Petition for Inclusion on the National List of Allowed Substances

-

Nickel

-

Micronutrient at

§205.601(j)(6)(ii)

SEP -4 2009

§205.601

Nickel Micronutrient

3 September 2009

wilson jones.

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Petition

Inclusion of Nickel
in §205.601(j)(6)(ii)

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Appendix A

University of Georgia
Cooperative Extension Service
Commercial Pecan Management

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Appendix B

Label of Conventional
Nickel Foliar Fertilizer

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MSDS's
Applicable Nickel Salts

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Petition

Inclusion of Nickel in §205.601(j)(6)(ii)

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September 3, 2009

Program Manager, USDA/AMS/TM/NOP
Room 4008-So., Ag Stop 0268
1400 Independence Ave., SW
Washington, DC 20250

Dear Sir or Madam,

Enclosed with this cover letter are two copies of a petition to add the micronutrient nickel to the National List at §205.601.

If you have any questions or require additional information or clarification, please let me know. My contact information is given above.

Sincerely,

Richard Theuer

Nickel Micronutrient Petition - §205.601

SEP -4 2009

The petitioner seeks inclusion of the micronutrient NICKEL, in the form of acceptable salts, on the National List as "Synthetic substances allowed for use in organic crop production, § 205.601." Specifically, the petitioner requests that paragraph 205.601(j)(6)(ii) be amended by insertion of the word "nickel" so that this paragraph will read as follows: "Sulfate, carbonate, oxides, or silicates of zinc, copper, iron, manganese molybdenum, ***nickel***, selenium, and cobalt."

Information on the substances being petitioned:

1. The substances' chemical or material common name: Nickel (salts).

Nickel is a metallic element found as such in meteorites and present in ores as sulfides, arsenides, antimonides and oxides or silicates. The specific synthetic salts of nickel consistent with the current salt restrictions of §205.601(j)(6)(ii) are nickel sulfate, nickel carbonate hydroxide, and nickel hydroxide. [Nickel silicate does not appear to exist.] Lignin sulfonate is allowed at §205.601(j)(4) as a "chelating agent" so that nickel in this form would become permissible if made with nickel sulfate. §205.601(j)(6) specifically prohibits use of the nitrates and chlorides of micronutrients in organic production. Nickel nitrate and nickel chloride are intentionally excluded from the scope of this petition.

ARS/USDA research found that the following nickel materials have equivalent potency, listed in decreasing order of desirability for use in organic production:

a) A non-synthetic "Organic* Nickel" aqueous leachate of specially grown *Alyssum* (a nickel hyperaccumulator) biomass (in the form of nickel malate and probably nickel citrate) [*fertilizer industry definition of "organic"]. The composition and method of production of this form of nickel are patented by ARS/USDA and a license has been granted to a potential producer. Unfortunately, this material is not available commercially. [The market is very small and production scale may not be commercially viable.]
b) Nickel sulfate hexahydrate – subject of this petition
c) Nickel lignin sulfonate – permissible via §205.601(j)(4) if this petition is allowed (nickel lignin sulfonate is likely to be more compatible in the context of worker safety)
d) Nickel glucoheptonate – not included in this petition
e) Nickel chloride hexahydrate - impermissible per §205.601(j)(6)(ii)
f) Nickel nitrate hexahydrate – impermissible §205.601(j)(6)(ii)
g) Nickel sulfamate tetrahydrate – impermissible since sulfamate is not an allowed synthetic substance at §205.601

Nickel deficiency in plants can be caused by nickel-deficient soil or by conditioned nickel deficiency caused by high soil levels of competitive metals (particularly zinc) that compete for and block nickel uptake by the plant. In the case of conditioned deficiency, foliar application of nickel is generally required until the soil micronutrient balance is corrected.

Given the competitive inhibition of soil nickel uptake by high levels of zinc, ARS/USDA recommends foliar application of nickel. ATTRA recommends that fertilizers for foliar application be totally soluble to avoid clogging the spray equipment. Consequently, the synthetic nickel source most practically useful as well as consistent with the regulation is the sulfate salt. Nickel carbonate and nickel hydroxide (which are soluble in dilute acid) may be able to be processed to form soluble forms by reaction with permitted non-synthetic substances (e.g., acetic acid/vinegar, citric acid).

The petitioned nickel salts may be incorporated into fertilizers applied to soil.

2. Manufacturers

The petitioned nickel salts are simple inorganic chemicals available from reagent grade chemical distributors (Fisher Scientific, Mallinckrodt, et al.) and other sources.

Science Stuff, Inc.
1104 Newport Ave
Austin, TX 78753
(512) 837-6020

Sciencelab.com, Inc.
14025 Smith Rd.
Houston, Texas 77396
800-901-7247

Acros Organics N.V.
One Reagent Lane
Fair Lawn, NJ 07410
800-227-6701

Mallinckrodt Baker, Inc.
222 Red School Lane
Phillipsburg, NJ 08865
908-859-2151

Fisher Scientific
One Reagent Lane
Fair Lawn, NJ 07410
201-796-7100

3. The intended use.

Use of the petitioned nickel compounds as micronutrients in organic crop production and the current use of nickel compounds in conventional crop production are restricted to situations of nickel deficiency documented by testing.

4. Crops for which the substance will be used.

Nickel was established as an essential micronutrient for plants over a quarter century ago. In the last ten years, the Southeastern Fruit and Tree Nut Research Laboratory of the Agricultural Research Service of USDA established that nickel deficiency is economically important. The major agricultural crop adversely affected by

nickel deficiency is pecans. Nickel deficiency in pecans has been most clearly documented in the Coastal Plain of the Southeastern United States.

> *"Nickel was discovered to be deficient in pecan orchards, causing replant problems and symptoms known as "mouse ear". It was discovered that timely foliar sprays of nickel corrected or prevented mouse-ear and replant problems. Subsequent observations of other crops indicate that nickel is a limiting factor in many crop situations. Examples include River Birch, plum, peach, walnut, almond, apple, citrus, grapes, containerized woody ornamentals, and in certain tissue culture associated situations. Observations also indicate that Ni is a limiting factor in disease resistance of a variety of crops. Findings indicate that lack of understanding in nickel nutrition could be a major factor limiting profitability in a host of horticultural crops. Circumstantial evidence indicates that there are many modalities of unknown cause (or causes previously attributed to other biotic or abiotic factors) that are in reality tied to insufficient nickel."* Source: Wood, B.W., Reilly, C.C., Nyczepir, A.P. 2004. Nickel deficiency is occurring in orchard trees [abstract]. Hortscience. 39(4):858.

The Southeastern Fruit and Tree Nut Research Laboratory published the following information on the rate and method of nickel application to correct deficiency.

> *Evaluation of several inorganic and organic forms of Ni have indicated that solutions from all sources function well to correct deficiencies when timely applied as a foliar spray to affected trees at Ni concentrations >10 mg.L-1. Addition of urea, ammonium nitrate, or nicotinic acid to Ni spray solutions increased apparent foliar uptake from Ni sprays. The lower critical level of Ni, based on foliar analysis, appears to be in the 3-5 mg.L-1 dw range, with the upper critical level appearing to be >50 mg.L-1 dw. The cause of Ni deficiency in soils possessing plenty of Ni is associated with excessive amounts of one or more metals (e.g., Ca, Mg, Fe, Mn, Cu, and Zn) which inhibit Ni uptake and/or utilization. Root damage by nematode feeding and cool/dry soils during early spring also contributes to Ni deficiency. Foliar application of Ni to foliage in the autumn and subsequent appearance of Ni in dormant season shoot tissues indicates that Ni can be mobilized from senescing foliage to dormant season shoots and is therefore available for early spring growth. Evidence indicates that pecan has a higher Ni requirement than most other crop species because it transports nitrogenous substances as ureides.* Source: Wood, B.W., Reilly, C.C., Nyczepir, A.P. 2005. Correcting Ni deficiency in pecan and other crops [abstract]. Hortscience. 40(4):1065.

More recent publications indicate that a single autumnal foliar application of a solution containing 100 mg/L of nickel prevented deficiency symptoms from occurring the following spring.

Appendix A is the document "Cultural Management of Commercial Pecan Orchards" produced by the University of Georgia Cooperative Extension Service. Note the discussion of nickel on page 6 and the picture illustrating "mouse ear" on page 7.

5. The source of the substance and a detailed description of its manufacturing or processing procedures from the basic component(s) to the final product.

The source of most commercial metallic nickel is the surface mineral ore "laterite." Laterite is a nickel oxide ore created when nickel sufide deposits are weathered by the environment and converted to the oxide. Laterite is generally by open pit mining. About 25% of world nickel production comes from recycling.

Nickel is isolated from laterite ore mainly by acid leaching. The pressure acid leach (PAL) process involves sulfuric acid in a high temperature and high pressure autoclave to dissolve the nickel and other metallic elements in the ore, such as cobalt. The autoclaves, as large as 30 meters in length and 4 meters in diameter, use a mixture of ore slurry and sulfuric acid at 280°C (535°F) under 5400 kilopascals of pressure. Heap leaching involves slow percolation of sulfuric acid through heaped ore for several months to dissolve the nickel and cobalt. Limestone is used to neutralize the acid and precipitate the mineral hydroxides.

Subsequent processing varies according to the nature of the ores and the quality and quantity of available process water. One method precipitates the metals as sulfides, re-dissolves them with oxygen under pressure followed by solvent extraction, and converts them to the metallic form by reduction with hydrogen. Another method precipitates the metals as hydroxides, re-dissolves them with ammonia, refines them with solvents, and reduces them to the metallic form by electrolysis.

Nickel sulfate of high purity (free of cadmium and other heavy metals) is produced by dissolving nickel metal or nickel oxides in sulfuric acid.

Reacting an aqueous solution of nickel sulfate with sodium carbonate precipitates nickel carbonate hydroxide.

Nickel hydroxide, also referred to as "green nickel oxide," is a precipitate formed when a solution of a soluble nickel salt such as the sulfate or nitrate is mixed with aqueous ammonia. True nickel oxide is made by pyrolysis of nickel hydroxide or carbonate and has the formula NiO.

6. A summary of any available previous reviews of the petitioned substance

Beginning about 2004, the American Association of Plant Food Control Officials (AAFPCO) evaluated the evidence for nickel deficiency in agricultural crops and the need to allow nickel in fertilizers. Based on this review, AAPFCO added nickel to its official list of permitted "micro plant nutrients" in 2007.

See No. 7 below.

7. Information regarding EPA, FDA, and State regulatory authority registrations.

In the United States and Canada, fertilizers are regulated by the individual States and Provinces. The authoritative body that establishes standards for fertilizers in North America is the American Association of Plant Food Control Officials (AAPFCO). In 2007, AAPFCO officially redefined its "Official Term T-9,"

> *"Secondary and Micro Plant Nutrients," to include nickel in the list of recognized micro plant nutrients. Official Term T-9 now reads as follows: "Secondary and Micro Plant Nutrients – Those other than the primary nutrients that are essential for the normal growth of plants and that may need to be added to the growth medium. Secondary plant nutrients shall include calcium, magnesium, and sulfur; micro plant nutrients shall include boron, chlorine, cobalt, copper, iron, manganese, molybdenum,* ***nickel (Official 2007)****, sodium and zinc. (Official 1965)"*

AAPFCO proposed the following standards for the risk-based acceptable concentration (RBC) of nickel in inorganic fertilizers: 250 ppm per 1% P2O5 in soil-applied NPK products and 1,900 ppm per 1% micronutrient in soil-applied micronutrient products. The RBC is the estimated maximum 'safe level' of that particular metal in that type of fertilizer product at one percent (1%) of the nutrient level (e.g., P2O5 or iron, manganese, zinc). These numerical values were recommended by AAPFCO's Board of Directors at its February 18, 2001, meeting to be incorporated into the fertilizer adulteration clause.

In 2004, AAFPCO established a minimum level of 0.0010% (10 ppm) for nickel in fertilizers claiming nickel.

8. Chemical Abstract Service (CAS) numbers of petitioned nickel salts; product labels.

Nickel compound	Chemical Abstracts Numbers (CAS No.)
Nickel carbonate	3333-67-3 carbonic acid nickel (2^+) salt
	16337-84-1 carbonic acid nickel salt
Nickel carbonate hydroxide	65405-96-1 [μ-[carbonato(2-)-O:O]] dihydroxytrinickel
	12607-70-4 [carbonato(2-)] tetrahydroxytrinickel
	39430-27-8 nickel carbonate hydroxide tetrahydrate
Nickel hydroxide	12054-48-7 nickel hydroxide (Ni(OH)2)
	11113-74-9 nickel hydroxide
Nickel sulfate	7786-81-4 nickel sulfate
	10101-98-1 nickel sulfate heptahydrate
	10101-97-0 nickel sulfate hexahydrate

See Appendix B for the label of a conventional foliar fertilizer containing nickel sulfate.

9. The substance's physical properties and chemical mode of action including

(a) Chemical interactions with other substances, especially substances used in organic production;

Nickel sulfate is a freely soluble and relatively non-reactive salt compatible with other fertilizer components.

(b) Toxicity and environmental persistence;

Like other metallic transition elements, including nutritionally essential ones like copper and zinc, nickel can be toxic to plants and animals, depending on the level. In 1999, the Environmental Protection Agency released a report[1] on the issue of heavy metals and other non-nutritive constituents (also referred to as contaminants) in fertilizers and liming materials (referred to as fertilizers hereafter). The report provided background information on fertilizer use, consumption patterns, composition and regulations. This information was gathered for fertilizers from natural mineral sources and those that are derived from industrial by-products. See No. 12 below.

(c) Environmental impacts from its use and/or manufacture;

As it is for other nutritionally essential metallic transition elements, mining and refining ore to convert to the metal and metal salts are environmentally intrusive. Waste water from certain nickel refining processes can be especially problematic.

(d) Effects on human health;

Nickel is an essential nutrient for plants and animals, serving as a cofactor or structural component of specific enzymes. Nickel deficiency has been demonstrated in higher animals but not in man. Foods naturally contain nickel, with the highest concentration in nuts and legumes, followed by chocolate milk powder and chocolate candy. There is no evidence in humans of adverse effects of nickel through consumption of a normal diet.

Accidental ingestion of high levels – 0.5 grams to 3.5 grams – of soluble nickel salts by adults caused illness but not death. Individuals with hypersensitivity to nickel may develop contact dermatitis following oral exposure to nickel. The 'No Adverse Effect Level' for soluble nickel salts in adult humans is 5 mg/kg/day.

Nickel alloys are sometimes used instead of the common mercury-silver dental amalgam for tooth restoration. Their high cost limits their use.

Air-borne nickel salts adversely affect the lungs. Cancer may result. Nickel is a known human carcinogen. See Item No. 10 below.

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[1] EPA document "Background Report on Fertilizer Use, Contaminants and Regulation." EPA 747-R-98-003. January, 1999.

(e) Effects on soil organisms, crops, or livestock.

Nickel deficiency has been observed in plants under laboratory conditions and in actual growing of both edible crop and ornamental plants. Nickel is an essential nutrient for higher animals but deficiency has not been observed under natural conditions.

Nickel is a heavy metal. High soil levels can be the result of using recycled industrial materials and sewage sludge.

Nickel is nutritionally essential, so animals are able to absorb much when the diet contains little and to absorb little when exposed to high environmental levels of soluble nickel. This auto-regulation of body nickel levels has been demonstrated most clearly in rainbow trout. See the annotated bibliography (Appendix C)

10. Safety information

Accidental acute ingestion of high levels – 0.5 grams to 3.5 grams – of soluble nickel salts caused illness but not death in adults. Individuals with hypersensitivity to nickel may develop contact dermatitis following oral exposure to nickel. The 'No Adverse Effect Level' for soluble nickel salts in adult humans is 5 mg/kg/day.

Material Safety Data Sheets (MSDS) for nickel sulfate, nickel carbonate and nickel hydroxide are attached as Appendix D.

NIEHS' 2002 Report on Carcinogens listed nickel compounds as "known" human carcinogens based on studies of workers showing excess deaths from lung and nasal cancers and on their mechanisms of action. Nickel is used in many industrial applications as catalysts and in batteries, pigments and ceramics. Inhalation of nickel salts in the form of an aerosol can cause lung damage and cancer, so respiratory protection should be a condition of foliar application (spraying) of nickel salts.

11. Research information

The attached annotated bibliography includes the abstracts of all research and research reviews that the author of this petition found and considered relevant to the petition.

12. Petition Justification Statement

The purpose of the petition process is to enable the National List to be changed in response to current advances in agricultural knowledge that is appropriate for organic production. The list of micronutrients currently on the National List was established in 1995 and finalized in 2000. At that time, it was known that nickel was essential for

plants but there was no evidence that nickel deficiency was an actual problem in the field. Now that we know that nickel deficiency is a problem, it is appropriate to update the micronutrient list in the regulation to include nickel. AAPFCO acknowledged this and updated its micronutrient list in 2007 to enable farmers to correct documented nickel deficiency.

Pecans, as well certain other crops, transport nitrogen from the roots to the leaves in the form of ureides – urea-containing molecules. The enzyme urease is required in the leaves to convert ureides to urea and ammonia that the plant can use for growth.

Urease is a metalloenzyme; the essential metal in urease is nickel. When insufficient nickel is available in the soil, urease levels fall and toxic levels of urea build up in the leaves, causing necrosis of the leaf edges. This condition is described as "mouse ear."

Soil can provide insufficient nickel if it is 'absolutely deficient' and also if the level of another metal (especially zinc) that competes with nickel for root transport is high enough to block nickel uptake. Soils used for pecans are frequently deficient in zinc. Long-term application of zinc to the soil to correct the zinc deficiency has made the soil zinc level high enough to create a conditioned nickel deficiency in the plants.

Currently, farmers attempting to establish organic pecan orchards or grow organic pecans in the Coastal Plain of the southeastern states are experiencing crop failures and tree mortality due to documented nickel deficiency. While nickel deficiency appears to dominate in acidic soils of the Atlantic and Gulf Coast Coastal Plain, it also occurs in alkaline soils of the central and southwestern U.S. and in northern Mexico. Thus, nickel deficiency in pecan is a problem from North Carolina to California.

Conventional pecan growers can treat nickel deficiency with nickel containing fertilizers because AAPFCO permitted nickel application in 2007. The National Organic regulation, however, prohibits nickel application to crops.

One of the purposes of the Organic Food Production Act was to facilitate interstate commerce in fresh and processed food that is organically produced. Unfortunately, in its present form, the regulations promulgated under the Act thwart production of organic pecans and hurt pecan growers attempting to grow organically.

As described above, an alternative to synthetic nickel salts is an extract of the biomass of a nickel-accumulator crop grown on nickel-rich soil that has been harvested and dried. Unfortunately, nickel-rich biomass is commercially unavailable at this time. Note that the accumulator crop must be grown where the soil is rich in nickel, currently southwestern Oregon, and made available where nickel deficiency is a problem, from North Carolina and Georgia to Texas, Oklahoma, and Missouri, and on to California.

Most fertilizers contain little nickel. The EPA[2] summarized the heavy metal content of fertilizers based on data from the California Department of Food and Agriculture (1997), the Washington State Department of Ecology (1997), and other sources. This report gives the following data.

Fertilizer description	Average Nickel, ppm (mg/kg)	Source in EPA 747-R-98-003
P_2O_5 fertilizers (average 44.3% P_2O_5)	27.5	Table G-1c
NPK fertilizers applied for P_2O_5 content (average 14.1%N; 42.9%P; 2.3%K)	28.36	Table G-2c
NPK fertilizers applied for N content (average 23.2%N; 2.4%P; 2.5%K)	0.51	Table G-3c
Potash (K_2O) fertilizers (average 55.6% K_2O)	1.4	Table G-4c
Sulfur nutrient fertilizers (average 67.1% S)	48.9	Table G-9c
Liming materials (average 81.3% $CaCO_3$)	11.6	Table G-11c

Non-synthetic mineral sources of nickel do not appear to be commercially available. Currently, no nickel ores are mined in the United States. A nickel sulfide ore was mined in Lancaster County, Pennsylvania, from 1853 until the mine closed in 1860. This nickel was the incentive to create the U.S. five-cent coin. Later, small deposits of Garnierite nickel silicate ore containing as much as 20% to 40% nickel were mined in North Carolina and Oregon. Today, Garnierite ores with high nickel amounts are depleted.

The Webster nickel mine in western North Carolina closed around 1920. The Riddle Mine in Douglas County, Oregon, the last nickel mine in the United States, closed in 1987. Attempts by Canadian mining interests to open new nickel mines in Josephine County, Oregon, and the Upper Peninsula of Michigan are being fought by environmentalists.

The important nickel ore Garnierite, a pale apple-green mineral, is a hydrous silicate of nickel and magnesium. Garnierite forms by lateritic weathering of ultramafic rocks and occurs in many nickel laterite deposits in the world. Garnierite is a semi-precious stone and is imported from New Caledonia as jewelry-grade material.

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[2] EPA document "Background Report on Fertilizer Use, Contaminants and Regulation." EPA 747-R-98-003. January, 1999.

Soil application of insoluble nickel sources may not reverse nickel deficiency in the short or intermediate term. Because the high soluble zinc levels in many of these soils inhibits root absorption of nickel, foliar application of nickel to nickel-deficient pecan trees may be required for successful cultivation of this crop until soil mineral balance is achieved.

An alternative cultural method is conventional cultivation, which permits nickel fertilization when it is needed to treat deficiency.

13. Confidential Business Information

This petition contains no Confidential Business Information (CBI) and no confidential commercial information. All of the information has been drawn from published sources. Copyrights of copyrighted documents have been respected. Consequently, journal articles are presented as abstracts only in the appended annotated bibliography, Appendix C.

14. Petitioner

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Raleigh, NC 27615
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Tel. 919-844-5843

Category 1. Adverse impacts on humans or the environment? Substance **Nickel Micronutrient**

Question	Yes	No	N/A[1]	Documentation (TAP; petition; regulatory agency; other)
1. Are there adverse effects on environment from manufacture, use, or disposal? [§205.600 b.2]	X			Most commercial metallic nickel is made from surface deposits of "laterite," a nickel oxide ore, mined in open pits. The nickel is dissolved with sulfuric acid. The sulfuric acid is ultimately neutralized with limestone.
2. Is there environmental contamination during manufacture, use, misuse, or disposal? [§6518 m.3]	X			Nickel is used in many industrial applications as catalysts and in batteries, pigments and ceramics. Industrial wastes (and sewage sludge) can contain sufficient nickel (a heavy metal) to exceed soil heavy metal maximum.
3. Is the substance harmful to the environment? [§6517c(1)(A)(i);6517(c)(2)(A)i]		X		Nickel is not harmful to the environment at the levels used to correct documented nickel deficiency in crops.
4. Does the substance contain List 1, 2, or 3 inerts? [§6517 c (1)(B)(ii); 205.601(m)2]		X		No.
5. Is there potential for detrimental chemical interaction with other materials used? [§6518 m.1]		X		Not at the levels used to correct documented nickel deficiency in crops.
6. Are there adverse biological and chemical interactions in agro-ecosystem? [§6518 m.5]		X		Not at the levels used to correct documented nickel deficiency in crops.
7. Are there detrimental physiological effects on soil organisms, crops, or livestock? [§6518 m.5]	X	X		Nickel is an essential micronutrient for crops and livestock. Animals and humans exposed to high levels of airborne nickel may develop cancer. Nickel and nickel salts are known carcinogens based on tests with laboratory animals.
8. Is there a toxic or other adverse action of the material or its breakdown products? [§6518 m.2]		X		Nickel is essential for plants and animals but it also is a carcinogen, especially with airborne exposure. Lung protection is required during spraying and other application of nickel-containing fertilizer.
9. Is there undesirable persistence or concentration of the material or breakdown products in environment?[§6518 m.2]	X	X		Nickel is a heavy metal so soil application of industrial waste (including sewage sludge) can lead to buildup in the soil. Application of nickel to nickel-deficient plants and soil should result in normal and desirable levels of soil nickel.
10. Is there any harmful effect on human health? [§6517 c (1)(A)(i) ; 6517 c(2)(A)i; §6518 m.4]	X			NIEHS lists nickel compounds as "known" human carcinogens based on studies of workers showing excess deaths from lung and nasal cancers. Nickel is used in many industrial applications as catalysts and in batteries, pigments and ceramics.
11. Is there an adverse effect on human health as defined by applicable Federal regulations? [205.600 b.3]			X	N/A - Nickel salts are substances under review for crops production.
12. Is the substance GRAS when used according to FDA's good manufacturing practices? [§205.600 b.5]			X	N/A - Nickel salts are substances under review for crops production.
13. Does the substance contain residues of heavy metals or other contaminants in excess of FDA tolerances? [§205.600 b.5]			X	N/A - Nickel salts are substances under review for crops production.

[1]If the substance under review is for crops or livestock production, all of the questions from 205.600 (b) are N/A—not applicable.

Category 2. Is the Substance Essential for Organic Production? Substance **Nickel Micronutrient**

Question	Yes	No	N/A[1]	Documentation (TAP; petition; regulatory agency; other)
1. Is the substance formulated or manufactured by a chemical process? [6502 (21)]	X			Petition. Nickel in ore is dissolved in sulfuric acid (to form nickel sulfate) and then converted to metallic nickel. Pure metallic nickel is dissolved in sulfuric acid to form nickel sulfate. Reacting nickel sulfate with sodium carbonate yields nickel carbonate hydroxide. Reacting nickel sulfate with aqueous ammonia yields nickel hydroxide.
2. Is the substance formulated or manufactured by a process that chemically changes a substance extracted from naturally occurring plant, animal, or mineral, sources? [6502 (21)]	X			Petition. Nickel in ore is dissolved in sulfuric acid (to form nickel sulfate) and then converted to metallic nickel. Pure metallic nickel is dissolved in sulfuric acid to form nickel sulfate. Reacting nickel sulfate with sodium carbonate yields nickel carbonate hydroxide. Reacting nickel sulfate with aqueous ammonia yields nickel hydroxide.
3. Is the substance created by naturally occurring biological processes? [6502 (21)]		X		Petition.
4. Is there a natural source of the substance? [§205.600 b.1]			X	N/A - Nickel salts are substances under review for crops production.
5. Is there an organic substitute? [§205.600 b.1]			X	N/A - Nickel salts are substances under review for crops production.
6. Is the substance essential for handling of organically produced agricultural products? [§205.600 b.6]			X	N/A - Nickel salts are substances under review for crops production.
7. Is there a wholly natural substitute product? [§6517 c (1)(A)(ii)]	X			A nickel-accumulator crop (such as Alyssum) can be grown on nickel-rich soil and the biomass can be harvested and dried. An aqueous extract of the biomass provides sufficient nickel to overcome nickel deficiency. Nickel-rich biomass is commercially unavailable at this time.
8. Is the substance used in handling, not synthetic, but not organically produced? [§6517 c (1)(B)(iii)]			X	N/A - Nickel salts are substances under review for crops production.
9. Is there any alternative substances? [§6518 m.6]				A nickel-accumulator crop (such as Alyssum) can be grown on nickel-rich soil and the biomass can be harvested and dried. An aqueous extract of the biomass provides sufficient nickel to overcome nickel deficiency. Nickel-rich biomass is commercially unavailable at this time.
10. Is there another practice that would make the substance unnecessary? [§6518 m.6]				A nickel-accumulator crop (such as Alyssum) can be grown on nickel-rich soil and the biomass can be harvested and dried. An aqueous extract of the biomass provides sufficient nickel to overcome nickel deficiency. Nickel-rich biomass is commercially unavailable at this time.

[1]If the substance under review is for crops or livestock production, all of the questions from 205.600 (b) are N/A—not applicable.

Category 3. Is the substance compatible with organic production practices?

Substance **Nickel Micronutrient**

Question	Yes	No	N/A[1]	Documentation (TAP; petition; regulatory agency; other)
1. Is the substance compatible with organic handling? [§205.600 b.2]			X	Petition is for nickel as a micronutrient in organic crop production.
2. Is the substance consistent with organic farming and handling? [§6517 c (1)(A)(iii); 6517 c (2)(A)(ii)]	X			Petition. Organic pecan growers are harmed by crop failures and tree mortality due to documented nickel deficiency. Conventional pecan growers can treat nickel deficiency with nickel containing fertilizers because AAPFCO permitted nickel application in 2007.
3. Is the substance compatible with a system of sustainable agriculture? [§6518 m.7]	X			Petition. Organic pecan growers are harmed by crop failures and tree mortality due to documented nickel deficiency. Organic pecan growing is not sustainable without nickel.
4. Is the nutritional quality of the food maintained with the substance? [§205.600 b.3]	X			Petition: Foods naturally contain nickel, with the highest concentration in nuts and legumes.
5. Is the primary use as a preservative? [§205.600 b.4]		X		
6. Is the primary use to recreate or improve flavors, colors, textures, or nutritive values lost in processing (except when required by law, e.g., vitamin D in milk)? [205.600 b.4]		X		
7. Is the substance used in production, and does it contain an active synthetic ingredient in the following categories: a. copper and sulfur compounds;		X		
b. toxins derived from bacteria;		X		
c. pheromones, soaps, horticultural oils, fish emulsions, treated seed, vitamins and minerals?		X		
d. livestock parasiticides and medicines?		X		
e. production aids including netting, tree wraps and seals, insect traps, sticky barriers, row covers, and equipment cleaners?		X		

[1]If the substance under review is for crops or livestock production, all of the questions from 205.600 (b) are N/A—not applicable.

NOSB RECOMMENDED DECISION

Form NOPLIST2. Full Board Transmittal to NOP

For NOSB Meeting: _____ | Substance: _____

A. Evaluation Criteria (Documentation attached; committee recommendation attached)

	Criteria Satisfied?
1. Impact on humans and environment	Yes ☐ No ☐ (see B below)
2. Availability criteria	Yes ☐ No ☐ (see B below)
3. Compatibility & consistency	Yes ☐ No ☐ (see B below)

B. Substance fails criteria?

Criteria category: _____

Comments:

C. Proposed Annotation: _____

_____

Basis for annotation:

To meet criteria above: ____ Criteria: _____

Other regulatory criteria: ____ Citation: _____

D. Final Board Action & Vote: Motion by: _____ Second: _____

Vote:

Yes: _____

No: _____

Abstain: _____

Agricultural		Nonagricultural		Crops	
Synthetic		Not synthetic		Livestock	
Allowed[1]		Prohibited[2]		Handling	
No restriction		Deferred4		Rejected[3]	

1—substance voted to be added as "allowed" on National List

Annotation: _____

_____

2—substance to be added to "prohibited" paragraph of National List

Describe why a prohibited substance: _____

_____

3—substance was rejected by vote for amending National List

Describe why material was rejected: _____

_____

4-substance was recommended to be deferred

Describe why deferred; if any follow-up is needed. If follow-up needed, who conducts follow-up. _____

_____

E. Approved by NOSB Chair to transmit to NOP:

_____ _____

Dave Carter, NOSB Chair Date

F. NOP Action: Include in FR to amend National List: ☐

Return to NOSB ☐ Reason: _____

_____ _____

Program Manager Date

NOSB COMMITTEE RECOMMENDATION

Form NOPLIST1. Committee Transmittal to NOSB

For NOSB Meeting: _____ | Substance: _____

Committee: Crops ☐ Livestock ☐ Handling ☐

A. Evaluation Criteria (Documentation attached; committee recommendation attached)

	Criteria Satisfied?
4. Impact on humans and environment	Yes ☐ No ☐ (see B below)
5. Availability criteria	Yes ☐ No ☐ (see B below)
6. Compatibility & consistency	Yes ☐ No ☐ (see B below)

B. Substance fails criteria?

Criteria category: _____

Comments:

C. Proposed Annotation: _____

_____

Basis for annotation:

To meet criteria above: ____ Criteria: _____

Other regulatory criteria: ____ Citation: _____

D. Recommended Committee Action & Vote: Motion by: _____

Seconded: _____

Vote:

Yes: _____

No: _____

Abstain: _____

Agricultural		Nonagricultural		Crops	
Synthetic		Not synthetic		Livestock	
Allowed[1]		Prohibited[2]		Handling	
No restriction		Deferred4		Rejected[3]	

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_____

4-substance was recommended to be deferred

Describe why deferred; if follow-up is needed. If follow-up needed, who will follow up _____

_____

E. Approved by Committee Chair to transmit to NOSB:

_____ _____

Committee Chair Date

THE UNIVERSITY OF GEORGIA
COOPERATIVE EXTENSION
Colleges of Agricultural and Environmental Sciences & Family and Consumer Sciences

Cultural Management of Commercial Pecan Orchards

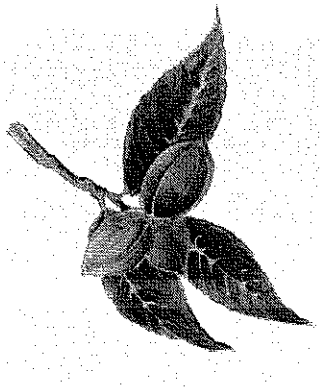
2

Appendix A

University of Georgia
Cooperative Extension Service
Commercial Pecan Management

Contents

Cultural Management of Commercial Pecan Orchards



Lenny Wells, Extension Horticulturist
Kerry A. Harrison, Extension Engineer

Commercial pecan production is a complex practice, requiring considerable knowledge of the pecan tree and of the limiting factors involved in the production of the pecan crop. One of the major constraints involved in the production of pecans is the alternate or irregular bearing tendency, which is an innate survival characteristic of the tree. Years of heavy crop load are termed "on" years, while those with poor crop loads are termed "off" years. In order for a commercial pecan operation to be consistently successful, the goal of the operation should be annual production of a moderate crop of high quality nuts, rather than the production of a high yield in a single given year. Culturally, there are several basic factors that will help to promote optimum profitability with a commercial pecan orchard.

Fertilization

Proper nutrition is a key requirement for the reliable production of quality pecans. Like other plants, pecans require nutrients for optimal growth and fruit production. Fertilizers supply nutrients to soils and help correct nutrient deficiencies. Three of the six macronutrients needed by pecans are commonly deficient in Georgia soils. These include nitrogen, potassium and magnesium. Three of the seven micronutrients required by pecans are commonly deficient as well: zinc, boron and iron. Recent attention has also been given to deficiencies of nickel in pecan orchards located on Coastal Plain soils.

The most reliable indicator of the nutritional needs of the pecan tree is the foliar or leaf analysis. Soil samples are less efficient for determining nutritional deficiencies in mature orchards but are quite useful for monitoring soil pH and lime requirements. Leaf analysis is more sensitive to changes in nutrient supply because it measures amounts of specific nutrients in the leaves. Sample leaves for analysis between July 7 and August 7, as this is the period in which nutrient concentrations in the leaves are most stable. Nutrient sufficiency levels are shown in Table 1.

Table 1. Leaf Sufficiency levels for essential elements of pecans

Element	Sufficiency Ranges
N (%)	2.5-3.3
P (%)	0.12-0.3
K (%)	1.25-2.5
Mg (%)	0.35-0.6
Ca (%)	1.3-1.5
S (%)	0.25-0.5
Zn (ppm)	50-100
Fe (ppm)	50-300
Mn (ppm)	100-800
B (ppm)	50-100
Cu (ppm)	6-30

Nitrogen

Nitrogen is the element that most commonly limits pecan growth and ultimately orchard profitability. It provides better tree growth, a higher percent kernel, and a healthier tree. When properly maintained, nitrogen helps to ensure optimal year to year production. Nitrogen deficiencies cause poor growth and poor tree health. Excessive nitrogen stimulates excess foliage, shading and, in some instances, reduced yield. The key to nitrogen management is to balance timely availability with tree demand.

The management of nitrogen fertilization in the pecan orchard will vary for irrigated versus non-irrigated orchards and from year to year within a given orchard, depending upon crop load.

Nitrogen uptake in the pecan tree is driven by demand. The two critical periods of nitrogen demand during the season are early foliage growth and kernel filling. The early spring foliage flush is nourished primarily from reserves held within the tree, while the nitrogen demand during the kernel fill stage is usually satisfied from soil uptake. If nitrogen is limited at kernel filling, then the tree will mobilize nitrogen from the foliage to the kernels.

Nitrogen recommendations have evolved greatly over the years. Historically, recommendations have been to apply nitrogen in March or as a split application in March and May or June. A general "rule of thumb" for mature, well managed, irrigated orchards is to apply 10 lbs. of nitrogen /acre for every 100 lbs. of expected crop.

In the absence of leaf sample recommendations, fertilize dryland orchards with 100-150 lbs. of nitrogen made in split applications. Apply the first in late February or early March so winter rains will help improve soil moisture and, ultimately, nitrogen uptake. The second application can be made in May or June. Soil moisture and nitrogen uptake can be more easily controlled by the grower in irrigated orchards, where trees are more efficient in using nitrogen.

An alternative to the March/May or June split is to split the application between mid-spring and late summer. This timing works best for orchards bearing heavy crops and where other limiting factors are controlled. Well-managed trees coming off an "off" year begin spring foliage growth with a full supply of nutrients stored in the stems, trunk and roots. As a result, they demand little nitrogen, and healthy trees will not remove much nitrogen from the soil at this time. A more efficient use of nitrogen may be to apply ⅓ of the nitrogen required when the shoots are 75 percent expanded, which generally occurs in mid to late April in Georgia.

During "off" years, the April application alone is sufficient. During July, trees may be assessed with regard to their crop load. If the crop load is heavy, apply another ⅓ of the full rate in early to mid August. Apply the final ⅓ in early September to help maintain healthy foliage for optimum kernel filling, leaf retention and adequate nutrient storage pools. For example, with an expected crop of 1500 lbs./acre, the nitrogen would be applied as 50 lbs/acre in mid-late April, 50 lbs. in early August, and 50 lbs in early September. If only two applications are made per year, apply half the recommended nitrogen in April and again in late August.

The kernel filling process pulls nitrogen from the leaves. During an "on" year with heavy crop loads, these leaves must have soil nitrogen to remain healthy and fully functional. Once the kernel-filling process is complete, healthy leaves will maintain sufficient nitrogen to produce energy stores. Trees in the "off" cycle of production will have sufficient nitrogen and will not need late-summer applications.

Though more complex than the March or March/May applications, spring and late summer splits make for more efficient use of nitrogen by the tree, better meet the needs of the tree, and can help reduce the severity of alternate bearing if the orchard is otherwise well managed.

Phosphorous

Phosphorous is important for energy storage as well as the production of wood and nuts. Despite its value, phosphorous levels in Georgia soils are often adequate, and additional phosphorous should not be applied unless called for by leaf analysis. The predominant symptom of phosphorous deficiency is dull green foliage with no intervienal chlorosis. Such deficiencies are often overlooked and are easily mistaken for mild nitrogen deficiency. In heavy bearing varieties, phosphorous deficiency can be expressed as a marginal leaf scorch, which may begin 7-10 days before shuck split, and premature defoliation. High concentrations of phosphorous can inhibit the uptake of nitrogen as well as iron, zinc and copper by the pecan tree.

Phosphorous is relatively immobile in the soil, so a single application of 60-100 lbs. of P_2O_5 incorporated at planting can last for several years. Subsequent applications, as needed, should also be incorporated because surface applications require several years to be of benefit to the orchard. Make applications any time leaf levels fall below 0.12 percent.

Potassium

Potassium is essential for the movement of carbohydrates, regulation of osmosis, and the activation of enzymes within the pecan tree. The resistance of pecan trees to winter injury is also heavily influenced by potassium levels.

Maintaining an appropriate balance of nitrogen and potassium within the tree is vital. If leaf potassium content is near minimum levels, heavy nitrogen applications will induce a visible potassium shortage termed "nitrogen scorch." Nitrogen scorch can lead to serious defoliation (Figure 1), which appears first on the basal shoots and leaves, progressing upward. Scorched areas occur along the leaf margins, and are circular or oblong and about the size of a dime (Figure 2). Desirable and Schley trees are often especially sensitive to the nitrogen-potassium imbalance.

Less severe symptoms of potassium deficiency begin as an irregular interveinal chlorosis. As potassium concentrations decrease through the season, the chlorosis may spread up the shoot and leaf. Necrotic spots may develop on the surface of the leaf.

Transport of potassium from the leaves to the fruit often accelerates potassium deficiencies, particularly in heavy crop years. Such deficiencies may induce premature defoliation, shoot die back, and small, poorly filled nuts.

The most common method of supplying potassium is by soil application of muriate of potash. Rates should be based on leaf potassium and the amount of nitrogen applied. The nitrogen/potassium ratio should be maintained at 2:1 for most varieties in order to prevent leaf scorch. Apply during February before the onset of winter rains. Where late summer applications of nitrogen are used, additional applications of potassium should be made at that time if potassium levels in the leaf are marginal.

Application of foliar sprays using 2 to 4 lbs. of potassium nitrate per 100 gallons of water with the second and third cover sprays may be beneficial in reducing potassium deficiency the first and second years following diagnosis.

Figure 1. Pecan tree suffering from serious defoliation in July due to nitrogen:potassium imbalance.

Magnesium

Magnesium deficiency is relatively rare in pecan trees; however, it can occur in trees growing on dry, acid, or sandy soils, especially in orchards with high soil potassium levels. Magnesium deficiencies are characterized by an intervienal chlorosis , which forms a "Christmas tree" pattern on the leaf. In very severe cases, a marginal leaf scorch may follow chlorosis.

Magnesium deficiency is best prevented by maintaining soil pH at 6.0-6.3 and by the use of dolomitic limestone as a liming material. Dolomitic limestone contains both calcium and magnesium, and generally provides an adequate amount of magnesium for most orchards. Growers will usually be able to observe increases in their leaf magnesium levels the second growing season following application. Where trees are identified as magnesium deficient, magnesium sulfate is more effective at raising leaf magnesium levels because it is more water soluble. Deficient trees will respond more quickly to foliar sprays of magnesium sulfate (5 lbs/100 gal) applied from the first leaf stage through July, but soil application of magnesium will still be necessary to maintain adequate levels in orchard soils.

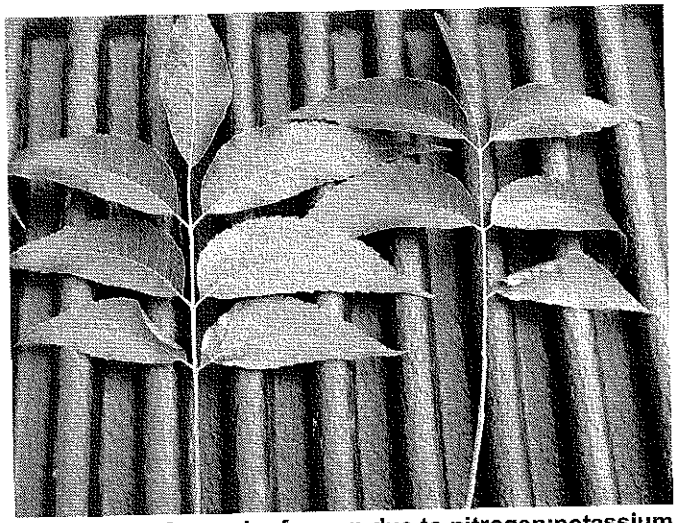


Figure 2. Leaf scorch of pecan due to nitrogen:potassium imbalance.

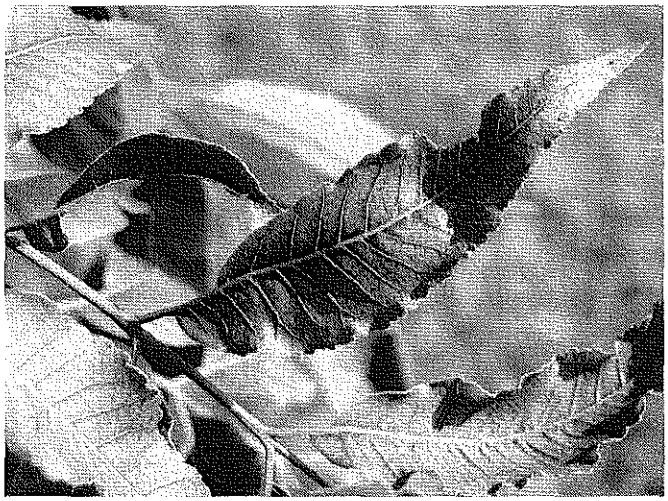


Figure 3. Symptoms of zinc deficiency in pecan.

Zinc

Zinc has a major influence on the economic return of a pecan operation due to its effect on flowering, fruit size, leaf efficiency and nut yield. It is particularly important to leaf expansion and shoot elongation. Zinc must be available to the tree at these specific times during the growing season. The most familiar characteristic of zinc deficiency is a curling of young leaves, causing a wavy leaf margin (Figure 3). Additional symptoms may be a rosette pattern, narrow leaves and terminal die-back.

Even with adequate soil levels, the availability of zinc in the soil depends upon soil pH, nitrogen, and phosphorous application. Liming soils with marginal zinc levels can reduce zinc uptake, particularly when nitrogen and phosphorous are applied in combination with lime. Zinc can usually be maintained at adequate levels under a liming program on acid soils if zinc is also applied. One advantage of soil applied zinc is that one application should provide an adequate supply of available zinc for many years to come.

Zinc moves slowly in the soil, requiring two or more years for a surface application to become effective. Therefore, foliar zinc applications are the most effective means of correcting the problem when deficiencies occur. Three to six applications per season are normally recommended, depending on the severity of the deficiency, with the first spray being applied about two weeks after bud break. Apply sprays at 2 week intervals over the period of shoot elongation. Apply foliar zinc any time leaf concentrations fall below 50 ppm or when visible symptoms of zinc deficiency are present.

Iron

As a component of chlorophyll, iron is essential to the process of photosynthesis. Iron deficiency rarely occurs from lack of iron in the soil but is induced by over-liming; cold, wet spring weather; or high soil concentrations of zinc, phosphorous or manganese. The deficiency generally occurs early in the growing season and clears up as the season progresses.

Symptoms of iron deficiency look similar to nitrogen deficiency, exhibiting chlorosis of the leaf. The inter-veinal chlorosis in iron deficiency sometimes retains very pronounced green veins. Also, with iron deficiency, young leaves are the first to be affected.

Depending upon the cause of iron deficiency, correction may take varying routes, including foliar applications of iron, changing the amount of lime applied to the orchard, or foregoing phosphorous applications. In most cases, the problem will clear up as the season progresses, especially when due to cool, wet spring conditions.

Iron deficiency is normally associated with older trees and may only occur in certain trees within the orchard. This deficiency may be genetically controlled, in which case it would be affected by rootstock.

Nickel

A deficiency of nickel is responsible for the condition known as mouse ear in pecan. Nickel is required by the urease enzyme in plants for the efficient conversion of urea to ammonia. When nickel is at an insufficient level in the plant, urea is not converted as efficiently and toxicity may develop.

Nickel has long been considered a nanonutrient, meaning that most plants require only very low levels of nickel to fulfill their requirements. The nickel content of most orchard soils in Georgia is minimal. In addition, nickel competes with zinc and other micronutrients for uptake by pecan roots. Due to the heavy applications of zinc applied to most managed orchards over many years, nickel is often not taken into the tree at a sufficient level, causing "mouse ear" symptoms.

Mouse ear first appears on the spring flush of growth. The most common symptom of mouse ear is a rounded or blunt leaflet tip. Affected leaves and leaflets are often smaller in size than healthy foliage. The rounded leaflet tips result from the buildup of urea to the point of toxicity in the leaf tissue (Figure 4). Additional information regarding mouse ear and nickel is available from the University of Georgia College of Agricultural and Environmental Sciences Web site:

http://pubs.caes.uga.edu/caespubs/pubcd/c893.htm

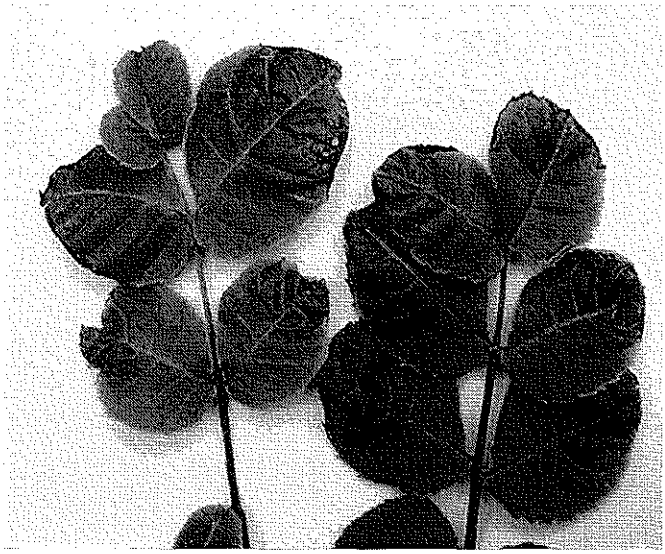


Figure 4. "Mouse Ear," a symptom of nickel deficiency in pecan.

Boron

Boron plays an important role in the movement of carbohydrates across cell walls and in the stability of the pollen germ tube. Inadequate boron can cause the germ tube to rupture, leading to reduced fertilization. Although leaf symptoms of boron deficiency in pecan are very rare, symptoms of deficiency and growth suppression can be expected to occur when leaf boron is less than 15 ppm. Normal growth is observed at 15-50 ppm, but preliminary results indicate that when leaf boron is between 50 and 100 ppm, pecan production is often enhanced through improved kernel percentage and better nut retention.

A major concern with regard to boron nutrition is the potential for toxicity. The acid soils of the southeastern United States are normally lower in boron than are alkaline soils. Soil-applied boron normally last for only one year, since boron is rapidly leached by rainfall and irrigation. Due to its affinity for leaching, soil boron levels may be affected by soil texture with a higher potential for leaching from sandy soils. Calcium, potassium and zinc compete with boron for uptake, so monitor leaf levels of all four nutrients. No studies have shown a yield response to soil-applied boron, but foliar applications have shown positive results.

Water Requirements

Water, particularly where nut quality is concerned, has more of an effect on pecan production than any other environmental factor. Drought stress affects nut size and filling as well as leaf and shoot growth. Adequate soil moisture is important at bud break in order to stimulate strong, vigorous growth from bloom through shell hardening for nut size, and during the nut filling stage for optimizing kernel percentage. If trees do not receive adequate soil moisture levels late in the season, shuck split and energy reserves are affected.

Pecan trees extract most of their water from the upper 32 inches of the soil profile. Though they are deep-rooted, most of the deep water available to the tree is considered survival water and is not useful for fruit production. The deeper the available water, the less water the tree will usually be able to absorb. Excessive water stress will cause the tree to shed leaves, drop nuts or only moderately fill the pecans.

The nut sizing period normally occurs from May 1 through August 15. Although not a critical water use stage for pecan, serious drought conditions during this period can affect yield. The most common visible effects of an extended drought during this period are excessive nut drop and "shell hardening" on small nuts. Lack of sufficient water during the nut sizing period causes small nuts and may lead to water stage fruit split, resulting from a sudden influx of water during the nut filling stage in some varieties.

The nut filling stage occurs from about August 15 to the first week of October, depending on variety. The most critical period for water use is during the first two weeks of September. Lack of sufficient water during the nut filling stage will lead to poorly filled nuts, which will result in poor nut quality.

Reports from other areas of the country indicate that as much as 350 gallons of water per day can be required by each tree during the nut filling stage. Based on this recommendation, if a mature orchard has a plant density of 12 trees per acre (60' x 60' spacing), then 4,200 gallons per acre per day may be needed. For a density of 20 trees per acre (46.5' x 46.5'), 7,000 gallons per acre per day may be needed.

Pecans can have high water requirements, using as much as 60 inches of total water (including rainfall) during the growing season. Drip and micro-irrigation system design capacity for a mature pecan orchard should be 3,600-6,000 gallons of water per acre per day. Additional information regarding drip irrigation for pecans can be obtained at the following Web site:

http://pubs.caes.uga.edu/caespubs/pubcd/B936.htm

Because of evaporation losses, solid-set sprinkler irrigation can require as much as three times more supplemental water as drip or micro-irrigation. Solid set irrigation systems should have a design capacity of 1.5–2.0 inches per week.

There is obviously a tradeoff between how much water the irrigation system can apply and the economics (initial cost) of the system. Growers should make certain they know the limitations of their irrigation

system and how the system should operate in order to minimize the system's weak points. Solid set irrigation systems obviously have different limitations than drip irrigation systems.

Whether an orchard is irrigated with solid-set sprinkler, drip or micro irrigation, an irrigation schedule that meets the needs of the pecan orchard will be required. There are several ways to schedule irrigation. Tensiometers, which are based on available soil moisture, and evaporation pans, which are based on evaporation rates, are the two most common scheduling methods. For solid-set systems, either method can be used but pan evaporation is the most common. Where drip or micro irrigation is used, tensiometers are the most common method. Solid-set sprinkler irrigation will allow moisture levels to fluctuate between 50 and 100 percent, where drip and micro irrigation maintain relatively constant soil moisture levels near emitters.

Operate systems for 12 hours or less per day to prevent water logging and oxygen depletion in the root zone. Roots thrive when water is present in the soil as a liquid film covering each soil particle while leaving soil interspaces filled with air. Growers should be aware of their system's delivery rate and adjust their operation times accordingly. At the maximum rate, the system should have the capability to deliver the required gallons per acre per day or inches per week.

Proper scheduling of the irrigation system during the year could save money in operating costs. If a system is designed to operate 12 hours per zone during peak water usage, then it could be used at only a fraction of that time during the early part of the season due to the tree's requirements at that stage. Depending on the irrigation system design, as few as 4 hours per zone may be sufficient during the first part of the growing season. Increase the operating time monthly until reaching the maximum 10-12 hours during peak water demand in late summer. A general operating schedule is provided in Table 2 for both solid set systems and drip or micro irrigation systems.

Table 2. General operating schedule for pecan irrigation.

Month	Drip/Micro % cycle	Sprinkler Inches/Acre
April	60	0.5"
May	70	0.75"
June	80	1.00"
July	90	1.25"
August	100	1.50"
September	100	1.50"
October	90	1.00"
November	60	0.5"

Crop Load Management

Excessive fruiting in pecan crops can cause economic losses due to reduced quality in the "on" year and, subsequently, a light return crop. Another effect may be tree limb breakage from excess weight. Several cultivars suffer from this type of over production including Cape Fear, Oconee, Schley, Stuart and Sumner.

Problems with over-production are related to a decrease in the number of leaves per fruit, especially in heavy crop years. On average, it takes 8-10 functional leaves to adequately fill one nut. Large fruited cultivars may require even more leaves per nut. Much of the stress on the leaves to fill nuts can be relieved by fruit thinning of prolific cultivars.

Fruit thinning may decrease total yield per tree for the current year, but this can be offset by an increase in marketable yield. The benefits of fruit thinning include increased nut quality in terms of higher kernel percentage and kernel grade as well as more stable yield and cash flow for the grower from year to year.

The process of kernel deposition requires a large amount of available carbohydrates. Each nut may compete with other nuts for carbohydrates produced by surrounding leaves or held in supporting branches. Fruit thinning prior to this competition provides each remaining nut with a greater supply of carbohydrates. The tree should still have enough carbohydrate reserves to support a flower crop the following season.

After fertilization, the ovule (the tissue that becomes the kernel) begins to expand and lengthen from the tip of the nut toward the stem. As the ovule expands, the space is filled with fluid until the ovule extends to the stem end of the nut. The shell begins to harden from the tip shortly after the nut reaches full size. After expansion is complete, kernel deposition begins.

In order to maximize return bloom, fruit thinning should be completed within two weeks from the time the ovule is one half expanded. If thinned too early, nuts will be difficult to shake from the tree. A straight, lengthwise cut through the center of the nut will expose the expanding ovule (a brown area) in order to help determine when it has reached 50 percent expansion (Figure 5). For much of the Georgia pecan belt, this will be in mid July. Fruit thinning after this time and throughout the water stage may improve nut quality for the current year, but will not substantially improve the return crop.

Fruit thinning can be accomplished with a conventional tree shaker equipped with donut or cylinder pads (Figure 6). These pads, filled with walnut hulls or glass beads, are not necessary but can help prevent trunk injury. A coating of silicon gel or grease between the

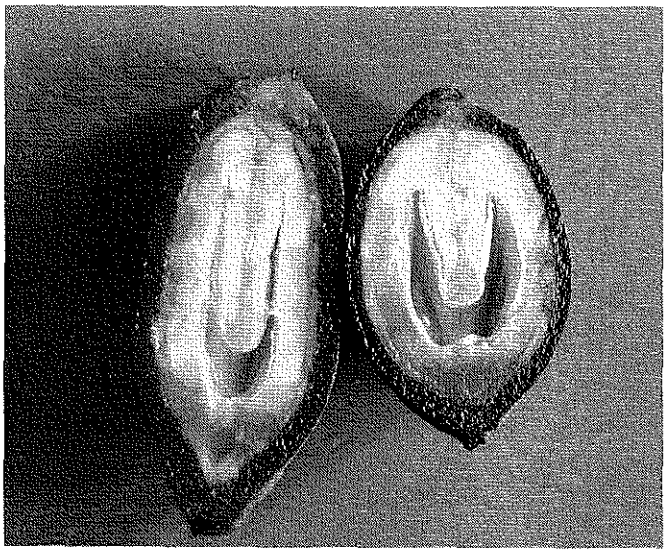


Figure 5. Development of the ovule in the pecan nut. The nut on the left is not ready to be fruit thinned. The nut on the right has reached 50 percent ovule expansion (brown area) and is ready to be thinned.

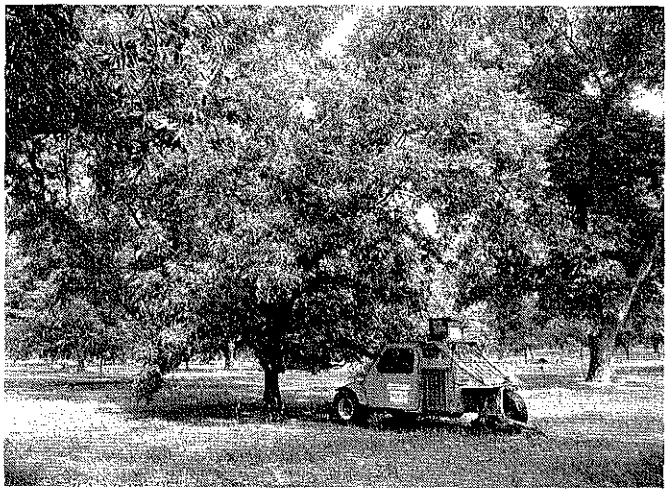


Figure 6. Fruit thinning of pecan.

Figure 7. Application of silicon gel between the flap and pad of a pecan shaker to prevent bark damage to pecan trees.

rubber flap and the pad serves as an additional precaution (Figure 7). This allows movement between the flap and pad, preventing movement of the bark during shaking. Position the shaker as level as possible on the ground and hold the tree in the center of the shaker head for shaking. Under conditions of recent heavy rainfall, the cambium layer under the bark may slip more easily, resulting in a greater risk of tree injury. In order to minimize trunk damage, delay nut thinning for a few days following rain. If rainfall has been plentiful and trees are flushing new growth, only a small number of trees should be shaken and observed for 36 hours for signs of bark splitting.

The quantity of nuts to remove varies depending upon nut size, cultivar, nut set, insect damage and disease incidence. Judging crop load is instrumental in determining if a crop needs thinning. This is a practice that takes experience. Trees with almost 100 percent of the shoots fruiting and a cluster size greater than three are overloaded and will benefit from thinning. Optimum crop load varies with cultivar and may range from 50-70 percent fruiting shoots. Many growers make the mistake of not removing enough nuts during thinning.

When shaking begins, do not be alarmed by the number of nuts that fall. Shake trees 2 to 3 seconds at a time, evaluated them, and shake them again if needed. Repeat this process two or three times when necessary until thinning is completed. The distribution of nuts removed throughout the tree may be enhanced by shaking from both north/south and east/west orientations. It is advisable to have one person on the ground watching what is happening as the trees are shaken. This person can communicate to the shaker operator when to stop and when to shake harder.

Proper management of crop load requires the grower to be familiar with the orchard. Not all cultivars or trees within a cultivar will require nut thinning in a given year. Thinning the pecan nut load in heavy crop years can be beneficial to nut quality and, for some varieties, to improving return bloom. Nut thinning is a sound management tool when used correctly. Growers should test nut thinning in a portion of their orchard and personally evaluate the results before using nut thinning as a regular practice.

Sunlight

Inadequate sunlight is highly limiting to pecan production. This situation may result from cloudy conditions, shading within the tree, or from shading among trees. Overcrowded trees become stressed as they receive insufficient sunlight, water and nutrients to fill the nuts and store carbohydrate reserves for the

following season. In the southeastern United States, a properly spaced orchard should allow approximately 50-60 percent sunlight to reach the ground at noon.

Cloudy conditions are common in the southeastern United States during the pecan growing season. Years in which cloud cover is heavy throughout the season, specifically during kernel development, can have a limiting effect on nut quality in the current season as well as on the following season's crop.

Shading within the tree is caused by high foliage density and can vary among cultivars. Certain cultivars bear on the periphery of the tree canopy, while others have open canopies and bear throughout the canopy. Such shading can be minimized but not eliminated for certain cultivars through training or pruning.

Overcrowding of trees within the orchard is the most common way in which shading limits pecan yield. In overcrowded situations, yield is higher on the borders, where shading is minimal and is reduced toward the center of the orchard. Dense orchard canopies lead to poor nut development and to death of the lower scaffold limbs, reducing the fruiting surface of the grove.

Although it is a common problem in many commercial orchards, overcrowding can be easily corrected by thinning the orchard. A variety of thinning techniques have been used to open up more light to the pecan orchard.

Historically, the most common method of thinning has been to remove every other row on the diagonal. Removing every tree in alternating rows takes out half of the trees, while leaving them in a square, although the orientation is rotated 45 degrees. This type of thinning normally results in a loss of a yield per acre for the first few years after thinning, but the loss will normally be made up in following years. Alternate-row thinning is the most aggressive manner of opening up light to the orchard. It does not take into account the yield potential of each tree and will undoubtedly remove some high yielding trees. In order to prevent a yield reduction immediately following thinning by this method, the procedure must be conducted on time, before the trees become heavily overcrowded.

More recently a highly effective method of selective thinning has been developed. Trees are individually rated by making a visual assessment of a tree's profit potential. This is determined by yield, pest pressure, nut quality, and value.

Cultivar, yield, tree condition, foliage, rots, lightning strikes, crown gall and limb-breakage are also involved in the rating. Ratings are subjective but still reflect profit potential. For example, a good canopy with a good off-season yielding Stuart tree may rate a 10, whereas a poor variety like Frotscher or Moore may rate low because of low nut quality. A Stuart with no crop in an "off " year would still rate low even though it is a good cultivar. Making an orchard map will help in keeping up with what trees you select. By this method, poor yielding trees, which provide no profit potential, can be eliminated, thereby opening up more sunlight for more productive trees. This will allow you to get a better idea of when you have reached the 40/60 canopy/open floor objective.

The selective thinning method requires a knowledge of the orchard and of individual trees in order to be effective. A minimum of 2 years of tree rating should be done before employing the selective thinning practice in order to evaluate an "on" and an "off" year.

When thinning an orchard, research indicates that it is best to do so prior to an "on" year. Trees can also be moved with tree spades to other locations where land is available, rather than simply cutting the tree down.

Leaf Retention

Healthy pecan foliage from the time of foliage development in the spring through November is critical to maintaining adequate carbohydrate storage pools through the dormant season.

Trees which ripen fruit late in the season normally retain their foliage later into the fall than those which ripen early. However, early ripening cultivars generally bring a better price for the grower, and are the preferred cultivars for most commercial growers. Care of the pecan foliage is important for these early ripening cultivars. A variety of biotic and abiotic factors may influence foliage and canopy health including disease, insects, nutritional status and adequate soil moisture.

Control of foliar diseases, including pecan scab, zonate leaf spot and powdery mildew, as well as insect pests such as a black pecan aphid (Figure 8), yellow pecan aphid and pecan leaf scorch mite, even in an "off" crop year, is imperative for leaf retention into the fall. Protection of the leaves during both "on" and "off" crop years will help ensure adequate storage pools for even crop production over time.

Maintaining fertility within the tree and soil, along with adequate soil moisture levels throughout the season, will also help to alleviate tree stress, thereby increasing the potential for leaf retention.

Pollination

Adequate pollination in the pecan orchard is vital but often overlooked. Research indicates that as much as a 30 percent fruit loss may occur when trees are

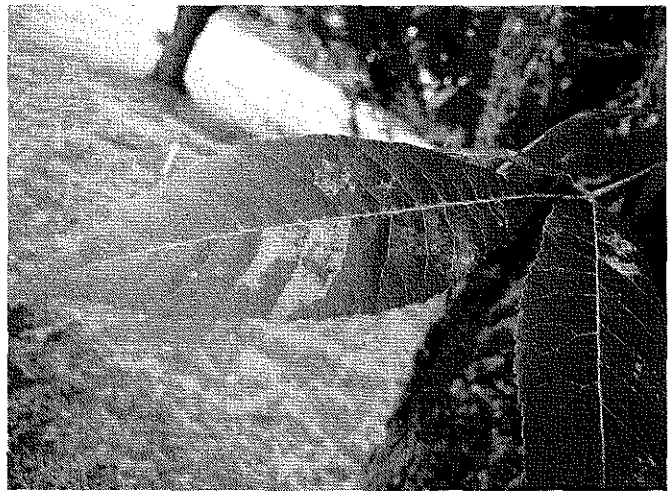


Figure 8. Damage to pecan foliage from black pecan aphid.

more than 150 feet, or about 3 rows, from a pollinator. Pollinators can be blocked in rows, or pollinator varieties with similar nut and kernel characteristics to main varieties can be positioned within main variety rows. Blocks of a particular variety should be no more than two to three rows in order to prevent substantial pollination problems. An effective alternative to blocking varieties is to place a pollinator at every fifth tree within every fifth row. This will provide adequate pollination and will not cause the crop to bring lower "blended" prices.

Orchards should contain at least two pollinator varieties to ensure that the entire pollination window for the main variety is covered. Pecans generally fall into two pollination categories. In type I varieties, the pollen matures before the pistil is receptive and are termed *protandrous*. Type II varieties, in which the pistil becomes receptive before the pollen matures, are termed *protogynous*.

Historically, it was believed that a combination of most Type I trees with most Type II trees would achieve reasonably good pollination for both types. Pecan pollination is much more complex than this two-class system, however. Shifts in flower maturity windows occur within a single variety as trees age. Flower maturity tends to occur earlier in older trees. The duration of these maturity windows shortens as the trees age.

A variety of factors may affect pollination. Uneven bud break due to abnormally warm or cool springs will lead to uneven pollen shed and disruption of flower maturity. Moist, cool conditions can delay pollen dispersal and extend pistil receptivity. Warm springs accelerate catkin development relative to that of female flowers, and reduce the duration of pollen dispersal and flower receptivity.

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For further information, contact your local county extension agent.

Learning for Life

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J. Scott Angle, Dean and Director

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Appendix B

Label of Conventional Nickel Foliar Fertilizer

NIPAN LLC

Nickel CBM™

General Information

Nickel CBM is designed for use as a foliar spray on pecans and other crops where the addition of nickel, copper, boron and molybdenum would be beneficial.

Nickel CBM can be tanked mixed with other nutrient and pest control products. Nickel CBM is not recommended for soil application.

Nickel CBM will disperse in water with little agitation. Many pesticides can be added and applied while spraying Nickel CBM. Follow this mixing sequence:

1. Water 2. Nickel CBM 3. Pesticide

Storage Instructions

Store in cool, dry place. May be stored in unheated facility. Undiluted Nickel CBM can be corrosive to tanks, pumps, meters and fittings. Absorbing on clay or other suitable absorbent can clean up spills from broken containers.

Warranty Statement: NIPAN LLC warrants that this product conforms to the chemical description on the label thereof and is reasonably fit for the purpose stated on the label only when used in accordance with directions under normal use conditions. It is impossible to eliminate all risks inherently associated with use of this product. Neither this warranty nor any other warranty of merchantability of fitness of a particular product expressed or implied, extends to the use of this product contrary to label instructions or under abnormal conditions not reasonably foreseeable to the seller, and the buyer assumes the risk of any such use.

Information about the components of this product may be obtained by writing to the manufacturer.

Guaranteed Analysis

Sulfur (S)	1.7%
Boron (B)	1.0%
Copper (Cu)	0.80%
Molybdenum (Mo)	0.08%
Nickel (Ni)	2.5%

Derived from copper sulfate, nickel sulfate, boric acid, sodium molybdate and citric acid.

KEEP OUT OF REACH OF CHILDREN

CAUTION

Harmful if swallowed. Avoid contact with skin, eyes, or clothing. Avoid breathing spray mist. May cause irritation of nose, throat and/or skin. Wear goggles and gloves when handling. In case of contact, immediately flush skin or eyes with water for several minutes.

Caution: Application of Molybdenum may result in forage crops containing levels of molybdenum, which are toxic to ruminant animals.

Caution: Over application or excessive use of boron can result in boron toxicity and crop injury.

Manufactured in the USA by:
NIPAN L.L.C.
P.O. Box 5611, 2867 Carroll Ulmer Rd.
Valdosta, GA 31603-5611.
™ Nickel CBM is trademark of NIPAN LLC.

Net Contents: 2.5 Gallons / 13.2 Liters

Directions For Use

Pecans:
For the correction and prevention of **mouse ear** on pecans and to reduce nut drop in June and improve disease resistance to scab. Three applications per season are recommended. The first spray for the correction and prevention of mouse ear and reduced nut drop; the second and third application improves disease resistance to scab and nut fill.

First application: Apply 2 pints/acre in the first spray or at the parachute stage.
Second application: Apply 2 pints/acre 14-21 days later

Third application: Apply 2 pints/acre, 14-21 days later

For severe **mouse ear** and newly transplanted pecan trees, apply 2 pints per acre in early October before leaf fall and the following spring.

Other crops:
Nickel CBM can be used in a balanced fertility program where supplemental micronutrient nutrition may improve growth, quality, disease resistance and yield. Improved nickel nutrition has been shown to increase disease resistance to rust.

Soybeans:
To increase root mass, nodulation and disease resistance to rust. Improved nickel nutrition of soybeans grown for seed can improve seed germination of saved seed.

Rate and timing: Ground application: Apply 12-16 oz in 15-20 gallons water per acre when soybeans are in R1 stage and again 14 to 21 days later.

Peanuts:
To increase root mass, nodulation and disease resistance to rust. Improved nickel nutrition of peanuts grown for seed can improve seed germination of saved seed.

Rate and timing: Ground application: Apply 8 oz per acre 50 days after planting. Repeat 14 to 21 days later for remainder of the season.

The addition of a non-ionic surfactant at the labeled rate is recommended to improve spray coverage.

Weight: 9.6 pounds/gallon

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Appendix C

Annotated Bibliography

Nickel Bibliography

ESSENTIALITY OF NICKEL FOR PLANTS OTHER THAN PECAN (1-18)

ESSENTIALITY OF NICKEL FOR PECAN (19-40)

EFFECTS OF NICKEL ON AQUATIC ANIMALS (41-51)

1. Roach, W. A. & Barclay, C. (1946) Nickel and multiple trace-element deficiencies in agricultural crops. Nature (Lond) 157: 696.

Wheat, potatoes, and broad beans were sprayed with solutions containing compounds of manganese, iron, boron, copper, zinc, and nickel in a factorial design. Each of the six elements gave statistically significant and economically important increases in yield. The authors believe that this is the first indication that nickel is of importance in increasing crop yield and also the first record of zinc deficiency in the British Isles that has been proved by the increase in yield as a result of treatment with zinc.

2. Dixon, N. E., Gazzola, C. et al. (1975) Jack Bean Urease (EC 3.5.1.5). A Metalloenzyme. A Simple Biological Role for Nickel? J Amer Chem Soc 97: 4131-4133.

Urease appears to be the first example of a nickel metalloenzyme, and nickel may well be an essential trace element in jack beans. Heretofore, nickel was the only element between vanadium and zinc in the fourth row of the periodic table which had not been recognized as essential, or as having some biological function.

3. Shimada, N., Ando, T. et al. (1980) Role of nickel in plant nutrition. Effects of nickel on the growth of tomato and soybean. Japanese J Soil Sci Plant Nutr 51: 493-496. 51: 493-496.

Urease leaf activity in tomato and soybean plants was found to increase with nickel additions. Low-nickel plants, grown with urea as the sole source of nitrogen, developed leaf tip necrosis from toxic accumulations of urea.

4. Eskew, D. L., Welch, R. M. & Cary, E. E. (1983) Nickel: An essential micronutrient for legumes and possibly all higher plants. Science 222: 621-623.

Soybean plants deprived of nickel accumulated toxic concentrations of urea (2.5 percent) in necrotic lesions on their leaflet tips. This occurred regardless of whether the plants were supplied with inorganic nitrogen or were dependent on nitrogen fixation. Nickel deprivation resulted in delayed nodulation and in a reduction of early growth. Addition of nickel (1 microgram per liter) to the nutrient media prevented urea accumulation, necrosis, and growth reductions. This evidence suggests that nickel is essential for soybeans and possible for higher plants in general.

5. Eskew, D. L., Welch, R. M. & Norvell, W. A. (1984) Nickel in higher plants: further evidence for an essential role. Plant Physiol 76: 691–693. Plant Physiol 76: 691-693.

Soybeans (Glycine max [L.] Meff.) grown in Ni-deficent nutrient solutions accumulated toxic orea concentrations which resulted in necrosis of their leaflet tips, a characteristic of Ni deficiency. Estimates of the Ni requirement of a plant were made by using seeds produced with different initial Ni contents. When compared to soybeans grown from seeds containing 2.5 na ms Ni, plants grown from seeds containing 13 nanograms Ni had a signcantly reduced incidence of leaflet tip necrosis. Plants grown from seeds conuaining 160 nanogams Ni produced leaves with almost no leaflet tip necrosis symptoms. Neither Al, Cd, Sn, nor V were able to substitute for Ni. In other experiments, a small excess of EDTA was included in the nutrient solution in addition to that needed to chelate micronutrient metals. Under these conditions, nodulated nitrogen-fing soybeans had a high incidence of leaflet tip necrosis, even when 1 micromolar NiEDTA was supplied. However, in nutrient solutions containing inoranic sources of N, 1 micromolar NiEDTA almost completely prevented leaflet tip necrosis, although no significant irease in leaf urease activity was observed. Cowpeas (Vigna ngNicudata [L.] Walp) grown in Ni-deficient nutrient solutions containing NO3 and NH4 also developed leaflet tip necrosis, which was analogous to that produced in soybeans, and 1 micromolar NiEDTA additions prevented these symptoms.

These findings further support our contention that Ni is an essential element for higher plants.

6. Brown, P. H., Welch, R. M. & Cary, E. E. (1987) Nickel: A Micronutrient Essential for Higher Plants. Plant Physiol 85: 801-803.

Nickel was established as an essential micronutrient for the growth of temperate cereal crops. Grain from barley (Hordeum vulgare L. cv `Onda'; containing 40 to 80 nanograms of Ni per gram dry weight) grown in solution culture with negligible Ni concentrations (< 30 nanograms of Ni per liter) exhibited greatly reduced germination rates (i.e. 50% less than grain from Ni-adequate plants) and seedling vigor of the viable grain was greatly depressed. Grain containing less than 30 nanograms per gram dry weight was inviable. Under Ni-deficient conditions, barley plants fail to produce viable grain because of a disruption of the maternal plant's normal grain-filling and maturation processes that occur following formation of the grain embryo. The observations that (a) barley plants fail to complete their life cycle in the absence of Ni and (b) addition of Ni to the growth medium completely alleviates deficiency symptoms in the maternal plants satisfies the essentiality criteria; thus, Ni should be considered a micronutrient for cereals. Because Ni is required by legumes, and is now established as essential for cereals, we conclude that Ni should be added to the list of micronutrients essential for all higher plant growth.

7. Brown, P. H., Welch, R. M. et al. (1987) Beneficial effects of nickel on plant growth. J Plant Nutr 10: 2125.

Wheat, barley, and oat plants, grown in the absence of added Ni (-Ni), accumulated 15 to 20 fold higher levels of urea in their leaf tips than Ni supplied plants (+Ni). Oat plants (-Ni) matured 15 days earlier that +Ni oat plants. When grown from low Ni seed, Ni-deprived barley plants had significantly (30%) lower root and shoot weights that +Ni

plants and displayed characteristic symptoms that could be eliminated by Ni supplementation. Tissue Fe-levels were positively correlated with tissue Ni levels.

8. Thirkell, D., Myles, A. D. et al. (1989) The urease of Ureaplasma urealyticum. J Gen Microbiol 135: 315-323.

The urease from Ureaplasma urealyticum (serotype 8) has been purified by immuno-affinity column chromatography. Two active nickel-containing forms of the enzyme were demonstrated by non-denaturing electrophoretic analysis and a single active peak of apparent molecular mass 190 kDa was shown by FPLC. Total inactivation and denaturation of the enzyme to give three subunit polypeptides (one of 72 kDa containing nickel, one of 14 kDa and one of 11 kDa) was achieved by treatment with SDS and boiling. Densitometry suggested that the active enzyme contains equimolar ratios of the three subunits and hence is a hexamer. The enzyme displayed a pH optimum of 6.9 and pI values were determined. Storage of the purified enzyme at -70 degrees C followed by thawing to 20 degrees C caused a partial breakdown to inactive subunits. Anti-urease monoclonal antibodies bound both to the active enzyme and to the inactive 72 kDa subunit, and the antibodies cross-reacted with ureases from all of the other human serotypes. Competition assays with the antibodies revealed four distinct epitopes of the enzyme, all distinct from its active site.

9. Brown, P. H., Welch, R. M. & Madison, J. T. (1990) Effect of nickel deficiency on soluble anion, amino acid, and nitrogen levels in barley. Plant Soil 125: 19-27.

Barley (*Hordeum vulgare* L. cv. 'Onda') plants were grown in nutrient solutions supplied either 0 (no Ni added), 0.6, or 1.0 µM NiSO4. Plants supplied 0 µM Ni developed Ni deficiency symptoms: Ni deficiency resulted in the disruption of nitrogen metabolism, and affected the concentration of malate and various inorganic anions in roots, shoots, and grain of barley.

The concentration of 10 of the 11 soluble amino acids determined were 50-200% higher in the 30-day-old shoots of plants supplied inadequate Ni levels that in shoots of Ni-supplied plants. The total concentration of all amino acids determined was higher in root and grain of Ni-deficient plants. Concentrations of NO3- and Cl- were also higher in Ni-deficient barley shoots that in Ni-sufficient barley shoots. In contrast, the concentration of alanine in shoots on Hi-deficient barley was reduced to one-third of the concentration in Ni-sufficient plants. The shoot concentrations of maltase and SO42- were also depressed under Ni-deficient conditions. Total nitrogen concentration in grain, but not in shoots, of Ni-deficient plants was significantly increased over that found in Ni-adequate plants.

Nickel deficiency results in marked disruption of N metabolism, malate, and amino acid concentrations in barley. These results are discussed in view of the possible roles on Ni in plants.

10. Piccini, D. F. & Malavolta, E. (1992) Effect of nickel on two common bean cultivars. J Plant Nutr 15: 2343-2350.

Two common bean (*Phaseolus vulgaris* L.) cultivars, Carioca and IAPAR-14, were grown in a nutrient solution in the presence of 0, 1, 2, and 4 mg/L of nickel (Ni). Both cultivars developed symptoms of toxicity in the roots and tops, but especially in the leaves. Dry matter was reduced by the higher Ni levels. The IAPAR-12 cultivar, however, was more tolerant than the Carioca cultivar to Ni. Yield decreased as Ni levels in the solutions were increased. There was no seed production at the 4 mg/LK Ni concentration for either of the two cultivars. A 50% reduction in leaf chlorophyll was observed when the 4 mg/L Ni was supplied, Mineral analysis of sever plant parts showed a buildup of Ni concentration in the roots which as accompanied by higher iron (FE) concentrations. Increasing Ni supply resulted in no significant changes in the concentrations of calcium (Ca), magnesium (Mg), manganese (Mn), and zinc (ZN) in the tissue. The level of nitrogen (N), phosphorus (P), potassium (K), copper (Cu), and Ni, however, increased, as a consequence either of either the dilution effect, interaction in absorption, or both. No explanation can be given for the observed differential tolerance.

11. Nicoulaud, B. A. L. & Bloom, A. J. (1998) Nickel Supplements Improve Growth when Foliar Urea is the Sole Nitrogen Source for Tomato. J. Amer. Soc. Hort. Sci. 123: 556-559.

Concentrations of up to 1.0 µM NiCl2 in a nutrient solution improved growth of tomato (Lycopersicon esculentum Mill. `T-5') seedlings that received foliar urea as their sole nitrogen source. Nickel in the nutrient solution decreased the amount of urea present in the shoots and increased the amount in the roots, although it had no significant effect upon leaf urease activity. These results indicate that a) the presence of nickel in the nutrient solution improves growth of plants receiving foliar urea and b) the effect of nickel was related more to increased urea translocation from shoot to root than to enhanced leaf urease activity.

12. Broderick, C. E. (1999) Promotion of germination and seedling development of lima beans (Phaseolus spp.) by nickel chloride. HortScience

34: 513.

Delaware is among the largest producers of lima beans in the United States, having more than 10,000 acres in this crop. The plants are raised from seeds, but seeds are notably prone to injury during handling. The seed has little or no endosperm and relies on reserve food materials in the cotyledons. Lima beans are legumes, and the seeds store nitrogenous compounds. Nickel is implicated in nitrogen metabolism, and nickel is now implicated as an essential mineral nutrient element. With the variable rate of germination of lima bean seeds, our objective was set to determine the effect of nickel on seedling development of the lima bean plant. Lima bean seeds were soaked for various periods in 10 solutions from 0 to 500 ppm nickel chloride. Soaking for 1 h in 100 ppm nickel chloride solution was determined to be the best treatment. The 100 ppm treatment then became the standard treatment for lima bean seeds. Hence, seeds were

treated with distilled water of the 100 ppm nickel chloride. The results are that the nickel chloride significantly improved the rate of germination of lima bean seeds. Germination rates were improved from 60% ± 5% to 76% ± 3%. The total effect of treatments with nickel chloride varied from one batch of seeds to another; however, promotion of germiantion was significant. Osmoregulation and seed priming as well as stimulation of nitrogen metabolism by nickel are possible explanations of the positive effects of nickel chloride.

13. Gerendás, J., Polacco, J. et al. (1999) Significance of nickel for plant growth and metabolism. J. Plant Nutr. Soil Sci. 162: 241-256.

Ni is the most recent candidate to be added to the list of 13 essential mineral elements for higher plants although failure to complete the life cycle in the absence of Ni has only been demonstrated in a few plant species. Ni is considered an essential element primarily because of its function as an irreplaceable component of urease which is responsible for the hydrolysis of urea N, and which seems to be the only proven nutritional function of Ni in higher plants. For production of full urease activity and growth on urea N a critical deficiency level of around 100 g kg - 1 DW seems appropriate, while plants depending on mineral N may have a lower Ni requirement. Ni has also other effects on plant growth, of which the phytosanitary action is possibly most significant in the field. The incorporation of Ni into urease apoprotein requires the active participation of several accessory proteins, and mutations in genes coding the accessory proteins as well as the urease apoprotein have been exploited to characterise aspects of urease activation. The mobility of Ni within the plant, as compared to other heavy metals, is usually high, although little is known of the uptake mechanisms and the form of transported Ni under Ni-deprived conditions. This as well as other effects of Ni that cannot be related to its structural component of urease, remain to be elucidated.

14. Ruter, J. M. (2004) Mouse ear disorder on river birch caused by nickel deficiency. (Abstr.). Hort Science 39: 892.

Mouse ear (leaf curl, little leaf, squirrel ear) has been a problem for growers of container-grown river birch (Betula nigra L.) since the early 1990's. Mouse ear has been noticed in several southeastern States as well as Minnesota, Ohio, Oregon, and Wisconsin, making it a national problem. The disorder is easy to detect in nurseries as the plants appear stunted. The leaves are small, wrinkled, often darker green in color, commonly cupped, and have necrotic margins. New growth has shortened internodes which gives plants a witches-broom appearance. Plants growing in native soil rarely express the disorder. Several common micronutrients have been evaluated with no results. A trial was initiated in June, 2003 to determine if nickel deficiency was the cause of mouse-ear. Symptomatic river birch trees growing in a pine bark substrate in containers were treated with foliar applications of nickel sulfate and a substrate drench. Topdress applications of superphosphate (0-46-0) and Miloroganite, products known to contain nickel, were also applied. At 16 days after treatment (DAT), up to 5 cm of new growth occurred on plants sprayed with nickel sulfate and foliar concentrations of nickel

in the new growth increased five fold compared to control plants. At 30 DAT, shoot length increased 60%, leaf area increased 83%, and leaf dry mass increased 81% for trees receiving a foliar application compared to non-treated control plants. Treating trees with a substrate drench alleviated symptoms, whereas treatment with superphosphate and Milorganite did not. Trees receiving a foliar or drench application had normal growth for the remainder of the growing season. Additional studies are underway to refine methods of application, rates, and sources of nickel suitable for use.

15. Ruter, J. M. (2005) Response of Betula nigra `BNMTF' to foliar and drench applications of nickel. (Abstract). HortScience 40: 996.

Mouse ear disorder on container-grown river birch (Betula nigra L.) is a national problem caused by a deficiency of nickel. Symptomatic plants have leaves which are small, wrinkled, darker green, cupped, and have necrotic margins. Research showed that mouse ear could be cured by applications of nickel sulfate (Ruter, 2004). Further research was needed to determine optimal rates of application for sprays and drenches and to determine if phytotoxicity occurs at high rates. A study was initiated at a nursery in South Georgia on 25 June 2003, using river birch in their second growing season in #15 containers. Plants were selected for uniformity of mouse ear disorder. Treatments included a control, urea (0.24 g·L-1) + surfactant (1.0 mL·L-1), 250, 500, 750, and 1000 mg·L-1 nickel sulfate sprays, and substrate drenches applied at 150 and 300 mg of Ni/pot. After 30 days, all plants treated with nickel sulfate had 100% normal growth, except the 150 mg of Ni/pot drench, which had 79% of the canopy showing normal growth. No phytotoxicity was noted. Plants receiving foliar sprays had a 66% to 72% increase in leaf area, a 64% to 68% increase in leaf dry mass, a 31% to 44% increase in stem length, and a 9% to 17% increase in specific leaf area compared to nontreated plants. Drench treatments increased leaf area up to 62%, leaf dry mass to 55% and stem length up to 29% over control plants. Nickel in the foliage of nontreated plants was 2.3 mg·kg-1. For the spray treatments, foliar Ni ranged from 5.5 mg·kg-1 for the 250 mg·L-1 treatment to 9.3 mg·kg-1 for the 1000 mg·L-1 treatment. Though plants at the high rate of drench treatment resumed normal growth, foliar Ni levels were not different from control plants. In general, if plants were treated with Ni, then foliar B, Fe, and Zn decreased.

16. Ruter, J. M. (2005) Effect of Nickel Applications for the Control of Mouse Ear Disorder on River Birch. J Environ Hort 23: 17-20.

Mouse ear (leaf curl, little leaf, squirrel ear) disorder has been a problem in container-grown river birch (Betula nigra L.) for several decades. The disorder is easy to detect in nurseries as the plants appear stunted due to shortened internodes which give the appearance of a witches-broom. The leaves are small, wrinkled, are often darker green in color, are commonly cupped and have necrotic margins. Plants grown in soil rarely express the disorder. A trial was initiated in June 2003 to determine if a deficiency of nickel was the cause of mouse-ear on river birch. Symptomatic river birch trees (Betula nigra BNMTF Dura-Heat™) in their second growing season in #15 containers were selected for uniformity of size and mouse ear. Treatments included a 1) control, 2) 789

ppm Ni sprays, 3) 394 ppm Ni sprays, 4) 0.005 lbs Ni/yd3 as a drench, 5) 26 g/pot triple superphosphate (0–46–0), and 6) 130 g/pot Milorganite. Nickel was applied as nickel sulfate, whereas triple superphosphate and Milorganite contain trace amounts of nickel. At 16 days after treatment, up to 5 cm of new growth was evident on plants sprayed with nickel. Thirty days after treatment shoot length increased up to 60%, leaf area increased 80 to 83%, and leaf dry mass increased 76 to 81% for plants sprayed or drenched with nickel sulfate. Plants treated with triple superphosphate or Milorganite did not resume normal growth. All plants treated with nickel sulfate in 2003 did not show symptoms of mouse ear after initiation of growth in 2004. Based on this research mouse ear disorder of river birch is caused by a deficiency of nickel which can be corrected by foliar or drench applications of nickel sulfate.

17. Guengerich, F. P. (2009) Thematic minireview series: metals in biology. J Biol Chem 284: 18557.

Metals have important roles in biochemistry ranging from essential to toxic. This prologue introduces the second of the Thematic Minireview Series on Metals in Biology, which includes minireviews on five metals: iron, zinc, nickel, vanadium, and arsenic. Three of the minireviews are focused on the roles of the metals in enzymes (iron, nickel, and vanadium). Zinc deficiency is discussed in another, and the arsenic minireview deals with the toxic and some potentially useful applications of the biological effects.

18. Hansch, R. & Mendel, R. R. (2009) Physiological functions of mineral micronutrients (Cu, Zn, Mn, Fe, Ni, Mo, B, Cl). Curr Opin Plant Biol 12: 259-266.

Micronutrients are involved in all metabolic and cellular functions. Plants differ in their need for micronutrients, and we will focus here only on those elements that are generally accepted as essential for all higher plants: boron (B), chloride (Cl), copper (Cu), iron (Fe), manganese (Mn), molybdenum (Mo), nickel (Ni), and zinc (Zn). Several of these elements are redox-active that makes them essential as catalytically active cofactors in enzymes, others have enzyme-activating functions, and yet others fulfill a structural role in stabilizing proteins. In this review, we focus on the major functions of mineral micronutrients, mostly in cases where they were shown as constituents of proteins, making a selection and highlighting some functions in more detail.

19. Wood, B. W., Reilly, C. C. & Nyczepir, A. P. (2003) Nickel corrects mouse-ear. Pecan Grower 15: 3-8.

The mouse-ear disorder of pecan is a long standing problem that has resisted a solution for nearly a 100 years. Information is discussed relating to how mouse-ear is induced and what orchard managers can do to correct the disorder. Orchard management factors such as soil pH, soil moisture, amount of timing of N applications, zinc application, phosphorous fertilizers, all potentially influence mouse-ear severity. Symptoms implicate a trace micro nutrient deficiency as the primary causal factor. The prime candidates are Cu and Ni. Field and greenhouse results indicate that the problem is in fact due to nickel deficiency.

20. Wood, B. W. (2004) Nickel nutrition of pecan. Pecan Grower 16: 4-7.

Most U.S. pecan orchards would benefit from one or two annual applications of Ni as a foliar spray. Such sprays should be applied at Ni rates of 25 to 100 ppm, depending on degree of Ni deficiency. Sprays can be applied in early spring within about 10 days after bud break or, in the case of severe deficiency, in the autumn. Autumn treatment ensures Ni is present in buds at time of spring bud break. Most orchards are thought to experience chronic (hidden hunger) Ni deficiencies. Acute deficiencies (those showing visual symptoms) are relative few, but the number is increasing as soils accumulate metals that reduce Ni uptake by plants (i.e., Zn, Cu, Ca, and Mg).

21. Wood, B. W., Reilly, C. C. & Nyczepir, A. P. (2004) Mouse-ear of pecan: a nickel deficiency. Hort Science 39: 1238-1242.

Mouse-ear (ME) is a potentially severe anomalous growth disorder affecting pecan [Carya illinoinensis (Wangenh.) K. Koch] trees. It is especially severe in second generation sites throughout much of the Gulf Coast Coastal Plain of the southeastern U.S., but can also occur in potted nursery trees. Orchard and greenhouse studies on trees treated with either Cu or Ni indicated that foliar applied Ni corrects ME. ME symptoms were prevented, in both orchard and greenhouse trees, by a single mid-October foliar spray of Ni (nickel sulfate), whereas non-treated control trees exhibited severe ME. Similarly, post budbreak spring spray applications of Ni to foliage of shoots of orchard trees exhibiting severe ME prevented ME symptoms on subsequent growth, but did not correct morphological distortions of foliage developed prior to Ni treatment. Foliar application of Cu in mid-October to greenhouse seedling trees increased ME severity the following spring. Post budbreak application of Ni to these Cu treated MEed seedling trees prevented ME symptoms in post Ni application growth, but did not alter morphology of foliage exhibiting ME prior to Ni treatment. Thus, high leaf Cu concentrations appear to be capable of disrupting Ni dependent physiological processes. Foliar application of Ni to ME prone trees in mid-October or soon after budbreak, is an effective means of preventing or minimizing ME. These studies indicate that ME in pecan is due to a Ni deficiency at budbreak. It also supports the role of Ni as an essential plant nutrient element.

22. Wood, B. W., Reilly, C. C. & Nyczepir, A. P. (2005) Correcting Ni deficiency in pecan and other crops [abstract]. Hort Science 40: 1065.

The recent discovery of the existence of nickel (Ni) deficiency in field plantings of pecan [Carya illinoinensis (Wangenh.) K. Koch] (Wood et. al., 2004) has led to efforts to identify appropriate management approaches to correct tree deficiency and to identify the causes for Ni deficiency. Evaluation of several inorganic and organic forms of Ni have indicated that solutions from all sources function well to correct deficiencies when timely applied as a foliar spray to affected trees at Ni concentrations >10 mg.L-1. Addition of urea, ammonium nitrate, or nicotinic acid to Ni spray solutions increased apparent foliar uptake from Ni sprays. The lower critical level of Ni, based on foliar

analysis, appears to be in the 3-5 mg.L-1 dw range, with the upper critical level appearing to be >50 mg.L-1 dw. The cause of Ni deficiency in soils possessing plenty of Ni is associated with excessive amounts of one or more metals (e.g., Ca, Mg, Fr, Mn, Cu, and Zn) which inhibit Ni uptake and/or utilization. Root damage by nematode feeding and cool/dry soils during early spring also contributes to Ni deficiency. Foliar application of Ni to foliage in the autumn and subsequent appearance of Ni in dormant season shoot tissues indicates that Ni can be mobilized from senescing foliage to dormant season shoots and is therefore available for early spring growth. Evidence indicates that pecan has a higher Ni requirement than most other crop species because it transports nitrogenous substances as ureides. Thus, there is evidence that Ni-metalloenzymes are playing either a direct or indirect role in ureide and nitrogen metabolism. It is postulated that crop species that are most likely to exhibit field level Ni deficiencies are those which transport N as ureides. Candidate crops will be discussed.

23. Bai, C., Reilly, C. C. & Wood, B. W. (2006) Nickel deficiency disrupts metabolism of ureides, amino acids, and organic acids of young pecan foliage. Plant Physiol 140: 433-443.

The existence of nickel (Ni) deficiency is becoming increasingly apparent in crops, especially for ureide-transporting woody perennials, but its physiological role is poorly understood. We evaluated the concentrations of ureides, amino acids, and organic acids in photosynthetic foliar tissue from Ni-sufficient (Ni-S) versus Ni-deficient (Ni-D) pecan (Carya illinoinensis [Wangenh.] K. Koch). Foliage of Ni-D pecan seedlings exhibited metabolic disruption of nitrogen metabolism via ureide catabolism, amino acid metabolism, and ornithine cycle intermediates. Disruption of ureide catabolism in Ni-D foliage resulted in accumulation of xanthine, allantoic acid, ureidoglycolate, and citrulline, but total ureides, urea concentration, and urease activity were reduced. Disruption of amino acid metabolism in Ni-D foliage resulted in accumulation of glycine, valine, isoleucine, tyrosine, tryptophan, arginine, and total free amino acids, and lower concentrations of histidine and glutamic acid. Ni deficiency also disrupted the citric acid cycle, the second stage of respiration, where Ni-D foliage contained very low levels of citrate compared to Ni-S foliage. Disruption of carbon metabolism was also via accumulation of lactic and oxalic acids. The results indicate that mouse-ear, a key morphological symptom, is likely linked to the toxic accumulation of oxalic and lactic acids in the rapidly growing tips and margins of leaflets. Our results support the role of Ni as an essential plant nutrient element. The magnitude of metabolic disruption exhibited in Ni-D pecan is evidence of the existence of unidentified physiological roles for Ni in pecan.

24. Nyczepir, A. P., Wood, B. W. & Reilly, C. C. (2006) Impact of Meloidogyne partityla on mouse-ear and nickel deficiency of pecan in Georgia [abstract]. Phytopathology. 96: S86.

The Mouse-ear (ME) malady of pecan is a nickel (Ni) deficiency that has most recently become an orchard replant disorder. Mouse-ear has been associated with nematode parasitism in some commercial pecan orchards in Georgia. A field microplot study of

pecan seedlings treated with either Meloidogyne partityla or Criconemoides xenoplax or both found that parasitism by M. partityla can increase severity of ME symptoms. Furthermore, severity of ME symptoms was inversely proportional ($P < 0.05$) to Ni concentration in the pecan leaflets, thus supporting previous findings that ME severity is due to a physiological Ni deficiency. Results also indicate that parasitism by C. xenoplax appears to have little or no influence on ME severity or Ni nutrition of pecan. These results provide useful insights into the interrelationship among the pecan root-knot nematode, severity of ME symptoms, and Ni deficiency in pecan. The need to further investigate potential management strategies of M. partityla on pecan is warranted.

25. Nyczepir, A. P., Wood, B. W. & Reilly, C. C. (2006) Association of meloidogyne partityla with nickel deficiency and mouse-ear of pecan. HortScience 41: 402-404.

Pecan [Carya illinoinensis (Wangenh.) K. Koch] trees exhibit nickel (Ni) deficiency in certain orchard situations. The symptoms are manifest as either 'mouse-ear' or 'replant' disorder and in certain situations is associated with nematode parasitism. A field microplot study of pecan seedlings treated with Meloidogyne partityla and Criconemoides xenoplax or both found that parasitism by M. partityla can result in enhancement in the severity of mouse-ear symptoms and a reduction in foliar Ni concentration. The Ni threshold for triggering morphological symptoms in young developing foliage was between 0.265 and 0.862 ug.g-1 dw, while the threshold for rosetting was between 0.007 and 0.064 ug.g-1 dw. Results indicate that parasitism by M. partityla, but not C. xenoplax, is a contributing factor to the induction of Ni deficiency in pecan and raises the possibility of nematode parasitism and Ni nutrition being a contributing factor to many plant maladies.

26. Reilly, C. C., Wood, B. W. & Bai, C. (2006) Beneficial effects of nickel in pecan production. Pecan Grower 17: 26-27.

Nickel was combined with standard fungicide sprays and applied to foliage and fruit of Wichita, Apache, Desirable, Oconee, and Kiowa Pecan. Treatments reduced scab severity on all varieties, with those best most susceptible to scab being most influenced. The preliminary data indicates that nickel can be used as a management tool to be combined with other disease management tools to reduce tree susceptibility to scab disease.

27. Wood, B. W., Chaney, R. L. & Crawford, M. (2006) Correcting micronutrient deficiency using metal hyperaccumulators: alyssum biomass as a natural product for nickel deficiency correction. Hort Science 41: 1231-1234.

The existence of nickel (Ni) deficiency in certain horticultural crops merits development of fertilizer products suitable for specific niche uses and for correcting or preventing deficiency problems before marketability and yields are affected. The efficacy of satisfying plant nutritional needs for Ni using biomass of Ni-hyperaccumulator species was assessed. Aqueous extraction of Alyssum murale (Waldst. & Kit.) biomass yielded a Ni enriched extract, that upon spray application, corrects and prevents Ni deficiency in

pecan [Carya illinoinensis (Wangenh.) K. Koch]. The Ni-Alyssum biomass extract was as effective at correcting or preventing Ni deficiency as was a commercial Ni-sulfate salt. Foliar treatment of pecan with either source at >10 mg'L-1 Ni, regardless of source, prevented deficiency symptoms whereas treatment at <10 mg'L-1 Ni was only partially effective. Autumn application of Ni to foliage at 100 mg'L-1 Ni during leaf senescence resulted in enough remobilized Ni to prevent expression of morphologically based Ni deficiency symptoms the following spring. The study demonstrates that micronutrient deficiencies are potentially correctable using extracts of metal accumulating plants.

28. Wood, B. W., Reilly, C. C. & Nyczepir, A. P. (2006) Nickel deficiency in trees: symptoms and causes. Acta Horticulturae 721: 83-98.

This communication reports that the mouse-ear or little-leaf disorder and the associated replant disease of pecan [Carya illinoinensis (Wangenh.) K. Koch] is a Ni deficiency and is cured by timely foliar application of Ni (at 100mgL-1), thus documenting the first known example of Ni deficiency in orchard crops. Deficiency is inducible on soils with adequate Ni content by a) excessively high soil Zn, Cu, Mn, Fe, Ca, or Mg; b) root damage by root-knot nematodes; or c) dry and or cool soils at time of bud break. Symptoms associated with Ni deficiency, but varying with severity of deficiency, include early-season leaf chlorosis, dwarfing of foliage, blunting of leaf/leaflet tips, necrosis of leaf or leaflet tips, curled lead/leaflet margins, dwarfed internodes, distorted bud shape, brittle shoots, cold-injury-like death of over-wintering shoots, diminished root system with dead fibrous roots, failure of foliar lamina to develop, rosetting and loss of apical dominance, dwarfed trees, and tree death. In addition to pecan, Ni deficiency is exhibited by river birch; and based on symptoms and soil characteristics, it also appears to occur in certain other woody perennial crops (e.g., plum, peach and Pyracantha sp., and citrus). Its occurrence in two prominent ureide transporting hydrophiles raises the possibility that such species are most likely to experience Ni associated disorders than are other species. Observations implicate excessive soil accumulation of light metals from long-term fertilizer usage as a primary cause of Ni deficiency. These results raise the possibility that Ni is a contributing factor in certain recalcitrant maladies and replant disorders of certain woody perennial crops and support the role of Ni as an essential nutrient element for higher plants. Ni deficiency in field situations appears to be far more common than generally recognized; thus, meriting greater attention in horticultural production strategies and greater awareness by horticulturalists.

29. Bai, C., Reilly, C. C. & Wood, B. W. (2007) Identification and quantitation of asparagine and citrulline using high-performance liquid chromatography (HPLC). Anal Chem Insights 2: 31-36.

High-performance liquid chromatography (HPLC) analysis was used for identification of two problematic ureides, asparagine and citrulline. We report here a technique that takes advantage of the predictable delay in retention time of the co-asparagine/citrulline peak to enable both qualitative and quantitative analysis of asparagine and citrulline using the Platinum EPS reverse-phase C18 column (Alltech Associates). Asparagine alone is eluted earlier than citrulline alone, but when both of them are present in

biological samples they may co-elute. HPLC retention times for asparagine and citrulline were influenced by other ureides in the mixture. We found that at various asparagines and citrulline ratios [= 3:1, 1:1, and 1:3; corresponding to 75:25, 50:50, and 25:75 (muMol ml(-1)/muMol ml(-1))], the resulting peak exhibited different retention times. Adjustment of ureide ratios as internal standards enables peak identification and quantification. Both chemicals were quantified in xylem sap samples of pecan [Carya illinoinensis (Wangenh.) K. Koch] trees. Analysis revealed that tree nickel nutrition status affects relative concentrations of Urea Cycle intermediates, asparagine and citrulline, present in sap. Consequently, we concluded that the HPLC methods are presented to enable qualitative and quantitative analysis of these metabolically important ureides.

30. Bai, C., Reilly, C. C. & Wood, B. W. (2007) Nickel deficiency affects nitrogenous forms and urease activity in spring xylem sap of pecan. Journal of the American Society for Horticultural Science 132: 302-303.

While nickel (Ni) deficiency occurs in certain agricultural crops, little is known regarding the influence of deficiency on metabolic or physiological processes. We studied the influence of Ni deficiency on the reduced-nitrogen (N) composition of early spring xylem sap of pecan [Carya illinoinensis (Wangenh.) K. Koch]. HPLC analysis of sap composition found that the presence of ureido-, amide- and amino-N substances and that they are quantitatively influenced by tree Ni nutritional status. Ureido-N forms quantitatively dominated amide-N forms in regards to both molar concentration and the forms in which reduced N-atoms are present; thus pecan appears to be predominately a ureide transporting species. The primary ureido-N substances in sap of Ni-sufficient (Ni-S) trees are citrulline ' asparagine ' xanthine > ureidoglycolate > allantoic acid > allantoin ' uric acid ' urea. Asparagine is the primary amide-N form, while there are only traces of amino-N forms (e.g., tryptamine and '-phenylethylamine) in xylem sap. Nickel deficiency substantially increased of citrulline and allantoic acid in xylem sap while decreasing asparagine, xanthine, and '-phenylethylamine concentrations. These Ni linked quantitative shifts in reduced-N forms indicate that Ni nutrition potentially affects intermediates of both the ureide catabolic pathway and the Urea Cycle, and the N:C economy of the tree. Xylem sap associated urease specific activity was also reduced as a consequence of Ni deficiency. These results indicate that Ni deficiency potentially disrupts normal N-cycling via disruption of ureide metabolism.

31. Wood, B. W. & Reilly, C. C. (2007) Nickle: Usage guidelines. Pecan South 10: 20-26.

Knowledge by practitioners regarding how to use nickel fertilizers in pecan orchard enterprises is a limiting factor in optimization of physiological efficiency and profitability of orchard enterprises. Knowledge by farmers and extension specialists about tree nickel nutritional needs, and how these needs are best satisfied, is meager. This review summarizes guidelines relating to how orchard nutritional management for nickel is best accomplished. This information assists pecan growers in efforts to optimize orchard yields and profits by ensuring optimal nickel nutritional status.

32. Wood, B. W. & Reilly, C. C. (2007) Nickel: Impact on horticultural characteristics. Pecan South 10: 24-33.

Knowledge by practitioners regarding the potential impact of nickel nutritional physiology on pecan orchard profitability is a limiting factor in optimization of physiological efficiency of orchard enterprises. Knowledge by farmers and extension specialists about the role of nickel, a newly recognized micronutrient, is meager. This review summarizes situations where nickel influences key horticultural traits that influence tree and orchard yields and profitability. This information assists in recognition and correction of subtle forms of nickel deficiency in orchards, thus, helping to maximize profitability.

33. Wood, B. W. & Reilly, C. C. (2007) Nickel: Relevance to orchard profitability. Pecan South 10: 16-19.

The nutritional physiology of essential micronutrients in pecan, especially that of nickel, is a limiting factor in optimization of physiological efficiency of orchard enterprises. Knowledge by farmers and extension specialists about the role of nickel, a newly recognized micronutrient, is meager. This review summarizes situations where nickel influences key metabolic and physiological characteristics that influence tree and orchard yields and profitability. This information assists in recognition and correction of subtle forms of nickel deficiency in orchards, thus, helping to maximize profitability.

34. Wood, B. W. & Reilly, C. C. (2007) Nickel: Impact on leaf morphology and growth. Pecan South 40: 13-16.

The nutritional physiology of essential micronutrients in pecan, especially that of nickel, is a limiting factor in optimization of physiological efficiency of orchard enterprises. Knowledge by farmers and extension specialists about the role of nickel, a newly recognized micronutrient, is meager. This review summarizes situations where nickel influences mouse-ear and orchard replant maladies affecting many orchards. This information assists in recognition and correction of nickel deficiency in orchards, thus, helping to maximize profitability.

35. Wood, B. W. & Reilly, C. C. (2007) Interaction of nickel and plant disease. In: Mineral Nutrition and Plant Disease (Datnoff, L. E., Elmer, W.H., Huber, D.M., editors., ed.), pp. 217-247. American Phytopathological Society Press, Minneapolis, MN.

It has long been recognized that mineral nutrition of host plants affects resistance to plant diseases or maladies, yet agricultural practitioners often exclude micronutrients as a husbandry strategy component even though their importance to plants for counteracting maladies, disease causing organisms, and facilitating normal plant growth and development is commonly recognized. This general disregard is especially true for nickel (Ni), the essential plant nutrient element to which the vast majority of practitioners are typically least concerned or aware. This lack of attention to Ni is largely because the

amount needed to prevent deficiency is minuscule and almost all soils contain far more Ni than is typically thought necessary for satisfying plant needs. For many crop species, Ni is more appropriately described as a pico- rather than a micronutrient. The relative number of Ni atoms per unit of plant tissue is the lowest of all known essential mineral elements, with the possible exception of molybdenum. Because Ni's role as an essential mineral element in plants is a relatively recent discovery and its deficiency in real-world crop production systems was only recently documented, little is known about Ni related maladies and interactions with disease pathogens and the potential for usage of Ni as a form of biological control of microbial pests. This publication reviews the interaction of Ni with plant maladies or disease within the context of Ni deficiency. Information is presented that indicates that nickel is a key factor affecting production of secondary plant metabolites, and thus influencing plant resistance to diseases.

36. Bai, C., Wood, B. W. & Reilly, C. C. (2008) Insights into the nutritional physiology of nickel. Acta Hort. (ISHS) 772: 365-368.

Nickel (Ni) is essential for plants, yet its physiological role is poorly understood. Ni-deficient and Ni-sufficient pecan [Carya illinoinensis (Wangenh.) K. Koch] trees were compared regarding the impact of Ni nutritional status on reduced nitrogen (N) forms present in xylem sap at spring bud break. High performance liquid chromatography (HPLC) of xylem sap of Ni-sufficient trees found organic reduced N-forms to be primarily ureides (73%; citrulline > xanthine > ureidoglycolate > allantoic acid ' allantoin ' uric acid ' urea), followed by amide-N (26%; asparagine), and amino-N (1%; tryptamine and '-phenylethylamine). Nickel deficiency reduced xylem sap concentration of xanthine, asparagine, and '-phenylethylamine, yet greatly increased citrulline and allantoic acid. These data indicate that pecan is likely a ureide-N transporter and that Ni deficiency potentially disrupts ureide catabolism and Urea Cycle functionality; thus potentially disrupting normal N-cycling during early spring when N reserves are being remobilized to sinks.

37. Wells, M. L. & Wood, B. W. (2008) Foliar boron and nickel applications reduce water-stage fruit-split of pecan. HortScience 43: 1437-1440.

Water-stage fruit-split (WSFS) is a relatively common and often major problem of certain pecan [Carya illinoinensis (Wangenh.) K. Koch] cultivars. This study evaluates the possibility that the malady can be influenced by improving tree micronutrient nutrition. Foliar sprays of boron (B) and nickel (Ni) to WSFS susceptible fruit of 'Cape Fear' and 'Sumner' are evaluated based on the possibility that either B or Ni potentially affects the severity of WSFS exhibited by trees. Although the incidence of WSFS on 'Cape Fear' was unaffected by micronutrient sprays, the severity of WSFS was substantially reduced in each of the three study years by foliar B application and in 2005 by foliar Ni application. Repeated foliar sprays of Ni also reduced WSFS of 'Sumner' fruit. These data indicate that improving either B or Ni nutrition can potentially reduce crop loss due to WSFS in certain orchard situations and provide evidence that insufficient availability of B and/or Ni to developing ovary tissues potentially predisposes developing fruit to WSFS when environmental triggers occur.

38. Wood, B. W. (2008) Nickle and plant disease [abstract]. Hort Science 43: 1055.

Knowledge of the nutritional physiology of nickel (Ni) is relatively meager. Accumulating evidence indicates that attention to management of Ni nutrition may potentially benefit yield, quality, disease resistance, and disease control of certain crop species, most notably those transporting ureido-nitrogen forms. Nickel deficiencies can trigger a) physiological diseases such as certain forms of 'mouse-ear', 'little-leaf', and 'orchard replant disease', and b) susceptibility to pathogens. Certain host-pathogen interactions exist in an approximate equilibrium that slightly shifts to favor one or the other as the timely bioavailability of Ni changes. An example is the interaction between day-lily and rust disease, with resistance greatly increasing with improved Ni nutrition. Within the context of disease management, disease suppression with Ni is greatest with resistant genotypes rather than with highly susceptible genotypes. Certain crops appear to become predisposed to infection due to Ni deficiency. Once plants are infected, subsequent stress can aggravate the severity of susceptibility by impairing nutrient acquisition and/or utilization and triggering other diseases. There appears to be little or no indirect effect of Ni on disease when endogenous concentrations are within the sufficiency range, but presence of hidden-hunger-type deficiency potentially influences resistance. A transitory Ni deficiency in crops has the potential to affect disease resistance via primary (Tricarboxylic Acid Cycle, Urea Cycle, and Ureide catabolic pathway) and secondary metabolism (Malonic acid pathway, Mevalonic acid pathway, Methylerythritol phosphate pathway); thus affecting key pathways producing nitrogen-containing secondary products, phenolics and terpenes linked to plant defense. Ni also affects disease via a direct fungicidal effect on certain fungal pathogens (e.g. pecan scab fungus). The manipulation of the Ni component of crop nutrition is potentially an important cultural control facet for plant disease. Accumulating evidence indicates that Ni nutrient management should be an integral component of sustainable horticulture for certain Ni-loving crops.

39. Wood, B. W. (2008) Increasing foliar Zn:Ni or Cu:Ni concentration ratios increase severity of nickel deficiency symptoms [abstract]. Hort Science 43: 1294.

The influence of essential micronutrients on the endogenous bioavailability of Ni is unknown. This study examines the linkage between Ni deficiency and endogenous foliar concentration of Ni, Zn, and Cu. It was hypothesized that expression of morphological symptoms of Ni deficiency by pecan [Carya illinoinensis (Wangenh.) K. Koch] is influenced by the ratio of either Zn, or Cu, to Ni in symptomatic foliage. The hypothesis was tested in a greenhouse study using `Desirable¿ seedlings trees growing in an orchard soil known to cause Ni deficiency in potted trees. Amendment of the potting soil with various amounts of either Zn-sulfate or Cu-sulfate produced seedling trees possessing a variety of Zn:Ni and Cu:Ni concentration ratios, and growth/morphological symptoms exhibiting different degrees of Ni deficiency. Symptomatic trees were later foliar-treated with Ni-malate, from Alyssum sp. biomass; hence, correcting deficiency symptoms in the subsequent year¿s growth. Severity of Ni deficiency was unrelated to foliar Ni concentration, but strongly linked to foliar Zn:Ni or Cu:Ni ratios. Deficiency

symptoms increased sigmoidally with increasing Zn:Ni or Cu:Ni ratio, and were correctable, regardless of the Zn:Ni or Cu:Ni ratio in seedling trees by a single foliar application of Ni-malate extracted from Alyssum biomass. Soil Zn or Cu supplements did not detectably affect foliar Ni concentration. These data indicate that Ni deficiency occurring in pecan orchards is potentially caused by either Zn or Cu fertilization induced reductions in the physiological availability of Ni. A reduction in Ni's bioavailability to physiological process might be due to either competitive inhibition or sequestration.

40. Wagle, P., Smith, M. et al. (2009) Foliar application of nickel and copper on pecan performance. HortScience in press.

The mobilization and conversion of reserve nitrogen (N) is critical for pecans [Carya illinoinensis (Wang.) K. Koch] during early spring when trees begin growing actively. Conversion of N reserves to translocatable forms (amides, amino acids, ureides) is adversely affected by a nickel (Ni) shortage. Nickel is required for urease (EC 3.5.1.5, urea amidohydrolase) activation and appears to function in additional enzymes affecting N metabolism. The orchard used for study received an unusually high amount of N annually from nitrate contaminated irrigation water. High N has induced copper (Cu) deficiency by inhibiting transport in plants. Copper, Ni and Ni + Cu were applied as a foliar spray application at the parachute stage of leaf development (19 April), followed by two additional applications at two weeks intervals. Nickel and Ni + Cu increased the weight per nut. Kernel percent and quality were not affected by treatment. Kernel necrosis, a malady characterized by necrotic tissue at the basal end of the kernel, was not affected by treatment. Pecan kernels contain about 70% oil that is compartmentalized in oil bodies. Leakage of these bodies into intercellular spaces, termed opalescence, affects appearance and reduces shelf-life. Treatments increased opalescence compared to the control, perhaps the result of increased oil content. The Ni + Cu treatment reduced yield and yield efficiency; whereas, other treatments were similar to the control. Trunk growth and area per leaf were increased by Ni application, but specific leaf weight was unaffected. Leaf P, K, Ca, Mg, Fe, and Mn were not affected by treatment, but Cu and Zn concentration of foliage and nuts were affected. Results indicate that foliar Ni application was beneficial, but Cu was not, in this particular orchard situation. Zinc sulfate was applied as a standard management practice to the control and was combined with Ni, Cu, and Ni + Cu treatments. Results indicate the possibility that combining Zn, Ni and Cu in the foliar application may reduce yield via either an excess salt-effect or by competitive inhibition of the timely physiological availability of a key nutrient.

41. Pane, E. F., Richards, J. G. & Wood, C. M. (2003) Acute waterborne nickel toxicity in the rainbow trout (Oncorhynchus mykiss) occurs by a respiratory rather than ionoregulatory mechanism. Aquat Toxicol 63: 65-82.

The acute mechanism of toxicity of waterborne nickel (Ni) was investigated in the rainbow trout (Oncorhynchus mykiss) in moderately hard (approximately 140 mg l(-1) as CaCO(3)) Lake Ontario water, where the 96-h LC(50) for juvenile trout (1.5-3.5 g) was 15.3 mg (12.7-19.0, 95% C.L.) dissolved Ni l(-1). No marked impact of Ni exposure on

average unidirectional or net fluxes of Na(+), Cl(-), or Ca(2+) was observed in juvenile trout exposed for 48-60 h to 15.6 mg Ni l(-1) as NiSO(4). Furthermore, when adult rainbow trout (200-340 g) were fitted with indwelling dorsal aortic catheters and exposed for 117 h to 11.6 mg Ni l(-1) as NiSO(4), plasma ions (Na(+), Cl(-), Ca(2+), and Mg(2+)) were all well conserved. However, mean arterial oxygen tension dropped gradually to approximately 35% of control values. This drop in P(aO(2)) was accompanied by an acidosis primarily of respiratory origin. P(aCO(2)) rose to more than double control values with a concomitant drop in arterial pH of 0.15 units. Acute respiratory toxicity was further evidenced by a significant increase in hematocrit (Ht), and plasma lactate, and a significant decrease in spleen hemoglobin (Hb). Following 117 h of exposure to 11.6 mg Ni l(-1), the gill, intestine, plasma, kidney, stomach, and heart accumulated Ni significantly, with increases of 60, 34, 28, 11, 8, and 3-fold, respectively. Brain, white muscle, liver, and bile did not significantly accumulate Ni. Plasma Ni exhibited a remarkable linear increase with time to levels approximately 30-fold higher than controls. We conclude that in contrast to most other metals, Ni is primarily a respiratory, rather than an ionoregulatory, toxicant at exposure levels close to the 96-h LC(50). The implications of a waterborne metal as an acute respiratory toxicant (as opposed to ionoregulatory toxicants such as Cu, Ag, Cd, or Zn) with respect to toxicity modeling are discussed.

42. Pane, E. F., Haque, A. et al. (2004) The physiological consequences of exposure to chronic, sublethal waterborne nickel in rainbow trout (Oncorhynchus mykiss): exercise vs resting physiology. J Exp Biol 207: 1249-1261.

In rainbow trout (Oncorhynchus mykiss), following chronic (42 day) exposure to both 384 microg Ni l(-1) and 2034 microg Ni l(-1), Ni accumulation was greatest in the gill, kidney and plasma, with the plasma as the main sink for Ni. Indeed, trapped plasma analysis revealed that extensive loading of Ni in the plasma accounted for substantial percentages of accumulated Ni in several tissues including the liver and heart. Accumulated Ni in the gill and kidney was less dependent on plasma Ni concentration, suggesting a more intracellular accumulation of Ni in these tissues. We present evidence for a clear, persistent cost of acclimation to chronic, sublethal Ni exposure. Chronic (40-99 day) exposure to sublethal waterborne Ni (243-394 microg Ni l(-1); approximately 1% of the 96 h LC(50)) impaired the exercise physiology, but not the resting physiology, of rainbow trout. Ni acted as a limiting stressor, decreasing maximal rates of oxygen consumption (MO2,max) during strenuous exercise in trout exposed for 34 days to sublethal Ni. This drop in high-performance gas exchange was attributed mainly to a reduction in relative branchial diffusing capacity (D(rel)) caused by thickening of secondary lamellae. Morphometric analysis of the gills of chronically exposed fish revealed overall swelling of secondary lamellae, as well as hypertrophic respiratory epithelia within secondary lamellae. Additionally, contraction of the lamellar blood pillar system and narrowing of interlamellar water channels occurred, possibly contributing to decreased high-performance gas exchange. Decreased aerobic capacity persisted in fish previously exposed to nickel despite a clean-water exposure period of 38 days and an almost complete depuration of gill Ni, suggesting that extrabranchial mechanisms of chronic Ni toxicity may also be important. Chronic impairment of such a

dynamically active and critical organ as the gill may depress the overall fitness of a fish by impairing predator avoidance, prey capture and migration success with obvious environmental implications.

43. Pane, E. F., Haque, A. & Wood, C. M. (2004) Mechanistic analysis of acute, Ni-induced respiratory toxicity in the rainbow trout (Oncorhynchus mykiss): an exclusively branchial phenomenon. Aquat Toxicol 69: 11-24.

In moderately hard Lake Ontario water (approximately 140 mg L(-1) as CaCO3) waterborne Ni (9.7-10.7 mg Ni L(-1)) is acutely toxic to adult rainbow trout (Oncorhynchus mykiss) exclusively via branchial mechanisms. Ventilation in resting trout (evaluated using a ventilatory masking technique) was adversely affected, as ventilation rate (VR), ventilation volume (VG), opercular stroke volume (VSV) and resting oxygen consumption (MO2) were all increased, and oxygen extraction efficiency (U%) decreased over 48 h of Ni exposure. Extensive gill Ni accumulation (41-fold over control levels) during 82 h of waterborne Ni exposure resulted in marked ultrastructural damage to the respiratory epithelium of the gill, including swelling of the secondary lamellae evidenced by changes to both the lamellar region (increased secondary lamellar tissue volume (VSL/V(LR), and to the secondary lamellae themselves (increased volume of tissue lying outside the pillar system (VOPS/VSL). Additionally, decreased lamellar height and increased lamellar width indicated a reduction in lamellar surface area available for gas diffusion. The relative diffusing capacity of experimental fish was only 59% of that of control fish. Infusion of Ni into the blood, achieving a similar time course and magnitude of plasma [Ni] elevation to that during waterborne exposure, failed to elicit any signs of respiratory toxicity typically diagnostic of acute, high level waterborne Ni exposure. Infusion of Ni into the blood for 96 h resulted in only minor accumulation of Ni in the gill, suggesting that acute Ni-induced respiratory toxicity is related to accumulation of high levels of Ni in the gill from the water. Additionally, infusion of Ni into the bloodstream led to significant extrabranchial Ni accumulation only in the kidney. White muscle, heart, liver, stomach, and intestine did not significantly accumulate Ni following infusion into the bloodstream and trapped plasma analysis revealed that, with the exception of the kidney, a substantial portion of Ni accumulated in tissues following infusion could be accounted for by extracellular (blood-bound) Ni.

44. Pane, E. F., Bucking, C. et al. (2005) Renal function in the freshwater rainbow trout (Oncorhynchus mykiss) following acute and prolonged exposure to waterborne nickel. Aquat Toxicol 72: 119-133.

Renal function was investigated in adult rainbow trout following acute and prolonged exposure to waterborne Ni in moderately hard Lake Ontario water (approximately 140 mgL(-1) as CaCO3). Fish were exposed for 36 days to a sublethal concentration of 442 microg Ni L(-1), followed by 96 h of exposure to 12,850 microg Ni L(-1) (approximately 33% of the 96 h LC50). Prolonged exposure markedly affected only the renal handling of Ni, with no substantial effect on the plasma concentration, urinary excretion rate (UER) or clearance ratio (CR) of Na+, Cl-, K+, Ca2+, Mg2+, inorganic phosphate (P(i)), glucose, lactate, total ammonia (T(amm)), protein and free amino acids (FAA).

Glomerular filtration rate (GFR) was reduced by 75% over 96 h of acute Ni challenge in both fish previously exposed to Ni and naive fish, with no significant change in urine flow rate (UFR), suggesting a substantial reduction in water reabsorption to maintain urine flow and water balance. Renal Mg2+ handling was specifically impaired by acute Ni challenge, leading to a significantly increased UER(Mg2+) and significantly decreased plasma [Mg2+] only in naive fish. Previously-exposed fish were well-protected against Ni-induced Mg2+ antagonism, indicating true acclimation to Ni. Only in naive, acutely challenged fish was there an increased UER of titratable acidity (TA-HCO3), net acidic equivalents, P(i), T(amm) and K+. Again, all of these parameters were well-conserved in previously-exposed fish during acute Ni exposure, strongly suggesting that prolonged, sublethal exposure protected against acute Ni-induced respiratory toxicity.

45. Pane, E. F., Glover, C. N. et al. (2006) Characterization of Ni transport into brush border membrane vesicles (BBMVs) isolated from the kidney of the freshwater rainbow trout (Oncorhynchus mykiss). Biochim Biophys Acta 1758: 74-84.

The transport of nickel (Ni) across the renal brush border membrane of the rainbow trout was examined in vitro using brush border membrane vesicles (BBMVs). Both transmembrane transport of Ni into an osmotically active intravesicular space, and binding of Ni to the brush border membrane itself, were confirmed. Nickel (Ni) uptake fitted a two component kinetic model. Saturable, temperature-dependent transport dominated at lower Ni concentrations, with a moderate linear diffusive component of Ni transport apparent at higher Ni concentrations. An affinity constant (K(m)) for Ni transport within the specifically described vesicular media was calculated as 17.9+/-1.9 microM, the maximal rate of transport (J(max)) was calculated as 108.3+/-3.7 nmol mg protein(-1) min(-1), and the slope of the linear diffusive component was calculated as 0.049+/-0.005 nmol mg protein(-1) min(-1) per microM of Ni. Efflux of Ni from BBMVs was fitted to an exponential decay curve with a half-time (T(1/2)) of 125.2+/-7.3 s. Ni uptake into renal BBMVs was inhibited by magnesium at a 100:1 Mg to Ni molar ratio, and by magnesium and calcium at a 1000:1 molar ratio. In the presence of histidine at a 100:1 histidine to Ni ratio, Ni uptake was almost completely abolished. At a 1:1 molar ratio, histidine inhibited Ni uptake by approximately 50%. Ni-histidine complexation was rapid, with a T(1/2) of 12.2 s describing the Ni-histidine equilibration time needed to inhibit Ni uptake into renal BBMVs by 50%. Characterization of Ni transport across cellular membranes is an important step in understanding both the processes underlying homeostatic regulation of Ni, and the toxicological implications of excessive Ni exposure in aquatic ecosystems.

46. Pane, E. F., McDonald, M. D. et al. (2006) Hydromineral balance in the marine gulf toadfish (Opsanus beta) exposed to waterborne or infused nickel. Aquat Toxicol 80: 70-81.

The effects of acute Ni exposure on the marine gulf toadfish (Opsanus beta) were investigated via separate exposures to waterborne nickel (Ni) and arterially infused Ni. Of the plasma electrolytes measured after 72 h of waterborne exposure (215.3 and

606.1 microM Ni in SW (salinity of 34)), only plasma [Ca2+] was significantly impacted (approximately 55% decrease at both exposure concentrations). At both exposure concentrations, plasma [Ni] was regulated for 24h, after which a linear accumulation over time occurred. Accumulation of Ni in the plasma, and in tissues in direct contact with seawater (gill, stomach, and intestine), was roughly proportional to the Ni concentration of the exposure water. Hydromineral balance in the intestinal fluid (IF) was markedly impacted, with Na(+), Cl(-), SO(4)(2-), K+, and Mg2+ concentrations elevated after 72 h of exposure to waterborne Ni. Following arterial Ni infusion (0.40 micromolNikg(-1)h(-1)), perturbation of hydromineral balance of the intestinal fluid was specific only to Na+ (significantly elevated by Ni infusion) and Mg2+ (significantly decreased by Ni infusion). Nitrogen excretion was not significantly impacted by Ni infusion. In all tissues save the kidney, Ni accumulation via infusion was only a fraction of that observed during waterborne exposures. Remarkably, the kidney Ni burden following infusion was almost identical to that resulting from both waterborne exposures, suggesting homeostatic control. Ni excretion, dominated at 24 h by extrarenal routes, was primarily a function of renal excretion by 72 h of infusion. The sum excretion from infused toadfish was relatively efficient, accounting for over 40% of the infused dose by 72 h. Mechanistic knowledge of the mechanisms of toxicity of waterborne Ni in marine systems is a critical component to the development of physiologically based modeling approaches to accurately predict Ni toxicity in marine and estuarine ecosystems.

47. Pane, E. F., Patel, M. & Wood, C. M. (2006) Chronic, sublethal nickel acclimation alters the diffusive properties of renal brush border membrane vesicles (BBMVs) prepared from the freshwater rainbow trout. Comp Biochem Physiol C Toxicol Pharmacol 143: 78-85.

Brush border membrane vesicles (BBMVs) were prepared from the kidneys of rainbow trout exposed acutely (72 h; 13,380 microg Ni L(-1)), chronically (11 months; 289 microg Ni L(-1)), or chronically and acutely, to waterborne nickel (Ni). Uptake of 63Ni into renal BBMVs was temperature-dependent and fitted a two component kinetic model composed of a saturable, Michaelis-Menten component prominent at lower Ni concentrations, and a moderate linear diffusive component apparent at higher Ni concentrations. Chronic Ni exposure reduced the permeability of the BBM to Ni, evidenced by a significantly reduced slope of the linear diffusive component of BBMV uptake. Efflux of Ni from 63Ni-loaded renal BBMVs was not significantly altered by acute Ni challenge. Both Ca2+ and Mg2+ inhibited uptake of Ni into renal BBMVs when present at a molar ratio to Ni of 1000:1. Mg2+-induced inhibition, however, was concentration-dependent and significant in BBMVs prepared from chronically Ni-acclimated fish at far lower molar ratios of 100 and 10 to 1. The data suggest that subtle, long-term modulation of membrane structure and function in the rainbow trout may be a compensatory response to chronic waterborne Ni exposure. Additionally, the data challenge the assumptions of constancy of the physiological parameters underlying physiologically based approaches to modeling metal toxicity. Such approaches are currently employed to derive water quality criteria for some metals.

48. Chowdhury, M. J., Bucking, C. & Wood, C. M. (2008) Is nickel an essential metal for aquatic animals? Integr Environ Assess Manag 4: 266-267.

Overall, the previously mentioned studies suggest that Ni is subject to homeostatic regulation in aquatic animals or at least in the rainbow trout. This is only one characteristic property of known essential metals. Further research is necessary on regulatory physiology and biological requirement to prove whether Ni is essential to aquatic animals. Environmental risk assessment of essential elements requires a different approach compared to nonessential metals because both deficiency and excess require proper attention in setting environmental quality criteria. Therefore, it is important that the essentiality of Ni is known so that regulatory guidelines are determined based on optimal concentrations for organisms.

49. Chowdhury, M. J., Bucking, C. & Wood, C. M. (2008) Pre-exposure to waterborne nickel downregulates gastrointestinal nickel uptake in rainbow trout: indirect evidence for nickel essentiality. Environ Sci Technol 42: 1359-1364.

Nickel (Ni) may be both a toxicant and a micronutrient, but its essentiality to aquatic animals is not established. Interactions between branchial and gastrointestinal routes of metal uptake are important for understanding metal regulation and essentiality in aquatic animals. Adult rainbowtrout (Oncorhynchus mykiss) were pre-exposed to a sublethal concentration of waterborne Ni (7.43 micromol L(-1)) or a control water (0.12 micromol L(-1)) for 45 days, and subsequently, a gastrointestinal dose of radiolabeled Ni (1.08 micromol kg(-1) wet wt) was infused into the stomach of both non-pre-exposed and Ni pre-exposed trout to test whether pre-exposure to waterborne Ni would affect gastrointestinal uptake. The fish pre-exposed to waterborne Ni exhibited a markedly greater level of total Ni in the blood plasma (approximately 10-fold) but not in red blood cells (RBC). Pre-exposure downregulated the gastrointestinal uptake of radiolabeled Ni (new Ni) in the plasma and RBCs, providing evidence for the first time of homeostatic interaction between the two routes of Ni uptake. The plasma and RBC concentrations of new Ni in the non-pre-exposed and Ni pre-exposed groups were linear in the first 2 h and then approached a plateau. Only a small fraction of the infused dose (1.6-3.7%) was found in the internal organs of both groups at 24 h. Waterborne Ni, but not the infused Ni, greatly increased total Ni levels in the gills (6.1 fold), kidney (5.6 fold), scales (4.2 fold), and gut tissues (1.5-4.2 fold). It appears that gut, kidney and scales play important roles for Ni homeostasis by providing uptake, clearance and storage sites. Overall, our results suggest that Ni is subject to homeostatic regulation in the rainbow trout, a property that is characteristic of essential metals.

50. Pandey, S., Parvez, S. et al. (2008) Effects of exposure to multiple trace metals on biochemical, histological and ultrastructural features of gills of a freshwater fish, Channa punctata Bloch. Chem Biol Interact 174: 183-192.

The trace metals are frequently encountered as mixtures of essential and non-essential elements. Therefore, evaluation of their toxic effects individually does not offer a realistic estimate of their impact on biological processes. We studied effects of a mixture

of four essential and toxic metals (Cu, Cd, Fe and Ni) on biochemical and morphological characteristics of the gills of a biomarker freshwater fish Channa punctata (Bloch) using environmentally relevant concentrations. Fish were exposed to metal mixture through tank water for 7, 15 and 30 days. Biochemical studies as well as light microscopy (LM) and scanning electron microscopy (SEM) revealed significant metal exposure-induced alterations in gills. Besides ultastructural changes, activities of antioxidant enzymes such catalase (CAT), glutathione S-transferase (GST) and superoxide dismutase (SOD) were significantly altered in the gills of exposed fish. The reduced glutathione (GSH) was significantly ($p<0.001$) decreased, while lipid peroxidation (LPO) was significantly ($p<0.001$) increased. The main alterations in general morphology of fish gills included spiking and fusion of secondary lamellae, formation of club-shaped filaments, and vacuolization and necrosis of filament epithelium in the interlamellar regions. SEM studies showed gradual increase of the density and apical surface area of the chloride cells and transformation of the surface structure of the pavement cells. The results of this study indicate adaptive as well a toxic responses in fish gills exposed to mixture of trace metals. Low concentrations of trace metal appear to compromise the antioxidant defense of gills. Lesions in the gill morphology caused by the effect of low concentrations of trace metals could lead to functional alterations and interference with fundamental processes such as maintenance of osmoregulation, gas exchange and xenobiotic metabolism in the exposed fish populations.

51. Nadella, S. R., Fitzpatrick, J. L. et al. (2009) Toxicity of dissolved Cu, Zn, Ni and Cd to developing embryos of the blue mussel (Mytilus trossolus) and the protective effect of dissolved organic carbon. Comp Biochem Physiol C Toxicol Pharmacol 149: 340-348.

Marine water quality criteria for metals are largely driven by the extremely sensitive embryo-larval toxicity of Mytilus sp. Here we assess the toxicity of four dissolved metals (Cu, Zn, Ni, Cd) in the mussel Mytilus trossolus, at various salinity levels while also examining the modifying effects of dissolved organic carbon (DOC) on metal toxicity. In 48 h embryo development tests in natural seawater, measured EC50 values were 6.9-9.6 microg L(-1) (95% C.I.=5.5-10.8 microg L(-1)) for Cu, 99 microg L(-1) (86-101) for Zn, 150 microg L(-1) (73-156) for Ni, and 502 microg L(-1) (364-847) for Cd. A salinity threshold of >20 ppt (approximately 60% full strength seawater) was required for normal control development. Salinity in the 60-100% range did not alter Cu toxicity. Experimental addition of dissolved organic carbon (DOC) from three sources reduced Cu toxicity; for example the EC50 of embryos developing in seawater with 20 mg C L(-1) was 39 microg Cu L(-1) (35.2-47.2) a 4-fold increase in Cu EC50. The protective effects of DOC were influenced by their distinct physicochemical properties. Protection appears to be related to higher fulvic acid and lower humic acid content as operationally defined by fluorescence spectroscopy. The fact that DOC from freshwater sources provides protection against Cu toxicity in seawater suggests that extrapolation from freshwater toxicity testing may be possible for saltwater criteria development, including development of a saltwater Biotic Ligand Model for prediction of Cu toxicity.

5

Appendix D

MSDS's
Applicable Nickel Salts

MSDS Material Safety Data Sheet

From: Mallinckrodt Baker, Inc.
222 Red School Lane
Phillipsburg, NJ 08865

Mallinckrodt CHEMICALS J.T.Baker

24 Hour Emergency Telephone: 908-859-2151
CHEMTREC: 1-800-424-9300

National Response in Canada
CANUTEC: 613-996-6666

Outside U.S. and Canada
Chemtrec: 703-527-3887

NOTE: CHEMTREC, CANUTEC and National Response Center emergency numbers to be used only in the event of chemical emergencies involving a spill, leak, fire, exposure or accident involving chemicals.

All non-emergency questions should be directed to Customer Service (1-800-582-2537) for assistance.

NICKEL SULFATE

1. Product Identification

Synonyms: Nickel (II) sulfate hexahydrate (1:1:6); sulfuric acid, nickel (2+) salt, hexahydrate
CAS No.: 7786-81-4 Anhydrous; (10101-97-0 Hexahydrate)
Molecular Weight: 262.88
Chemical Formula: NiSO4 6H2O
Product Codes:
J.T. Baker: 2808
Mallinckrodt: 6400

2. Composition/Information on Ingredients

Ingredient	CAS No	Percent	Hazardous
Nickel Sulfate	7786-81-4	90 - 100%	Yes

3. Hazards Identification

Emergency Overview

WARNING! HARMFUL IF SWALLOWED OR INHALED. CAUSES IRRITATION TO SKIN, EYES AND RESPIRATORY TRACT. MAY CAUSE ALLERGIC SKIN OR RESPIRATORY REACTION. CANCER HAZARD. CAN CAUSE CANCER. Risk of cancer depends on duration and level of exposure.

SAF-T-DATA$^{(tm)}$ Ratings (Provided here for your convenience)

Health Rating: 3 - Severe (Cancer Causing)
Flammability Rating: 0 - None
Reactivity Rating: 0 - None
Contact Rating: 3 - Severe (Life)
Lab Protective Equip: GOGGLES & SHIELD; LAB COAT & APRON; VENT HOOD; PROPER GLOVES
Storage Color Code: Blue (Health)

Potential Health Effects

Inhalation:
Causes irritation to the respiratory tract. Symptoms may include coughing, sore throat, and shortness of breath. Lung damage may result from a single high exposure or lower repeated exposures. Lung allergy occasionally occurs, with asthma type symptoms.
Ingestion:
Toxic. Symptoms may include abdominal pain, diarrhea, nausea, and vomiting. Absorption is poor, but should it occur, symptoms may include giddiness, capillary damage, myocardial weakness, central nervous system depression, and kidney and liver damage.
Skin Contact:
Causes irritation. May cause skin allergy with itching, redness or rash. Some individuals may become sensitized to the substance and suffer "nickel itch", a form of dermatitis.
Eye Contact:
Causes irritation, redness, and pain.
Chronic Exposure:
Prolonged or repeated exposure to excessive concentrations may affect lungs, liver and kidneys. Chronic exposure to nickel and nickel compounds is associated with cancer.
Aggravation of Pre-existing Conditions:
Persons with pre-existing skin disorders, impaired respiratory or pulmonary function, or with a history of asthma, allergies, or sensitization to nickel compounds may be at an increased risk upon exposure to this substance.

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4. First Aid Measures

Inhalation:
Remove to fresh air. If not breathing, give artificial respiration. If breathing is difficult, give oxygen. Get medical attention.
Ingestion:
Induce vomiting immediately as directed by medical personnel. Never give anything by mouth to an unconscious person. Get medical attention.
Skin Contact:

Wipe off excess material from skin then immediately flush skin with plenty of water for at least 15 minutes. Remove contaminated clothing and shoes. Get medical attention. Wash clothing before reuse. Thoroughly clean shoes before reuse.

Eye Contact:

Immediately flush eyes with plenty of water for at least 15 minutes, lifting lower and upper eyelids occasionally. Get medical attention immediately.

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5. Fire Fighting Measures

Fire: Not considered to be a fire hazard.

Explosion: Not considered to be an explosion hazard.

Fire Extinguishing Media: Use any means suitable for extinguishing surrounding fire.

Special Information: In the event of a fire, wear full protective clothing and NIOSH-approved self-contained breathing apparatus with full facepiece operated in the pressure demand or other positive pressure mode.

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6. Accidental Release Measures

Ventilate area of leak or spill. Wear appropriate personal protective equipment as specified in Section 8. Spills: Sweep up and containerize for reclamation or disposal. Vacuuming or wet sweeping may be used to avoid dust dispersal. US Regulations (CERCLA) require reporting spills and releases to soil, water and air in excess of reportable quantities. The toll free number for the US Coast Guard National Response Center is (800) 424-8802.

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7. Handling and Storage

Keep in a tightly closed container, stored in a cool, dry, ventilated area. Protect against physical damage. Isolate from incompatible substances. Areas in which exposure to nickel metal or soluble nickel compounds may occur should be identified by signs or appropriate means, and access to the area should be limited to authorized persons. Wear special protective equipment (Sec. 8) for maintenance break-in or where exposures may exceed established exposure levels. Wash hands, face, forearms and neck when exiting restricted areas. Shower, dispose of outer clothing, change to clean garments at the end of the day. Avoid cross-contamination of street clothes. Wash hands before eating and do not eat, drink, or smoke in workplace. Containers of this material may be hazardous when empty since they retain product residues (dust, solids); observe all warnings and precautions listed for the product.

---

8. Exposure Controls/Personal Protection

Airborne Exposure Limits:

-OSHA Permissible Exposure Limit (PEL): soluble Nickel compounds as Ni: 1 mg/m3 (TWA)

ACGIH Threshold Limit Value (TLV): soluble Nickel compounds as Ni: 0.1 mg/m3 (TWA), A4 - Not classifiable as a human carcinogen

Ventilation System:
A system of local and/or general exhaust is recommended to keep employee exposures below the Airborne Exposure Limits. Local exhaust ventilation is generally preferred because it can control the emissions of the contaminant at its source, preventing dispersion of it into the general work area. Please refer to the ACGIH document, *Industrial Ventilation, A Manual of Recommended Practices*, most recent edition, for details.
Personal Respirators (NIOSH Approved):
If the exposure limit is exceeded and engineering controls are not feasible, a half facepiece particulate respirator (NIOSH type N95 or better filters) may be worn for up to ten times the exposure limit or the maximum use concentration specified by the appropriate regulatory agency or respirator supplier, whichever is lowest.. A full-face piece particulate respirator (NIOSH type N100 filters) may be worn up to 50 times the exposure limit, or the maximum use concentration specified by the appropriate regulatory agency, or respirator supplier, whichever is lowest. If oil particles (e.g. lubricants, cutting fluids, glycerine, etc.) are present, use a NIOSH type R or P filter. For emergencies or instances where the exposure levels are not known, use a full-facepiece positive-pressure, air-supplied respirator. WARNING: Air-purifying respirators do not protect workers in oxygen-deficient atmospheres.
Skin Protection:
Rubber or neoprene gloves and additional protection including impervious boots, apron, or coveralls, as needed in areas of unusual exposure.
Eye Protection:
Use chemical safety goggles and/or full face shield where dusting or splashing of solutions is possible. Maintain eye wash fountain and quick-drench facilities in work area.
Other Control Measures:
Eating, drinking, and smoking should not be permitted in areas where solids or liquids containing soluble nickel compounds are handled, processed, or stored. NIOSH recommends pre-placement and periodic medical exams, with maintaining of records for all employees exposed to nickel in the workplace.

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9. Physical and Chemical Properties

Appearance: Blue-green crystals.
Odor: Odorless.
Solubility: 75.6 g/100 cc water @ 15.5C
Specific Gravity: 2.03
pH: ca. 4.5
% Volatiles by volume @ 21C (70F): 0
Boiling Point: 103C (217F) (Loses 6H2O)
Melting Point: 100C (212F) Loses water
Vapor Density (Air=1): No information found.
Vapor Pressure (mm Hg): No information found.
Evaporation Rate (BuAc=1): No information found.

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10. Stability and Reactivity

Stability: Stable under ordinary conditions of use and storage. At 53.3C (128F), the substance undergoes transition to transparent green crystals. Solutions are acidic.
Hazardous Decomposition Products: Burning may produce sulfur oxides.
Hazardous Polymerization: Will not occur.

Incompatibilities: Strong acids.
Conditions to Avoid: Incompatibles.

11. Toxicological Information

Oral rat LD50: 264 mg/kg. Investigated as a tumorigen, mutagen, reproductive effector.

--------\Cancer Lists\----------

Ingredient	---NTP Carcinogen--- Known	Anticipated	IARC Category
Nickel Sulfate (7786-81-4)	No	Yes	1

12. Ecological Information

Environmental Fate:
No information found.
Environmental Toxicity:
Dangerous to the environment. Very toxic to aquatic organisms; may cause long term adverse effects in the aquatic environment.

13. Disposal Considerations

Whatever cannot be saved for recovery or recycling should be managed in an appropriate and approved waste disposal facility. Processing, use or contamination of this product may change the waste management options. State and local disposal regulations may differ from federal disposal regulations. Dispose of container and unused contents in accordance with federal, state and local requirements.

14. Transport Information

Domestic (Land, D.O.T.)

Proper Shipping Name: RQ, ENVIRONMENTALLY HAZARDOUS SUBSTANCES, SOLID, N.O.S. (NICKEL SULFATE)
Hazard Class: 9
UN/NA: UN3077
Packing Group: III
Information reported for product/size: 100LB

International (Water, I.M.O.)

Proper Shipping Name: ENVIRONMENTALLY HAZARDOUS SUBSTANCES, SOLID, N.O.S. (NICKEL SULFATE)

Hazard Class: 9
UN/NA: UN3077
Packing Group: III
Information reported for product/size: 100LB

International (Air, I.C.A.O.)

Proper Shipping Name: ENVIRONMENTALLY HAZARDOUS SUBSTANCES, SOLID, N.O.S. (NICKEL SULFATE)
Hazard Class: 9
UN/NA: UN3077
Packing Group: III
Information reported for product/size: 100LB

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15. Regulatory Information

--------\Chemical Inventory Status - Part 1\--------------------------------

Ingredient	TSCA	EC	Japan	Australia
Nickel Sulfate (7786-81-4)	Yes	Yes	Yes	Yes

--------\Chemical Inventory Status - Part 2\--------------------------------

Ingredient	Korea	--Canada-- DSL	NDSL	Phil.
Nickel Sulfate (7786-81-4)	Yes	Yes	No	Yes

--------\Federal, State & International Regulations - Part 1\----------------

Ingredient	-SARA 302- RQ	TPQ	------SARA 313------ List	Chemical Catg.
Nickel Sulfate (7786-81-4)	No	No	No	Nickel compo

--------\Federal, State & International Regulations - Part 2\----------------

Ingredient	CERCLA	-RCRA- 261.33	-TSCA- 8(d)
Nickel Sulfate (7786-81-4)	100	No	No

Chemical Weapons Convention: No TSCA 12(b): No CDTA: No
SARA 311/312: Acute: Yes Chronic: Yes
Fire: No Pressure: No Reactivity: No (Pure / Solid)

WARNING: THIS PRODUCT CONTAINS A CHEMICAL(S) KNOWN TO THE STATE OF CALIFORNIA TO CAUSE CANCER.

Australian Hazchem Code: 2[Z]
Poison Schedule: S6
WHMIS: This MSDS has been prepared according to the hazard criteria of the Controlled Products Regulations (CPR) and the MSDS contains all of the information required by the CPR.

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16. Other Information

NFPA Ratings: Health: **2** Flammability: **0** Reactivity: **0**

Label Hazard Warning:

WARNING! HARMFUL IF SWALLOWED OR INHALED. CAUSES IRRITATION TO SKIN, EYES AND RESPIRATORY TRACT. MAY CAUSE ALLERGIC SKIN OR RESPIRATORY REACTION. CANCER HAZARD. CAN CAUSE CANCER. Risk of cancer depends on duration and level of exposure.

Label Precautions:

Do not get in eyes, on skin, or on clothing.
Do not breathe dust.
Keep container closed.
Use only with adequate ventilation.
Wash thoroughly after handling.

Label First Aid:

If swallowed, induce vomiting immediately as directed by medical personnel. Never give anything by mouth to an unconscious person. In case of contact, wipe off excess material from skin then immediately flush eyes or skin with plenty of water for at least 15 minutes. Remove contaminated clothing and shoes. Wash clothing before reuse. If inhaled, remove to fresh air. If not breathing, give artificial respiration. If breathing is difficult, give oxygen. In all cases, get medical attention.

Product Use:

Laboratory Reagent.

Revision Information:

MSDS Section(s) changed since last revision of document include: 3, 11, 12.

Disclaimer:

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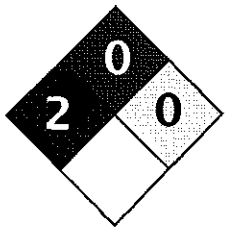
Prepared by: Environmental Health & Safety
Phone Number: (314) 654-1600 (U.S.A.)

ScienceLab.com
Chemicals & Laboratory Equipment



Health	2
Fire	0
Reactivity	0
Personal Protection	E

Material Safety Data Sheet
Nickel carbonate, basic MSDS

Section 1: Chemical Product and Company Identification

Product Name: Nickel carbonate, basic

Catalog Codes: SLN1270, SLN1882

CAS#: 3333-67-3

RTECS: QR6200000

TSCA: TSCA 8(b) inventory: Nickel carbonate, basic

CI#: Not available.

Synonym:

Chemical Name: Not available.

Chemical Formula: NiCO3.2Ni(OH)2.4H2O

Contact Information:

Sciencelab.com, Inc.
14025 Smith Rd.
Houston, Texas 77396

US Sales: **1-800-901-7247**
International Sales: **1-281-441-4400**

Order Online: ScienceLab.com

CHEMTREC (24HR Emergency Telephone), call:
1-800-424-9300

International CHEMTREC, call: 1-703-527-3887

For non-emergency assistance, call: 1-281-441-4400

Section 2: Composition and Information on Ingredients

Composition:

Name	CAS #	% by Weight
Nickel carbonate, basic	3333-67-3	100

Toxicological Data on Ingredients: Nickel carbonate, basic LD50: Not available. LC50: Not available.

Section 3: Hazards Identification

Potential Acute Health Effects:
Very hazardous in case of ingestion, of inhalation. Hazardous in case of skin contact (sensitizer), of eye contact (irritant). Slightly hazardous in case of skin contact (irritant).

Potential Chronic Health Effects:
Very hazardous in case of ingestion, of inhalation.
Hazardous in case of skin contact (sensitizer), of eye contact (irritant).
Slightly hazardous in case of skin contact (irritant).
CARCINOGENIC EFFECTS: Classified A1 (Confirmed for human.) by ACGIH, 1 (Proven for human.) by IARC, + (PROVEN) by OSHA. Classified 2 (Reasonably anticipated.) by NTP.
MUTAGENIC EFFECTS: Not available.
TERATOGENIC EFFECTS: Not available.
DEVELOPMENTAL TOXICITY: Not available.
The substance is toxic to lungs, mucous membranes.
Repeated or prolonged exposure to the substance can produce target organs damage.

Section 4: First Aid Measures

Eye Contact: Check for and remove any contact lenses. Do not use an eye ointment. Seek medical attention.

Skin Contact:
After contact with skin, wash immediately with plenty of water. Gently and thoroughly wash the contaminated skin with running water and non-abrasive soap. Be particularly careful to clean folds, crevices, creases and groin. Cover the irritated skin with an emollient. If irritation persists, seek medical attention.

Serious Skin Contact:
Wash with a disinfectant soap and cover the contaminated skin with an anti-bacterial cream. Seek medical attention.

Inhalation: Allow the victim to rest in a well ventilated area. Seek immediate medical attention.

Serious Inhalation: Not available.

Ingestion:
Do not induce vomiting. Loosen tight clothing such as a collar, tie, belt or waistband. If the victim is not breathing, perform mouth-to-mouth resuscitation. Seek immediate medical attention.

Serious Ingestion: Not available.

Section 5: Fire and Explosion Data

Flammability of the Product: Non-flammable.

Auto-Ignition Temperature: Not applicable.

Flash Points: Not applicable.

Flammable Limits: Not applicable.

Products of Combustion: Not available.

Fire Hazards in Presence of Various Substances: Not applicable.

Explosion Hazards in Presence of Various Substances:
Risks of explosion of the product in presence of mechanical impact: Not available.
Risks of explosion of the product in presence of static discharge: Not available.

Fire Fighting Media and Instructions: Not applicable.

Special Remarks on Fire Hazards: Not available.

Special Remarks on Explosion Hazards: Not available.

Section 6: Accidental Release Measures

Small Spill:
Use appropriate tools to put the spilled solid in a convenient waste disposal container. Finish cleaning by spreading water on the contaminated surface and dispose of according to local and regional authority requirements.

Large Spill:
Use a shovel to put the material into a convenient waste disposal container. Finish cleaning by spreading water on the contaminated surface and allow to evacuate through the sanitary system.

Section 7: Handling and Storage

Precautions:
Keep locked up Do not breathe dust. Wear suitable protective clothing In case of insufficient ventilation, wear suitable respiratory equipment If you feel unwell, seek medical attention and show the label when possible. Avoid contact with skin and eyes

Storage:
Carcinogenic, teratogenic or mutagenic materials should be stored in a separate locked safety storage cabinet or room.

Section 8: Exposure Controls/Personal Protection

Engineering Controls:
Use process enclosures, local exhaust ventilation, or other engineering controls to keep airborne levels below recommended exposure limits. If user operations generate dust, fume or mist, use ventilation to keep exposure to airborne contaminants below the exposure limit.

Personal Protection:
Splash goggles. Lab coat. Dust respirator. Be sure to use an approved/certified respirator or equivalent. Gloves.

Personal Protection in Case of a Large Spill:
Splash goggles. Full suit. Dust respirator. Boots. Gloves. A self contained breathing apparatus should be used to avoid inhalation of the product. Suggested protective clothing might not be sufficient; consult a specialist BEFORE handling this product.

Exposure Limits: Not available.

Section 9: Physical and Chemical Properties

Physical state and appearance: Solid. (Solid crystalline powder.)

Odor: Not available.

Taste: Not available.

Molecular Weight: 376.24 g/mole

Color: Green. (Light.)

pH (1% soln/water): Not applicable.

Boiling Point: Not available.

Melting Point: Decomposes.

Critical Temperature: Not available.

Specific Gravity: 2.6 (Water = 1)

Vapor Pressure: Not applicable.

Vapor Density: Not available.

Volatility: Not available.

Odor Threshold: Not available.

Water/Oil Dist. Coeff.: Not available.

Ionicity (in Water): Not available.

Dispersion Properties: Not available.

Solubility: Insoluble in cold water.

Section 10: Stability and Reactivity Data

Stability: The product is stable.

Instability Temperature: Not available.

Conditions of Instability: Not available.

Incompatibility with various substances: Not available.

Corrosivity: Non-corrosive in presence of glass.

Special Remarks on Reactivity: Not available.

Special Remarks on Corrosivity: Not available.

Polymerization: No.

Section 11: Toxicological Information

Routes of Entry: Eye contact. Inhalation. Ingestion.

Toxicity to Animals:
LD50: Not available.
LC50: Not available.

Chronic Effects on Humans:
CARCINOGENIC EFFECTS: Classified A1 (Confirmed for human.) by ACGIH, 1 (Proven for human.) by IARC, + (PROVEN) by OSHA. Classified 2 (Reasonably anticipated.) by NTP.
The substance is toxic to lungs, mucous membranes.

Other Toxic Effects on Humans:
Very hazardous in case of ingestion, of inhalation.
Hazardous in case of skin contact (sensitizer).
Slightly hazardous in case of skin contact (irritant).

Special Remarks on Toxicity to Animals: Not available.

Special Remarks on Chronic Effects on Humans: Not available.

Special Remarks on other Toxic Effects on Humans: Not available.

Section 12: Ecological Information

Ecotoxicity: Not available.

BOD5 and COD: Not available.

Products of Biodegradation:
Possibly hazardous short term degradation products are not likely. However, long term degradation products may arise.

Toxicity of the Products of Biodegradation: The products of degradation are as toxic as the original product.

Special Remarks on the Products of Biodegradation: Not available.

Section 13: Disposal Considerations

Waste Disposal:

Section 14: Transport Information

DOT Classification: Not a DOT controlled material (United States).

Identification: Not applicable.

Special Provisions for Transport: Not applicable.

Section 15: Other Regulatory Information

Federal and State Regulations:
California prop. 65: This product contains the following ingredients for which the State of California has found to cause cancer, birth defects or other reproductive harm, which would require a warning under the statute: Nickel carbonate, basic
California prop. 65: This product contains the following ingredients for which the State of California has found to cause cancer which would require a warning under the statute: Nickel carbonate, basic
Pennsylvania RTK: Nickel carbonate, basic
Massachusetts RTK: Nickel carbonate, basic
TSCA 8(b) inventory: Nickel carbonate, basic
SARA 313 toxic chemical notification and release reporting: Nickel carbonate, basic
CERCLA: Hazardous substances.: Nickel carbonate, basic

Other Regulations: OSHA: Hazardous by definition of Hazard Communication Standard (29 CFR 1910.1200).

Other Classifications:

WHMIS (Canada): CLASS D-2A: Material causing other toxic effects (VERY TOXIC).

DSCL (EEC):
R36- Irritating to eyes.
R43- May cause sensitization by skin contact.
R45- May cause cancer.

HMIS (U.S.A.):

Health Hazard: 2

Fire Hazard: 0

Reactivity: 0

Personal Protection: E

National Fire Protection Association (U.S.A.):

Health: 2

Flammability: 0

Reactivity: 0

Specific hazard:

Protective Equipment:
Gloves.
Lab coat.
Dust respirator. Be sure to use an approved/certified respirator or equivalent. Wear appropriate respirator when ventilation is inadequate.
Splash goggles.

Section 16: Other Information

References: Not available.

Other Special Considerations: Not available.

Created: 10/10/2005 10:57 AM

Last Updated: 11/06/2008 12:00 PM

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Material Safety Data Sheet

Nickel(II) hydroxide, c.p., 60-61% Ni

MSDS# 53189

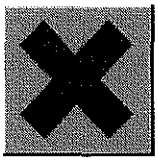
Section 1 - Chemical Product and Company Identification

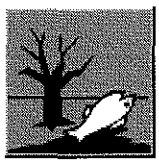
MSDS Name:	Nickel(II) hydroxide, c.p., 60-61% Ni
Catalog Numbers:	AC275020000, AC275020010, AC275020050, AC275022500
Synonyms:	Nickel Dihydroxide; Nickelous Hydroxide.
Company Identification:	Acros Organics BVBA Janssen Pharmaceuticalaan 3a 2440 Geel, Belgium
Company Identification: (USA)	Acros Organics One Reagent Lane Fair Lawn, NJ 07410
For information in the US, call:	800-ACROS-01
For information in Europe, call:	+32 14 57 52 11
Emergency Number, Europe:	+32 14 57 52 99
Emergency Number US:	201-796-7100
CHEMTREC Phone Number, US:	800-424-9300
CHEMTREC Phone Number, Europe:	703-527-3887

Section 2 - Composition, Information on Ingredients

CAS#:	12054-48-7
Chemical Name:	Nickel dihydroxide
%:	100
EINECS#:	235-008-5

Hazard Symbols: XN N





Risk Phrases: 20/22 40 43 50/53

Section 3 - Hazards Identification

EMERGENCY OVERVIEW

Warning! May cause respiratory tract irritation. Harmful if inhaled. May cause allergic skin reaction. Cancer suspect agent. May be harmful if swallowed. May cause eye and skin irritation. May cause cancer based on animal studies. Target Organs: Lungs, skin.

Potential Health Effects

Eye:	May cause eye irritation.
Skin:	May cause skin irritation. Causes "nickel itch" which is a dermatitis resulting from sensitization to nickel, which is characterized by skin eruptions, followed by discrete ulcers that may discharge and become crusted, or by eczema.
Ingestion:	May cause irritation of the digestive tract. May be harmful if swallowed.
Inhalation:	Harmful if inhaled. May cause respiratory tract irritation.
Chronic:	May cause cancer according to animal studies. Symptoms of overexposure to nickel can cause sensitization, dermatitis, allergic asthma and pneumonitis.

Section 4 - First Aid Measures

Eyes: Flush eyes with plenty of water for at least 15 minutes, occasionally lifting the upper and lower eyelids. Get medical aid.

Skin: Get medical aid. Flush skin with plenty of water for at least 15 minutes while removing contaminated clothing and shoes. Wash clothing before reuse.

Ingestion: Get medical aid. Do NOT induce vomiting. If conscious and alert, rinse mouth and drink 2-4 cupfuls of milk or water.

Inhalation: Remove from exposure and move to fresh air immediately. If not breathing, give artificial respiration. If breathing is difficult, give oxygen. Get medical aid. Do NOT use mouth-to-mouth resuscitation. If breathing has ceased apply artificial respiration using oxygen and a suitable mechanical device such as a bag and a mask.

Notes to Physician:

Section 5 - Fire Fighting Measures

General Information: As in any fire, wear a self-contained breathing apparatus in pressure-demand, MSHA/NIOSH (approved or equivalent), and full protective gear. During a fire, irritating and highly toxic gases may be generated by thermal decomposition or combustion.

Extinguishing Media: Use agent most appropriate to extinguish fire. Use water spray, dry chemical, carbon dioxide, or appropriate foam.

Autoignition Temperature: Not applicable.

Flash Point: Not applicable.

Explosion Limits: Lower: Not available

Explosion Limits: Upper: Not available

NFPA Rating: health: 2; flammability: 0; instability: 0;

Section 6 - Accidental Release Measures

General Information: Use proper personal protective equipment as indicated in Section 8.

Spills/Leaks: Vacuum or sweep up material and place into a suitable disposal container. Clean up spills immediately, observing precautions in the Protective Equipment section. Avoid generating dusty conditions. Provide ventilation.

Section 7 - Handling and Storage

Handling: Wash thoroughly after handling. Remove contaminated clothing and wash before reuse. Use with adequate ventilation. Minimize dust generation and accumulation. Avoid contact with eyes, skin, and clothing. Keep container tightly closed. Avoid ingestion and inhalation.

Storage: Store in a tightly closed container. Store in a cool, dry, well-ventilated area away from incompatible substances.

Section 8 - Exposure Controls, Personal Protection

Chemical Name	ACGIH	NIOSH	OSHA - Final PELs
Nickel dihydroxide	0.2 mg/m3 TWA (inhalable fraction, as Ni) (listed under Nickel, inorganic compounds, insoluble).	0.015 mg/m3 TWA (as Ni, except Nickel carbonyl) (listed under Nickel compounds).10 mg/m3 IDLH (as Ni, except Nickel carbonyl) (listed under Nickel compounds).	1 mg/m3 TWA (as Ni) (listed under Nickel soluble compounds).1 mg/m3 TWA (as Ni) (listed under Nickel insoluble compounds).

OSHA Vacated PELs: Nickel dihydroxide: 0.1 mg/m3 TWA (as Ni) (listed under Nickel soluble compounds)1 mg/m3

TWA (as Ni) (listed under Nickel insoluble compounds)

Engineering Controls:

Facilities storing or utilizing this material should be equipped with an eyewash facility and a safety shower. Use adequate ventilation to keep airborne concentrations low.

Exposure Limits

Personal Protective Equipment

Eyes: Wear appropriate protective eyeglasses or chemical safety goggles as described by OSHA's eye and face protection regulations in 29 CFR 1910.133 or European Standard EN166.

Skin: Wear appropriate protective gloves to prevent skin exposure.

Clothing: Wear appropriate protective clothing to prevent skin exposure.

Respirators: A respiratory protection program that meets OSHA's 29 CFR 1910.134 and ANSI Z88.2 requirements or European Standard EN 149 must be followed whenever workplace conditions warrant respirator use.

Section 9 - Physical and Chemical Properties

Physical State: Powder
Color: lime green
Odor: none reported
pH: Not available
Vapor Pressure: Not available
Vapor Density: Not available
Evaporation Rate: Not available
Viscosity: Not available
Boiling Point: Not available
Freezing/Melting Point: 230 deg C (446.00°F)
Decomposition Temperature:
Solubility in water: Negligible
Specific Gravity/Density: 4.15
Molecular Formula: H2NiO2
Molecular Weight: 92.70

Section 10 - Stability and Reactivity

Chemical Stability: Stable under normal temperatures and pressures.

Conditions to Avoid: Incompatible materials, dust generation, excess heat, strong oxidants.

Incompatibilities with Other Materials: Strong acids.

Hazardous Decomposition Products: Carbon monoxide, irritating and toxic fumes and gases, carbon dioxide, nickel oxide.

Hazardous Polymerization: Has not been reported.

Section 11 - Toxicological Information

RTECS#: CAS# 12054-48-7: QR7040000

RTECS:

LD50/LC50: **CAS# 12054-48-7:** Inhalation, rat: LC50 = 1200 mg/m3/4H;
Oral, rat: LD50 = 1515 mg/kg;
Skin, rat: LD50 = >2 gm/kg;

.

Carcinogenicity: Nickel dihydroxide - California: carcinogen, initial date 10/1/89 NTP: Known carcinogen (Nickel compounds). IARC: Group 1 carcinogen

Epidemiology: Epidemiological studies have shown an increased incidence of cancers among nickel refinery workers.An increased incidence of lung and nasal cavity cancers has been noted among women in nickel smelters and refineries.

Teratogenicity: No information found

Reproductive: No information found

Neurotoxicity: No information found

Mutagenicity: No information found

Other: The hazards associated with nickel may be seen in this product.See actual entry in RTECS for complete information.

Section 12 - Ecological Information

Ecotoxicity: Daphnia: Daphnia: 51mg/L; 48H

Other: For more information, see "HANDBOOK OF ENVIRONMENTAL FATE AND EXPOSURE DATA."

Section 13 - Disposal Considerations

Chemical waste generators must determine whether a discarded chemical is classified as a hazardous waste. US EPA guidelines for the classification determination are listed in 40 CFR Parts 261.3. Additionally, waste generators must consult state and local hazardous waste regulations to ensure complete and accurate classification. RCRA P-Series: None listed. RCRA U-Series: None listed.

Section 14 - Transport Information

US DOT
Shipping Name: ENVIRONMENTALLY HAZARDOUS SUBSTANCES, SOLID, N.O.S.
Hazard Class: 9
UN Number: UN3077
Packing Group: III
Canada TDG
Shipping Name: Not available
Hazard Class:
UN Number:
Packing Group:

USA RQ: CAS# 12054-48-7: 10 lb final RQ; 4.54 kg final RQ

Section 15 - Regulatory Information

US Federal

TSCA

CAS# 12054-48-7 is listed on the TSCA Inventory.

Health & Safety Reporting List	None of the chemicals are on the Health & Safety Reporting List.
Chemical Test Rules	None of the chemicals in this product are under a Chemical Test Rule.
Section 12b	None of the chemicals are listed under TSCA Section 12b.
TSCA Significant New Use Rule	None of the chemicals in this material have a SNUR under TSCA.
CERCLA Hazardous Substances and corresponding RQs	CAS# 12054-48-7: 10 lb final RQ; 4.54 kg final RQ
SARA Section 302 Extremely Hazardous Substances	None of the chemicals in this product have a TPQ.
Section 313	This material contains Nickel dihydroxide (listed as Nickel compounds), 100%, (CAS# 12054-48-7) which is subject to the reporting requirements of Section 313 of SARA Title III and 40 CFR Part 372.
Clean Air Act:	CAS# 12054-48-7 listed as Nickel compounds is listed as a hazardous air pollutant (HAP). This material does not contain any Class 1 Ozone depletors. This material does not contain any Class 2 Ozone depletors.
Clean Water Act:	CAS# 12054-48-7 is listed as a Hazardous Substance under the CWA. None of the chemicals in this product are listed as Priority Pollutants under the CWA. CAS# 12054-48-7 is listed as a Toxic Pollutant under the Clean Water Act.
OSHA:	
STATE	Nickel dihydroxide can be found on the following state right to know lists: California, New Jersey, Pennsylvania, Minnesota, (listed as Nickel soluble compounds), Minnesota, (listed as Nickel insoluble compounds), Massachusetts.

California Prop 65: The following statement(s) is(are) made in order to comply with the California Safe Drinking Water Act: WARNING: This product contains Nickel dihydroxide, a chemical known to the state of California to cause cancer.

California No Significant Risk Level: None of the chemicals in this product are listed.

European/International Regulations

European Labeling in Accordance with EC Directives

Hazard Symbols: XN N

Risk Phrases:

R 20/22 Harmful by inhalation and if swallowed.

R 40 Limited evidence of a carcinogenic effect.

R 43 May cause sensitization by skin contact.

R 50/53 Very toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.

Safety Phrases:

S 22 Do not breathe dust.

S 36 Wear suitable protective clothing.

S 60 This material and its container must be disposed of as hazardous waste.

S 61 Avoid release to the environment. Refer to special instructions/safety data sheets.

WGK (Water Danger/Protection)

CAS# 12054-48-7: Not available

Canada

CAS# 12054-48-7 is listed on Canada's DSL List

Canadian WHMIS Classifications: D2A, D2B

This product has been classified in accordance with the hazard criteria of the Controlled Products Regulations and the MSDS contains all of the information required by those regulations.

CAS# 12054-48-7 is listed on Canada's Ingredient Disclosure List

Section 16 - Other Information

MSDS Creation Date: 7/28/1999

Revision #3 Date 10/03/2005
