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 Supplement to
The Art of Getting Well
Molybdenum for *Candida albicans*
Patients and Other Problems

Sources are given in references.

Authors of contributions/quotations are alphabetically arranged; major author, if any, is underlined.

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ABSTRACT: The essential trace element, molybdenum, (pronounced mo-lib'de-num) is discussed in relation to its various metabolic pathways. Diagnostic approaches for molybdenum include applied kinesiological procedures based on strong muscles weakening when a patient sniffs aldehydes, ammonia, or Clorox, or tastes sulfur-containing amino acids. Other patterns indicating a need for molybdenum are the same as would be seen in a need for iron and/or excess of copper. Each of these metabolic pathways are shown to be important in the problems of the *Candida albicans* patient, as well as other patients. Protocols for supplementation and natural sources of molybdenum are given.

INTRODUCTION: Molybdenum is an essential trace element in human nutrition which is understood about as well as it is pronounced. In fact, there is no laboratory testing which has been standardized for the evaluation of molybdenum. Although it has been measured in both blood and hair, the normal values for these tests have yet to be established, and although it is accepted as an essential nutrient for humans, there has yet to be a recommended daily allowance or minimum daily requirement officially established. However, its importance in numerous patients, including those with *Candida albicans* allergy, has been paramount.

Molybdenum has been studied both directly in the blood and hair¹, indirectly by looking at other metabolites which relate to the presence of molybdenum^{2,3} and by applied kinesiological (A.K.) analysis by Dr. Richard Mowles⁴.

Molybdenum is necessary for the function of at least three important enzymes in the body: 1) aldehyde oxidase for our bodies' handling of aldehydes it produces and those encountered in the environment; 2) xanthine oxidase for the conversion of purines into uric acid; and 3) sulfite oxidase for the conversion of irritating sulfites into harmless sulfates. In addition, molybdenum is found in many biological processes in conjunction with iron and is found to cause a response in AK indicators similar to that of iron. Also, molybdenum is an antagonist to copper and vice versa. Considering all of the above factors has led to our understanding of how molybdenum is usually a necessary adjunct to the treatment of *Candida albicans* allergy patients and has speeded the recovery of most of these patients even above and beyond the effective natural procedures which were described in a previous paper by Mowles and this author⁵.

MOLYBDENUM AND ALDEHYDES: Chemical aldehydes are best known as fragrances. The body also produces various aldehydes as part of its normal metabolic pathways. One pathway in

the metabolism of the essential amino acid, threonine, is its conversion into acetaldehyde and then on into acetic acid for eventual production of acetyl coenzyme A.

THREONINE → ACETALDEHYDE → ACETIC ACID → ACETYL CoA

Figure 1

Acetaldehyde is a particularly toxic substance which, in addition to being produced from threonine and ethanol, is a product of the metabolism (i.e. fermentation) of carbohydrate in yeast -- hence, the *Candida* connection. Acetaldehyde is thought to be the major source of tissue damage in alcoholics rather than ethanol itself. The conversion of acetaldehyde into acetic acid is shown in Figure 2. Note that this reaction requires NAD (niacinamide), and aldehyde oxidase is dependent on FAD (riboflavin), iron (Fe), and Molybdenum (Mo).



Figure 2

Candida albicans patients and any other patients who complain of sensitivities to various fragrances and airborne odors will be found to have a problem with an olfactory challenge with an aldehyde and will be found to be in need of one or more of the nutrients associated with the metabolism of aldehydes, that is niacinamide, riboflavin, iron and/or molybdenum. Mowles⁴ had patients sniff a dilute source of formaldehyde and observed the results in muscle testing of patients. In 15 patient trials with weakening on smelling formaldehyde, 14 were found to strengthen on molybdenum. Our clinical procedure paralleled that of Mowles, although we used different sources of aldehydes.

Our original investigation involved using nail polish remover (Cutex® brand) as a source of acetone. We were attempting to use an olfactory challenge for ketones since transketolase enzyme is vitamin B₁ (thiamine) dependent. We had performed the functional blood test for red blood cell transketolase on a number of patients and found some of them to show a need for B₁. Knowing that nail polish remover is primarily acetone, we attempted olfactory challenging to observe the results. Eventually, we switched to using pure acetone for olfactory challenging because of the sporadic results we observed from using the nail polish remover.

A close look at the contents of the Cutex bottle revealed that besides acetone, a fragrance and a food color had been added. It is useful here to mention when testing with sniffing acetone, a strengthening of weak muscles or a weakening of strong muscles has been demonstrated to be associated with a need for vitamin B₁. The proper B₁ tablet, either high synthetic doses or low, natural source, or occasionally both, when insalivated, will block the weakening response to sniffing acetone in those patients who show it, and will likewise mimic the strengthening response in that group of patients.

Although we still occasionally use the nail polish remover as a screening test for acetone and B₁ involvement, we now know that many of our original sporadic observations were due to the presence of a fragrance (i.e., an aldehyde) in the product. We obtained a source of benzaldehyde, which is the smell of almonds and quite

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In the meantime, we had communicated with Mowles and found out about his results with formaldehyde. In an effort to find a less offensive odor, we opted for the benzaldehyde. Further searching led to a source of acetaldehyde itself, and that is now our first choice when challenging the aldehyde oxidase activity with olfactory testing.

When we initially screen a patient, we perform several olfactory challenges by having our patients sniff such substances as Clorox[®], acetone, an aldehyde, and ammonia, which will be discussed later. We observe for strengthening of weak muscles, and especially a weakening of strong muscles. In an aldehyde sensitive patient, there will be a generalized weakening of all of their muscles when they sniff an aldehyde. We have observed some patients who are sensitive to one aldehyde and not to another, and although this is uncommon, it is useful to keep more than one source of pure or diluted pure aldehyde present, for use in ruling out an aldehyde problem in a difficult patient.

When a patient weakens with sniffing an aldehyde, assume a problem with the aldehyde oxidase enzyme system. To further evaluate this pattern, check a muscle or muscles which are weak in the clear using oral insalivation of each of the four substances associated with the metabolism of aldehyde oxidase system: niacinamide, riboflavin, iron and molybdenum. We usually start with molybdenum because it is the most commonly found fact in our practice.

When molybdenum, or one of the other three substances, strengthens the weak muscles, we then have the patient again sniff the aldehyde with the neutralizing substance in the mouth. Sometimes, one of the nutrients will strengthen a weak muscle but will not negate the aldehyde sniff response. We only supplement the nutrient which both strengthens the weak muscle and negates the weakening response of the aldehyde sniff.

Patients with aldehyde sensitivity will demonstrate a number of symptoms. The most severe cases we have observed are those patients with systemic *Candida albicans* allergy syndrome. Many of these patients are incredibly sensitive to any type of fragrance. This becomes easily understood in light of the idea that *Candida* in the G.I. tract, vagina, or elsewhere in the body is giving off acetaldehyde as part of its normal metabolism. The excess stress which this must put on the aldehyde oxidase enzyme systems in the body's tissues leaves them unable to keep up with the extra demand. Supplementation of molybdenum and/or niacinamide, riboflavin, and iron will improve the patient's ability to handle the *Candida* generated aldehydes, as well as those encountered in the environment.

When an aldehyde sensitivity exists, there is a considerable tissue irritation due to the buildup of these substances. It appears from our clinical observations that some patients fall into a vicious cycle of aldehyde sensitivity where there is a depletion of one or more of the aldehyde oxidase related nutrients which leads to an increase tissue irritation from *Candida* produced aldehyde. This tissue irritation lowers tissue resistance which sets up vulnerability to future invasion by the *Candida albicans* or other infectious agents reinitiating the cycle over again. This pattern often accompanies the "pseudoinfection syndrome" previously discussed by this author⁶, and appears to be responsible, at least in part, for many patients with chronic vaginitis from a yeast infection.

We have observed at least one patient who has been plagued with recurrent vaginal yeast infections, at least once monthly, for many years. Every possible allopathic approach has been unsuccessful in controlling the recurrence of these infections. Mycostatin (Nystatin) as vaginal suppositories has been useful in controlling the acute infection, but the response is slow and has no effect on recurrence. We suspected systemic *Candida albicans* involvement,

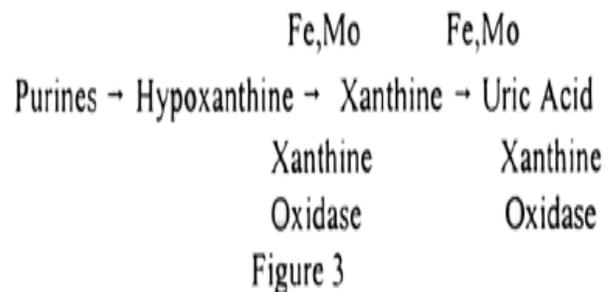
but this patient showed none of the characteristic patterns which we rely on for diagnosis of this syndrome⁷. She will be able to return to normal tissue resistance following infection and be able to ward off further infections.

The patients with aldehyde sensitivity have complained of many symptoms. Some of these symptoms seem to be directly related to the aldehydes and others from different sources. It is difficult to differentiate which symptoms are aldehyde related in most patients, but the most dramatic responses from controlling aldehyde sensitivity have been in *Candida albicans* patients, on whom everything else had been tried. The addition of molybdenum, based on aldehyde olfactory sensitivity muscle testing has resulted in turnarounds of our most difficult *Candida albicans* patients. The most noticeable changes are seen in the sore, achy, sluggish, "flu-type" symptoms of which many *Candida* patients complain. The energy returns, the generalized musculoskeletal achiness improves, and mental sluggishness disappears, and sinus and nasal congestion clears up. Based on these observations on difficult patients, we now screen every *Candida albicans* patient with the aldehyde sniffing test and take appropriate measures sooner rather than later in these patients. The addition of molybdenum, in particular, has been a great boon to us in handling the *Candida* patient, and getting them out of whatever rut or vicious cycle the aldehyde sensitivity has put them into. Molybdenum is also important in caring for a number of other metabolic problems associated with the *Candida* patient, as well as other patients.

MOLYBDENUM AND AMMONIA METABOLISM:

Mowles' study included olfactory challenging with ammonia in addition to formaldehyde⁴. There are many facets to olfactory challenging with ammonia which have been discussed by this author in his seminars and are the source of future papers⁷. Mowles chose to focus his study on the relationship of ammonia weakening response to the strengthening response from molybdenum. In twelve patient trials where the patient weakened on sniffing ammonia, seven of these patients were found to have weak muscles strengthen with insalivation of molybdenum.

Molybdenum is necessary for the function of xanthine oxidase enzyme. Iron is also necessary for the function of this enzyme. Xanthine oxidase converts hypoxanthine into xanthine and then converts xanthine on into uric acid. (See Figure 3.) These reactions are essential in the metabolism of purines. It follows then, that patients with low serum uric acid levels should be checked for a need for molybdenum (and/or iron).



Uric acid is excreted in the urine and is one way in which the body may rid itself of ammonia. In Figure 4, one can see that each molecule of uric acid contains four nitrogen molecules. Since the body can synthesize purines from amino acids, carbon dioxide, ammonia, and formate, a lack of xanthine oxidase function could presumably cause a back-up in the metabolism of purines and block one pathway of utilization of ammonia. The ammonia from amino acids which would otherwise be used in the synthesis of purines

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appears that in a molybdenum requirement, the body's ability to metabolize methionine is blocked. This may be seen in amino acid profiles by an elevated methionine¹. This would make sense in the case of a magnesium deficiency as can be seen in Figure 6. But in the case of a need for molybdenum we would also expect to find a weakening from cysteine since the role of molybdenum is played beyond cysteine in the metabolic flow chart. We only occasionally see weakening from cysteine in these patients. We occasionally see strengthening from cysteine or no response from cysteine in these patients, but we are surprised at the infrequency with which we observe cysteine to cause weakness in these patients.

Some of these patients will show a strengthening response to taurine. Taurine has been discussed in an earlier paper by this author in its function as a free radical scavenger for the free radical OCl (hypochlorite ion or hypochlorous acid.)⁶ In light of a molybdenum requirement, we have observed some patients who weaken on sniffing Clorox who are taurine responders and who also have this weakening response negated by molybdenum insalivation. Although this does not make sense based on the biochemistry presented, there must be some sort of negative feedback mechanism when sulfite oxidase is unable to metabolize sulfites. This feedback must affect the conversion of methionine, causing it to back up, thereby causing the weakening response to methionine we observe in some patients with a molybdenum requirement.

In a few difficult patients who fall into the "fast oxidizers" or "over-oxidized" category, we find a generalized muscle weakness pattern when we first examine the patient. The use of the antioxidants glutathione in combination with selenium has caused strengthening in a number of these patients who did not respond to water or a multiple vitamin or mineral nutrient as recommended by Goodheart¹¹. These patients always have symptoms of free radical pathology and are among the most difficult we see. Because of the nature of cysteine being a part of the tripeptide, glutathione, and because of our occasional observations of patients requiring cysteine for production of taurine for OCl free radical quenching, we attempted to check two of these multiple weakness patients with a combination of molybdenum (in place of glutathione) with selenium. We have seen a combination of these two minerals cause a generalized strengthening effect in two multiple weakness patients. One of these patients also responded to a glutathione-selenium combination and the other one responded only to the molybdenum-selenium combination.

Candida albicans allergy [may be] due to the chronic, long term stimulation of the immune system, free radical release, and antioxidant depletion. Imagine the chemical stress a patient's tissues must be under when constantly exposed to free radicals, sulfites, acetaldehyde, and ammonia, all of which have accumulated in the tissues due to an unmet molybdenum requirement of the patient. Add to this the other factors involved in the *Candida* patient and it is easy to see why *Candida* can be difficult to treat, and why molybdenum can be such a great help in treating these patients.

MOLYBDENUM AND IRON METABOLISM: In 1981, this author reported the common observation of iron neutralizing retrograde weakening of patients' muscles¹². Mowles observed that many retrograde type patients responded to molybdenum either in conjunction with or separately from iron⁴. Molybdenum plays a synergistic role with iron in the body. Clinically we are observing that any time one might use iron, one may also find a need for molybdenum.

Molybdenum and iron are related in a number of biological processes other than xanthine oxidase and aldehyde oxidase. In plants, there are iron-molybdenum dependent nitrogenase enzymes, for example, as well as several other areas which Mo and iron work together in important roles¹³. In humans, the highest concentrations

of molybdenum are found in the liver, kidney (where xanthine oxidase is present) and the adrenal glands. High concentrations are also found in bone and skeletal muscle although, at present, Mo's functions in these tissues are unknown¹³.

We have found muscle testing indicators for Mo to be the same as for iron. This is especially true when a patient has previously shown an A.K. need for iron and still has the same indicator for iron despite supplementation. These patients usually also respond to Mo. We speculate that, for whatever reason, possibly molybdenum's antagonist role to copper, to be discussed in the next section, Mo enhances many of iron's roles. This might include iron's hematopoietic role and its role in myoglobin in muscles. Perhaps, these are some of Mo's unknown functions in bone and skeletal muscle.

Identifying a need for molybdenum has solved several curious cases of apparent iron deficiency anemia when patients were non-responsive in spite of iron supplementation. We now consider a potential for molybdenum whenever we observe a lowered red blood cell count, lowered hemoglobin, lowered hematocrit, or lowered mean cellular hemoglobin (MCH). We have not seen enough of these patients to prove this relationship, but our anecdotal experience is enough to make us suspect a need for molybdenum any time we suspect or find a need for iron. We have also observed Mo cause a response in the case of aerobic metabolism through myoglobin formation. Molybdenum has occasionally been seen to neutralize these aerobic muscle testing weaknesses in some of our musculoskeletal patients.

Candida albicans is an anaerobic organism and therefore grows more readily in an anaerobic environment. Anything which interferes with the ability of the body to carry oxygen to its tissues may enhance the growth of *Candida*. Molybdenum's apparent functions paralleling those of iron may aid in producing a higher level of tissue oxygenation and thereby decrease the anaerobic environment in which *Candida* flourishes.

MOLYBDENUM AND COPPER: Molybdenum and copper are antagonists. Just as iron and molybdenum are synergists, iron and copper are antagonists if they are not in balance with each other, so are molybdenum and copper antagonists. In patients who demonstrate copper toxicity patterns, we have traditionally employed zinc and manganese to chelate the excess copper out of the tissues. Molybdenum is also useful for this purpose. Molybdenum antagonizes copper absorption and in a number of experiments in animals, copper and molybdenum have been shown to be directly antagonistic to each other.

Mowles and I have observed an extremely high correlation in our practices of copper toxicity in patients with *Candida albicans*⁵ as well as in women with menstrual and premenstrual disorders from functional hormonal imbalances¹⁴. It is possible that our geographical location (R.M.-Roanoke, Virginia, and W.S.-Chapel Hill, North Carolina) is responsible for this correlation. However, we find such a consistent pattern of low iron-high copper that we suspect copper's antagonism of iron and the resultant tendency toward an anaerobic environment for the yeast to grow as a major factor in these patients, as previously mentioned. [Editor's note: in the brewing industry, replacing copper vats with another metal resulted in brewer's yeast being unable to grow, or ferment barley- rice into beer.]

Occasionally, we see a patient who has *Candida* and is low in copper. This seems contradictory to our other findings unless it is remembered that copper plays an essential role in working with iron when they are in balance. As we have observed a need for Mo in some iron non-responsive iron deficiency type anemias, others have reported a similar pattern of copper deficiency. Too much or

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When we identify a copper toxic patient, we look very closely for any indication to give molybdenum. This includes any of the above parameters or simply testing any gamma-2 muscle weakness. Gamma-2 (patient-started) weakness implies systemic chemical imbalances which are monitored by supraspinal (e.g. hypothalamus) levels¹⁵ and seem to be involved in all cases in which we find a need for molybdenum.

SOURCES AND DOSAGES OF MOLYBDENUM: The average adult intake of molybdenum in the U.S. has been variously reported as 350 mcg and between 120 mcg and 140 mcg¹³. When a patient requires molybdenum based on one or more of the parameters mentioned in this paper, it is always our policy to supplement the patient with molybdenum for a period of time. We initially recommend 300 mcg. per day and continue this level for one to two months, depending on the patient's clinical response. We always insist that the patient taste (chew or suck) the supplement to activate the taste bud receptors which have direct input to the hypothalamus. After the first month or two, and when the patient's condition has shown improvement, we decrease the dosage to 200 mcg per day until the patient has taken the supplement for a total of four months.

The length of time to supplement a nutrient can often be based on clinical judgment and symptom response. In the case of molybdenum (and a number of other nutrients, especially those which have a relationship to red blood cells), however, we always maintain supplementation for a period of at least four months. This is based on the suggestion of Dr. George Miroff¹⁵ who recommends that any nutrient which is associated with hematopoiesis be taken for a long enough period of time that each RBC in the body gets its full share. In other words, since the life of a RBC is 120 days, it is necessary to take these nutrients for at least 120 days to ensure that the entire blood supply has had the advantage of this nutrient.

We have observed a number of patients who took molybdenum (or other nutrients which aid in RBC production and/or are taken up by the RBC's only during hematopoiesis) for one or two months and became asymptomatic, only to have the symptoms return one or two months later after stopping the supplement. Starting at 300 mcg and gradually reducing the dosage over four months time period has proven successful in our practice. In some difficult patients, we continue supplementation as long as they are symptomatic at a 100 to 200 mcg level.

It is important to note that in cattle, molybdenum excess has been shown to decrease fetal growth¹. Although no studies have indicated this in humans, we usually stop molybdenum supplementation in our pregnant patients, just to be on the safe side. If the obvious need for molybdenum returns, we will again supplement this nutrient, but at 100 mcg or less as a precaution. Since there is no recognized standard for Mo need, we must rely on good clinical judgment regarding dosage.

After a period of four months, we stop Mo supplementation if the patient is asymptomatic. We recommend that the patient consume more Mo containing foods, if they are not already doing so. Foods which are high in molybdenum are shown below in order of their molybdenum concentrations¹.

Grains: Buckwheat, wheat germ, barley oats.

Vegetables: lima beans, canned beans, soybean meals, lentils, green beans.

Liver and sunflower seeds are also high in molybdenum.

Since adding the diagnostic protocols for Mo in our office, we have found scores of patients who showed a need for this essential trace element. The remarkable response in symptoms in patients with *Candida albicans* as well as other difficult patients has caused us to check each new patient for this mineral. Molybdenum is beginning

to creep its way into multiple nutrient formulae by many companies, but the need for the addition of specific Mo supplementation is still a common finding even in patients on these multiple nutrient supplements. With the growing awareness of *Candida albicans* and other metabolic disturbances which relate to the molybdenum pathways, it is felt that a significant percentage of your patients may be helped in their recovery by identifying the need for and supplying the essential, but little known trace element, molybdenum.

SUMMARY OF MAJOR CLINICAL INDICATORS FOR MOLYBDENUM:

1. Muscles weaken on sniffing an aldehyde (e.g. acetaldehyde, formaldehyde, benzaldehyde, etc.) and insalivation of Mo neutralizes this weakness.

2. Muscles weaken on sniffing Clorox (hypochlorite) and/or tasting methionine and/or cysteine and insalivation of Mo neutralizes this weakness.

3. Muscles weaken on sniffing ammonia and insalivation of Mo neutralizes this weakness.

4. Whenever iron is indicated, Mo might also be indicated, such as in: aerobic testing muscle weakness patterns; retrograde position weakness patterns, low hemoglobing, RBC count, hematocrit, or MCH.

5. Whenever copper causes muscle weakness, Mo may be necessary to chelate out excess copper. Also discussed: If strong muscles weaken or weak muscles strengthen on sniffing acetones, this indicates a need for B₁.

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* Pronunciation of molybdenum is mo-lib-de-num.

Table 1:
Molybdenum Intake, ESADD, Blood, Food Sources, Benefits

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Molybdenum is not abundant in the Earth's crust or in seawater. Average soil contains only 1.5 mg per kilogram. Normal seawater contains only 0.1 mg per liter. Drinking water can contain more or less at 0-6.2 mg/liter, and mineral spring water contains 2.31-3.31 mcg per liter, less than tap water.

Molybdenum is present in all bacterial, plant and animal tissues since 3 ubiquitous enzymes necessary for life require molybdenum for enzymatic processes. Because of that, the burning of fossil fuels derived from plant/animal material puts molybdenum in the air and water, affecting our daily intake. Urban air can contain as much as 10-30 mcg per cubic meter of air while rural air contains much less at 0.1-3.2 mcg/cubic meter. In steel and molybdenum roasting plants, workers can be exposed to as much as 1-19 mg of molybdenum per cubic meter of air, which is associated with gout-like symptoms, unless protective breathing masks are worn (Lener & Bibr, 1984).

Molybdenum Intake and Excretion

Category	Age	Copper mg	Intake Mo mcg	Excretion Mo mcg
Food	US adult	3-5	Intake 335	
Water	US adult		2.8	
Air	US adult		0.1	
Urine	US adult			190
Feces	US adult			125
Perspiration	US adult			20
Total			337	335
All Sources	UK Adult		94-162	No data
	Hospitalized Patients		250-1,000	No data
Normal Values	All Ages		210-460	No data

Dietary factors influencing intake: Supplemental copper and molybdenum taken at the same time combine and are excreted in the urine. Sulfur reduces absorption of molybdenum by blocking protein carriers and increasing excretion of molybdenum at 400-500 mg/l of water, used in Russian metallurgy plants to prevent Mo intoxication where excessive amounts are present in the air. High protein diets block absorption of molybdenum and enhance excretion. On the other hand, high carbohydrate diets enhance absorption and retention of molybdenum. (Roschik & Lukashev, 1978; Lener & Bibr, 1984; <http://www.imoa.org.uk/imoadata/THERAPY.HTM>)

National Research Council Estimated Safe and Adequate Daily Dietary Intakes 1989

Category	Age	Mo mcg	Copper mg
Infants	0-0.5	15-30	0.4-0.6
	0.5-1	20-40	0.6-0.7
Children and Adolescents	1-3	25-50	0.7-1
	4-6	30-75	1.0-1.5

7-10	50-150	1.0-2.0
11+	75-250	1.5-2.5
Adults	75-250	1.5-3.0

Blood Plasma Values (Lener & Bibr, 1984)

US Population	5.0-157.3 ng/g
England, males	0.08 ng +/-0.3 ng/g
Females	1.2 - 0.5 ng/g
Blood donors	1.8 ng/g
Hospital Personnel	3.4 ng/g
Patients with degenerative diseases	1.2 ng/g
Patients undergoing treatment	7.2 +/- .3 ng/g
Pregnancy, 12-19 weeks	129.5 ng/g
Pregnancy, labor	338.5 ng/g

Food Sources

Even though all foods contain atomic amounts of molybdenum since all cells require molybdenum in enzymatic processes, few foods contain substantial amounts of molybdenum that affect dietary intake significantly. Diets emphasizing meat and other animal sources produce low dietary intakes, with the exception of milk product diets. Good sources are milk products, the leafy portions of vegetables but not the root, legumes, cauliflower, liver, and whole grains. One Danish study found that the only food intake enhancing blood levels of molybdenum was butter. Tea contains trace amounts, coffee none. Distilled alcoholic beverages contain none; beer, wine, and Scotch contain trace amounts. A theoretical diet where the staples might consist largely of legumes and leafy vegetables could lead to excessive intake of molybdenum. Soil concentrations and conditions affect the levels found in foods (Lener & Bibr, 1984). Organic vegetables contain 50% to 1000% more trace minerals than conventionally grown produce. Synthetic fertilizers discourage the mineralization of plants and lower macronutrient quality. Organic food can be 8 times as high in macronutrients, according to one random sampling in a Chicago study; twice as high in other studies (Cooter, 1995; Heltman, 1997).

Leaf vegetables	2-26 mcg per kilogram
Legumes	1,000-6,080 mcg per kilogram
Cauliflower	1,150-2,260 mcg per kilogram
Liver	200 mcg per kilogram
Whole grains	200-485 (Buckwheat is the highest; barley medium; corn lowest)

Potential Benefits of Molybdenum Enriched Soils and Diets

(<http://www.imoa.org.uk/imoadata/physi/MOLY.HTM> & / [BIOCHEM.HTM](http://www.imoa.org.uk/imoadata/BIOCHEM.HTM); Lener & Bibr, 1984; Roschik & Lukashev, 1978; Schmitt, 1991; Cooter, 1993)

High molybdenum levels in soil

Lower cavities (caries) incidence in populations of England, Columbia, New Guinea, South Africa, Hungary, Ohio
150-200 mg molybdenum per liter of water
Caries inhibition in rats

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Soil Molybdenum enrichment, possibly the supplementation of vitamin C and Mo in humans; Mo supplementation in rats & mice

Lower incidence of esophageal cancer, dysplasia, and mortality rates; lowered carcinogenic nitrosamine content in plants; protective against nitrosamines in rats; reverses throat cancers in rats; cures mammary tumors in mice; reduces GI-tract/rectum cancer incidence in animals

Adequate molybdenum

Stronger bone formation

Enhancement of phosphorous metabolism: decreased excretion of calcium & phosphorous, increased deposition in bone; enhances retention of potassium & iodine in plasma, but may decrease uptake of iodine by thyroid

Molybdenum supplementation (amounts to be determined by orthomolecular physician, average 100-300 mcg/day for 3 months, gradually reduced and replaced by food sources)

Enhances enzymatic activity of xanthinase, nitrogenase, alcohol dehydrogenase, detoxifies sulfites, enhances antioxidant uric acid production, protects against aldehyde/aromatic chemicals such as perfume and formaldehyde, alleviates premenstrual symptoms caused by copper overload, alleviates symptoms of candidiasis

Molybdenum supplementation

Synergistic with iron in red blood cells and helpful in resolving iron deficient anemias that do not respond to iron alone

Molybdenum supplementation at high experimental levels in mice for short periods of time

Reverses diabetes, normalizes blood sugar levels, restores defective insulin production, enhances coenzyme A activity in diabetic mice

Molybdenum supplementation and experimental tetrathiomolybdate chelation therapy

Protects against mercury, lead, cadmium, copper poisoning in animal studies, may reduce zinc levels

Tetrathiomolybdate, experimental chelation therapy

Effective cure for Wilson's Disease showing neuropsychiatric symptoms in human studies and LEC rats without side effects or neurological degeneration

Molybdenum soil enrichment

Enhances *Axobacter chroococcum* & other soil bacterial fixation of nitrogen

Produces lush green productive plants at normal levels; cures yellows and curbits; excessive liming can inhibit plant growth and produce copper deficiency in ruminant animals, moose, deer, cattle, sheep, and horses

Personal note: I was hospitalized for pneumonia and partial kidney failure for six days in May 2001. The consequence of being given one steroidal drug induced diabetes in me for the duration of the treatment. I had been given insulin shots twice a day to compensate. On the second day, I took one 100 mcg tablet of molybdenum to see if would help. My blood sugar levels normalized each day of three days I ingested one 100 mcg tablet, and the insulin shots were discontinued. This may have enormous practical application to diabetics who must monitor their blood sugar levels and take insulin shots.

Table 2:

Molybdenum Deficiency and Toxicity

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Molybdenum deficiency has not been found in free living human beings, but has been encountered in patients undergoing long-term parenteral IV nutrition unless 350 mcg of ammonium molybdate has been included in the nutritional formula. Deficiency symptoms include tachycardia, headache, mental disturbances, and fi-

nally coma (Sardesai, 1993).

There are also very rare instances of children born with defective molybdenum dependent enzymes, sulfite oxidase and xanthine dehydrogenase. Only 3 of such instances have been reported. This condition is characterized by infantile seizures, myoclonus, abnormal muscle tone, spastic symptoms, facial shape abnormalities, ocular lens abnormalities, mental retardation, brain atrophy, and homocystinuria. Life expectancy is very short unless the defective gene lacking a molybdenum cofactor is only sulfite oxidase and does not include xanthine dehydrogenase. A low sulfur diet has been promising in one case of defective sulfite oxidase enzymes. Otherwise, such children do not survive, and supplemental molybdenum does not change the outcome, nor have other attempts at treatment affected the outcome (Endres et al., 1988; Shih et al., 1977).

Molybdenum has low toxicity for humans and is rarely found outside of the former USSR mining and metallurgy industries where protective breathing masks were not worn to prevent the inhalation of molybdenum containing dusts and fumes. A similar circumstance has been reported for artists in metal sculpture who use steel rods in their work. Rare instances of gout-like symptoms and headaches have been reported in artists who do not wear respiratory protection and ventilate their working area (Lesser & Weiss, 1985). Also, patients who must undergo long-term hemodialysis treatments experience dialysis caused arthritis from excessive molybdenum after many treatments (Hozokawa, 1994).

There are also rare instances of molybdenum toxicity for ruminant animals, cattle, sheep, horses, moose, and deer, which have a higher requirement for copper intake and a lower requirement for molybdenum. This condition is known as molybdenosis or copper deficiency. In humans, copper deficiency has never been encountered, including areas where the soil and food are low in copper content (Roschik & Lukashev, 1978).

However, there are two towns in Armenia where the soil contains excessive molybdenum and dietary intakes from the food grown in the region average 10-15 mg per day of molybdenum. In this circumstance, gout-like symptoms are also prevalent in 18-31% of the adult population after life-time exposures to these high levels. In test areas where the copper intake was also high, this condition does not develop. In India, where the sorghum staple consumed contains 1.5 mg of molybdenum dietary intake per day, children growing up in this area develop genu valgum, knock-knees, a bone deformity, after years of intake. The soil in the region also contains unusually high amounts of fluorine, which contributes to the problem (Lener & Bibr, 1984). In other areas where molybdenum daily intake is as much as 350 mcg, this condition does not develop.

Molybdenum Levels in Populations Studies

Influencing factors: Supplemental copper and molybdenum taken at the same time combine and are excreted in the urine. Sulfur reduces absorption of molybdenum by blocking protein carriers and increasing excretion of molybdenum at 400-500 mg/l of water, used in Russian metallurgy plants to prevent Mo intoxication where excessive amounts are present in the air. High protein diets block absorption of molybdenum and enhance excretion. On the other hand, high carbohydrate diets enhance absorption and retention of molybdenum. (Roschik & Lukashev, 1978; Lener & Bibr, 1984; <http://www.imoa.org.uk/imoadata/THERAPY.HTM>).

Molybdenum	Normal	Deficient	Toxic	Result	Remedy
Soil	0.2-5mg/kg	>0.2mg/kg Linxian, South Africa, high throat		Normal: Healthy plant	Soil enrichment; Supplementation

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		cancer		
Armenian soil: 2 towns		Soil: 77mg/kg Diet: 10-15 mg/day	Gout-like symptoms, bone deformities, Edema, skin flushing	Higher copper intake
India	Normal sorghum 350mcg/d	Excess Mo in sorghum 1.5 mg/d	Knock-knees; genu Valgum	Lowered Mo Intake: no more than 350 mcg in children
New Zealand lambs	0.03 ppm in soil		Xanthine Disease, renal calculi	Mo soil enrichment
Australia, US, Chicks	Deficient Mo in feed		Impaired bone/cartilage formation	Feed supplement 0.5-2.5 Mg/kg
Sweden, Somerset, Kern Co. cattle, horses, sheep, moose		6 mg/kg soil from fly ash, industrial pollution, excessive liming	Teart, diarrhea, weight & hair loss, depigmentation, sterility	Supplemental addition of copper to feed
Azerbaijan, lambs		2.5x> Mo/Cu ratio	15-39% enzootic ataxia	Reversed ratio of Cu to Mo
Swiss Alps, Engadin, lambs		30 mcg/l of water	Malmagliar: weight loss, anorexia, diarrhea	Supplemental copper addition

Precautionary Labels and Warnings

The International Molybdenum Association (IMO) has conducted extensive tests on molybdenum toxicity for bacteria, algae, fish, and rats and found that all toxicity levels are above levels that the European Union requires precautionary labels and warnings for. You can easily access the data at: (<http://www.imoa.org.uk/hse.htm>) or at (<http://www.imoa.org.uk/>) and click on "Health, Safety & the Environment." For humans, it would take many cases, not bottles, of supplements at one time to produce acute toxicity. According to Pfeiffer (1978) the only consequence of a possible massive dosage at one time would be increased tension in the muscles of back, hip, and thighs.

High doses of 1.5 milligrams per day of molybdenum supplements have been taken by US adult males for a period of 24 days without adverse consequence. The study concluded that it was safe to take such an amount for a period equal to or greater than 24 days (Turnlund, 1995). It should be emphasized this study was of adult men and did not include children at this dosage level, nor did it include pregnant women, or humans with gout.

This dosage level is known to begin to dump excess accumulation of copper in the urine, which is not harmful in itself. However, if continued for long periods of time, it is believed that this level would eventually produce a conditioned copper deficiency (Deosthale & Gopalan, 1974). In Indian children, this level of dosage produces bone deformities after long-term intake. However, such a dosage might be a promising dietary approach to Wilson's Disease for adult men and women under the supervision of a knowledgeable orthomolecular physician, but as far as I know, has not been attempted. Much higher doses have been attempted with animal studies involving metal poisoning and cancer for short periods of time without adverse events. Applications to humans have not been attempted in the data I have reviewed. However, experimental molybdenum cancer drugs and chelation therapy drugs for Wilson's Disease are in the early stages of clinical trials with very promising results (Kopf-Maier, 1994).

Even though the IMO suggests no precautionary warnings are necessary for any molybdenum product, from industrial grade molybdenum compounds to dietary compounds, I do believe two cautions are in order.

1. Do not take molybdenum supplements above the Estimated Adequate and Safe levels during pregnancy since high doses have produced lower birth weight in all species studied, except under the direct supervision of a knowledgeable orthomolecular physician (See Molybdenum Tables: Appendix B. Schmitt (1991), a clinician who uses molybdenum supplements in his practice, will not use supplements of molybdenum at all during pregnancy because of this finding in animal studies. (See "Molybdenum Tables" for unanswered questions about rises in blood molybdenum levels during pregnancy and labor. Molybdenum blood levels double above normal levels, but the implications of this are still unclear to me.)

2. Taking molybdenum supplements may worsen symptoms for people diagnosed with gout since uric acid production is enhanced.

Speculations

I have searched for information, without success, on population studies for Climax, Colorado, the location of the largest molybdenum mine in the world. There I hoped to find evidence that would confirm data and add to information which is not complete about Armenian and Indian exposures to high levels. In Climax, Colorado, the tap water and ground water contain very high levels of molybdenum. I would expect to find the same incidence of gout-like symptoms, but lowered incidence of throat, forestomach, colon, and rectal cancers, lowered incidence of diabetes, no health problems associated with lead, cadmium, and mercury poisoning, the absence of Wilson's Disease among other things (see Molybdenum Tables: Appendix B). Cancer, diabetes, Wilson's Disease, heavy metal poisoning incidence were not among the issues studied in either Armenia or India.