg/g after taking MCP daily for two months. Most practitioners who use intravenous CaEDTA for heavy metal challenge testing use 50 mg CaEDTA/kg up to a maximum dose of 3,000 mg. A lead level of 92 mcg/g creatinine with only a 1,000 mg dose is unusually high and would indicate an immediate check for current exposures.

One subject who was given DMPS started with a urinary mercury level of 52 mcg/g creatinine, which was reduced to 22 mcg/g after taking a pectin product for seven months – a seemingly underwhelming reduction after seven months of treatment. The other DMPS case started with a very high urinary mercury level of 180 mcg/g creatinine, which dropped to 49 mcg/g after five months of pectin. The presence of such an unusually high level of mercury indicates this person was probably exposed to mercury by consuming a high-fish diet. If the subject had been eating fish regularly and then stopped after the first test, part of the reduction could easily be from normal mercury excretion rates; the serum half-life of methyl mercury is 70 days.

The other two cases had initial urine post-agent heavy metal tests conducted after ingestion of a pectin-containing product (not the MCP used in the other studies) that has never been validated to be an effective mobilizer of heavy metals. Since an unproven product was used for the initial test and no pre-flush urine heavy metal levels were determined, the initial urine levels were more likely due to current exposures and not reflective of total body burden. Without such standard protocols as pre- and post-testing and placebos in these studies, it is not possible to validate the results.

In both studies the discussion sections also contain statements about the effectiveness of pectins, alginates, and other products related to MCP that were either not referenced or were not substantiated by the given references.

Walter J. Crinnion, ND

Walter Crinnion, ND – 1982 graduate of Bastyr University; practice since 1982 with a special focus on treating chronic diseases caused by environmental toxic burden; conducts post-graduate seminars in environmental medicine; professor and Chair of Environmental Medicine, Southwest College of Naturopathic Medicine.
E-mail: w.crinion@scnm.edu