

**Multiple Chemical Sensitivity: Towards the End of Controversy**

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There are nine well accepted paradigms of human disease. The tenth may explain the features of multiple chemical sensitivity (MCS) and a group of related illnesses including chronic fatigue syndrome (CFS), fibromyalgia (FM) and posttraumatic stress disorder (PTSD); Gulf War syndrome appears to be a combination of all four. The elevated nitric oxide/peroxynitrite vicious cycle paradigm explains most of the most puzzling features of this group of previously unexplained illnesses (1-16) that afflict tens of millions of people in the U.S. and elsewhere. These illnesses have multiple overlaps with each other (2-5,13,16). They share many common symptoms and signs. They are repeatedly reported to be comorbid conditions. Cases of each of them typically show a common pattern of case initiation, with cases being preceded by and presumably induced by a short term stressor, only to be followed by a chronic illness that usually persists for life. These similarities have led many different researchers to propose that two, three or all four of them may share a common etiologic mechanism (3,5,16), but they were unable to suggest what that mechanism might be.

The short term stressors reported to initiate these illnesses are very diverse. Six have very well documented roles as initiators, viral infection, bacterial infection, physical trauma (particularly head and neck trauma), organophosphate/carbamate pesticide exposure, volatile organic solvent exposure and severe psychological stress. There are six additional stressors that are less well documented as initiators of these illnesses and thus may be viewed as candidate initiators. These latter six include pyrethroid pesticide exposure, organochlorine (chlordane or lindane) pesticide exposure, a protozoan infection (toxoplasmosis), ciguatoxin poisoning<sup>#</sup>, carbon monoxide poisoning and thimerosal exposure. All 12 of these are known to be able to initiate a sequence of events leading to increases in nitric oxide levels. Thus they all have a common biochemical end point, suggesting that they may act to initiate these illnesses through a common mechanism (1-5,7,13,16). The three classes of infection all act to raise nitric oxide levels primarily by inducing the inducible nitric oxide synthase (iNOS) whereas most of the others are known to act by increasing NMDA<sup>@</sup> receptor activity and such NMDA activity is. The pesticides involved fall into discrete classes, based both on their chemical structure and biochemical mode of action in both insects and in humans. Organophosphate and carbamate pesticides both act as inhibitors of the enzyme acetylcholinesterase, the enzyme that gets rid of acetylcholine. The pyrethroid pesticides act to open sodium channels in the brain. The organochlorine pesticides act to inhibit what are known as GABA<sub>A</sub> receptors, sites at which the compound GABA acts in the brain. The interesting thing here is that although these all act at different targets in the brain, they all can produce a common response, involving excessive activity of the NMDA receptors in the brain and excessive nitric oxide.

<sup>#</sup>Ciguatoxin is a toxic compound produced by certain tropical organisms which when eaten by tropical fish, make the fish toxic to people who eat them. The toxin called ciguatoxin or ciguatera toxin acts somewhat like the pyrethroid pesticides, leaving open sodium channels in the brain. <sup>@</sup>The NMDA receptors are receptors for glutamate found primarily in the central and peripheral nervous system. They are called NMDA receptors because they are specifically stimulated by the compound N-methyl-D-aspartate whereas

other

known to produce, in turn, increases in nitric oxide and its oxidant product, peroxynitrite. The NMDA activity is known to act by allowing an influx of calcium into the cell, leading to increased activity of the calcium dependent neural nitric oxide synthase (nNOS) activity (5). Thus the stressors do not all share a common pathway or common enzyme producing nitric oxide. What they do appear to share is a common response of increased nitric oxide and its oxidant product peroxynitrite.

So how might elevated levels of nitric oxide and peroxynitrite\*\* initiate these chronic illnesses? The proposed mechanism is that they initiate a biochemical/physiological vicious cycle mechanism which is responsible for both the chronic nature of these illnesses and is responsible for generating their diverse symptoms and signs. That vicious cycle mechanism is diagrammed in the figure on the following page. The arrows in the figure represent a total of 22 distinct mechanisms, 18 of which are quite well documented (1,5,7,13,16). The other 4 are based on what appear to be solid data, but are less established. The overall vicious cycle is quite plausible but what needs to be questioned is its physiological significance to these illnesses. One needs to focus, then, on the role of the various elements of this cycle in the chronic phases of these illnesses and that has been the focus of many of my own papers on this subject (1-7,10,12,13,16). Each of the following has been reported to occur in from two to four of these illnesses and typically when it has not been reported, it has not been studied: Elevated levels of nitric oxide, oxidative stress, elevated NF- $\kappa$ B activity, elevated levels of inflammatory cytokines, elevated NMDA activity, and increased vanilloid sensitivity#&. Intracellular calcium levels have not been studied but some properties produced by such calcium increase have been reported. A pattern of mitochondrial dysfunction characteristic of peroxynitrite-mediated damage has been reported in CFS and FM (1,16). So although it may certainly be argued that further studies are needed on many of these areas, the pattern of evidence that is available is supportive of the predictions of the vicious cycle mechanism. Many of the predictions of the cycle are also supported by studies of certain animal models of these illnesses. There is, for example, convincing published evidence for a key role of both NMDA activity and nitric oxide in certain animal models of MCS and substantial but less convincing evidence in PTSD models as well. There is an animal model of CFS that fits very well with the proposed mechanism but where some of the important predictions have never been tested. While there is no explicitly stated animal model for FM, whose characteristic symptom is widespread pain hypersensitivity, the mechanism of hyperalgesia in animals is known to involve all of the elements of the proposed vicious cycle (16). So in general, although the biochemical and physiological experimental data on these illnesses is limited, what data is available is in good agreement with the predictions of the cycle.

glutamate receptors are not. While the NMDA receptors appear to have the most important role of the glutamate receptors in these illnesses, in some cases other glutamate receptors may also have a role. @Nitric oxide is a compound found in the body that has important functions, particularly in controlling the circulatory system (it dilates the blood vessels), in the brain and in the immune system. However when its levels are too high, it can produce substantial pathophysiological effects, impacting the body in many negative ways. These elevated levels are proposed to be important in these illnesses and also occur a wide variety of chronic inflammatory diseases and in acute inflammatory responses

such as sepsis. Much of the damage produced by excessive nitric oxide is actually a consequence of its oxidant product, peroxynitrite.

• Peroxynitrite is a potent oxidant formed by the reaction of nitric oxide with another compound superoxide. It is a potent oxidant that is thought to break down to produce a number of reactive free radicals and cause various types of oxidative damage. • The vanilloid receptor is the receptor for the "hot" compound in hot peppers, known as capsaicin. We have argued in reference 7 that this receptor has a complex role in MCS and specifically that it is the likely target for volatile organic solvents that produce sensitive responses in that illness. It also is reported to have a role in fibromyalgia and in irritable bowel syndrome but has not been studied in these other illnesses.

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Fig. 1 legend. Vicious cycle diagram. Each arrow represents one or more mechanisms by which the variable at the foot of the arrow can stimulate the level of the variable at the head of the arrow. It can be seen that these arrows form a series of loops that can potentially continue to stimulate each other. An example of this would be that nitric oxide can increase peroxynitrite which can stimulate oxidative stress which can stimulate NF- $\kappa$ B which can increase the production of iNOS which can, in turn increase nitric oxide. This loop alone constitutes a potential vicious cycle and there are a number of other loops, diagrammed in the figure that can collectively make up a much larger vicious cycle. The challenge, according to this view, in these illnesses is to lower this whole pattern of elevations to get back into a normal range.

There are two types of puzzles surrounding the symptoms and signs of these illnesses. One is that they are very diverse, involving neuronal, neuroendocrine, circulatory, immune, biochemical and psychiatric properties. This has raised the question of how any understandable mechanism might be able to generate such a diverse group of symptoms and signs? A second puzzle is that these symptoms and signs are highly variable from one individual to another so both the pattern and the variability require satisfactory explanations. In my book (16) and elsewhere (2), I have provided explanations for 16 different symptoms and signs that are found with reasonable frequency, based on one or more elements of the vicious cycle. It should be noted that these explanations are put forth as plausible mechanisms, not as established mechanisms. They include such things as orthostatic intolerance, possibly caused by nitric oxide effects both as a vasodilator and its effects on the sympathetic nervous system; sleep dysfunction such as non-refreshing sleep, caused by elevated cytokines, by elevated nitric oxide and by elevated NF- $\kappa$ B activity; low NK cell activity, caused by oxidants and specifically by superoxide; fatigue which is found in all conditions with low energy metabolism may be caused by peroxynitrite mediated mitochondrial dysfunction. Even such psychiatric symptoms as anxiety

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(excessive NMDA activity in the amygdala) and depression (nitric oxide effects on the brain, locations still undetermined) may be explained by this mechanism.

The variability of the symptoms and signs may be explained by variation in tissue distribution of the underlying biochemistry. Nitric oxide, superoxide and peroxynitrite have limited diffusion in tissues (16) and the basic mechanisms outlined in the vicious cycle are cellular. It follows that one tissue may be impacted by this biochemistry whereas an adjacent tissue may be largely unaffected. The vicious cycle may propagate

the tissue distribution into the future, thus producing a relatively stable pattern of symptoms and signs which varies from one patient to another. An example of this is that if the amygdala is impacted by this biochemistry, a patient will be expected have symptoms of anxiety and possible panic attacks, but not otherwise. Similarly, if certain regions of the GI tract are impacted you may have irritable bowel syndrome (IBS) symptoms; note that IBS is reported to involve both excessive vanilloid activity and excessive nitric oxide.

#### **Multiple Chemical Sensitivity (MCS):**

Multiple chemical sensitivity is reported to be both the most common of these illnesses and it also has been the most puzzling. It is characterized by exquisite chemical sensitivity to a wide variety of chemicals, with such sensitivity being apparently induced by previous chemical exposure (4,5). There has not previously been an understanding as to how these chemicals act or how the exquisite sensitivity reported, on the order of 1000 times that of normals, can be generated. It has been clear, for some time, that MCS is not caused by an IgE-based allergy or fundamentally by an immune response of any kind, but rather involves neuronal dysfunction.

There are four classes of chemicals reported to commonly produce MCS and also trigger symptoms in those already sensitized. These are the organophosphate/carbamate pesticides, volatile organic solvents, pyrethroid pesticides, and organochlorine (chlordane and lindane) pesticides. The three groups of pesticides acting at their major site of action can each initiate a control sequence that lead to increases in NMDA activity and consequent increases in nitric oxide (4-6, 14,16). The putative target for organic solvents, the vanilloid receptor (7), is also known to be able to produce increases in NMDA activity and nitric oxide (7). Thus, we see a common response to each of these four classes of chemicals as possibly being central to the action of these chemicals in MCS. How then, might this response lead to an understanding of chemical sensitivity?

Apparently through a striking convergence of this mechanism with that proposed earlier by Dr. Iris Bell (17-20). Bell proposed that MCS is centered on the process of neural sensitization, providing substantial support for this view. Her ideas were the focus of a New York Academy of Science meeting (Ann N Y Acad Sci, vol. 933). The major mechanism of neural sensitization is thought to be long term potentiation (LTP) a mechanism thought to be involved on a highly selective basis in strengthening of synaptic transmission in the central nervous system, during learning and memory. LTP is known to involve NMDA receptors in the postsynaptic cell and also nitric oxide which diffuses back to the presynaptic cell, acting as what is known as a retrograde messenger%% to increase release of glutamate neurotransmitter (5). Thus, immediately you can see a striking convergence of these two theories. Each class of chemicals can act to stimulate the neural sensitization process proposed to be central to MCS. In addition, it is possible %% A retrograde messenger is a compound which does just this--it diffuses from the post-synaptic neuron to the presynaptic neuron, causing the latter to release more neurotransmitter. In this way it can increase the activity of a synapse, thus producing LTP. Nitric oxide is not the only known retrograde messenger but it may be the most important one.

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to propose a vicious cycle mechanism (actually part of the larger such mechanism diagrammed in Fig.1) that involves both excessive nitric oxide through the retrograde

messenger role already discussed and peroxynitrite, through its ability to inhibit mitochondrial function and therefore ATP generation (5). It is known that when cells containing NMDA receptors become energy-deprived, those receptors become hypersensitive to stimulation (5). ATP-depletion in the glial cells may also have a role in increasing NMDA activity because of decreased transport of extracellular glutamate, that main NMDA agonist acting in the brain.

It can be seen from the above, how high level chemical exposure may be able to initiate a vicious cycle mechanism involving excessive NMDA activity, nitric oxide and peroxynitrite that would render areas of the brain hypersensitive to further chemical exposure. There are also three other well-documented mechanisms that may also have a role: Increased vanilloid activity due to oxidants (7); breakdown of the blood-brain-barrier (BBB) due to the action of peroxynitrite (5), thus allowing increased chemical access to the brain; and decreased chemical metabolism due to inhibition of cytochrome P450 activity by nitric oxide (5,6). The notion is that the total of six proposed mechanisms will act synergistically with each other to produce the exquisite sensitivity reported in MCS. Of these mechanisms, there is experimental support for a role of the NMDA receptors and of nitric oxide (4-6), for the breakdown of the BBB in both an animal model of MCS and in humans (6,13), and for excessive vanilloid activity in MCS (7). The overall mechanism is supported by at least 38 different types of observations, 24 documented in ref. 5, 12 more in ref. 7 and two additional ones in ref. 13.

There is often also, what may be described as peripheral sensitivity mechanisms involved in MCS, as emphasized by the work of William Meggs (21-25). Meggs has discussed the role of such peripheral sensitivity responses as reactive airways dysfunction syndrome or RADS, a form of asthma initiated by chemical exposure, reactive upper airways dysfunction syndrome or RUDS, chemical sensitivity in the upper respiratory tract, again initiated by chemical exposure and induced skin hypersensitivity.

Several of the mechanisms involved in peripheral sensitivity are likely to be similar to those involved in central sensitivity but others, notably BBB breakdown and possibly the role of nitric oxide acting as a retrograde messenger will not be involved in such peripheral sensitivity. Each of these sensitivity responses are likely to be local, with local inflammatory responses such as mast cell sensitization (26) and neurogenic inflammation<sup>25</sup> (25) having important roles in the sensitivity mechanisms.

This model of MCS based on a vicious cycle mechanism centered on excessive nitric oxide, peroxynitrite and NMDA activity provides explanations for each of the previously puzzling features of that illness (5,13,16): Its initiation by three classes of pesticides and by volatile organic solvents (5,7,13); its chronic nature (4-6); the generation of exquisite sensitivity to these same classes of chemicals (5,7,13); the reported change in porphyrin metabolism (6); the central and peripheral sensitivity mechanisms (5-7); and the masking/desensitization phenomenon in MCS (7). It also is consistent with the recent report by Kimata of some possible specific biomarkers for MCS (27), biomarkers consistent with the apparent vanilloid receptor role in MCS (7).

#### **Overall Perspective of the Elevated Nitric Oxide/Peroxynitrite Vicious Cycle Mechanism:**

<sup>25</sup> Neurogenic inflammation has been reported by Meggs and coworkers in peripheral sensitivity regions involved in MCS. It is an overt inflammatory response at the nerve endings involving several inflammatory messengers. It should be noted that the

peripheral sensitivity responses seen in MCS are overt inflammatory responses.

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It can be seen from the above discussion that the etiologic mechanism discussed here provides a detailed and relatively complete explanation of both MCS and of several other related illnesses, including CFS, FM, PTSD and Gulf War syndrome. It provides an explanation of how the various diverse stressors may initiate these illnesses, why they are chronic and how many of the diverse symptoms and signs of these illnesses may be generated.

The putative role of the nitric oxide/peroxynitrite vicious cycle mechanism with variable tissue distribution in these illnesses suggests that we can answer in the affirmative the question raised by Miller (28): "Are we on the threshold of a new theory of disease?"

#### **Therapy:**

The proposed mechanism of these illnesses suggests a number of approaches to therapy that will be discussed in more detail elsewhere (16). These include the use of various antioxidants acting as peroxynitrite scavengers, acting to lower NF- $\kappa$ B activity, acting a chain-breaking antioxidants to decrease oxidative chain reactions and acting as superoxide scavengers. They also include such agents as magnesium supplements, and the drugs dextromethorphan or memantine with both of these acting to lower NMDA activity. The vitamin B12 form hydroxocobalamin acts as a nitric oxide scavenger (8) and may be used either as an IM injection or as a nasal spray or as an inhalant to lower nitric oxide levels. Additional therapeutic approaches may be aimed at helping restoring mitochondrial function in the face of peroxynitrite or superoxide-mediated damage, through the use of L-carnitine (29) or of complex nutritional mixtures (30,31) or coenzyme Q10 supplements. These and other therapeutic approaches should be based, in addition, on attempts to minimize exposure of patients to anything that might otherwise exacerbate the basic biochemistry/physiology central to the putative etiology. Such exacerbation may be a consequence of chemical exposure in MCS, excitotoxin exposure in FM and possibly other illnesses and excessive exercise leading to "post-exertional malaise" in CFS, as well as exposure to food antigens in individuals suffering from food allergies.

Combinations of therapeutic approaches based on this mechanism may well be more effective in the treatment of these illnesses than have been treatments mainly aimed at the lessening of symptoms that have been used in the past.

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**Secret Hazards of Pesticides**  
Office of New York State Attorney General Eliot Spitzer

# The Secret Hazards of Pesticides:

## Inert Ingredients

Attorney General of New York  
New York State  
Office of the Attorney General  
Environmental Protection Bureau  
February 1996

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## **The Secret Hazards of Pesticides: Inert Ingredients**

Look at any label on a pesticide product and you will find a list of "active" ingredients, with a few long chemical names, and then typically the phrase "inert ingredients," with only a single percentage figure given and no listing of individual ingredients. The active ingredients are the chemicals used to control the target pest and must be listed on the label. The so-called "inert" ingredients are used as carriers for the active ingredients, to help dissolve them, make them easier to apply or to preserve them.

Unfortunately, many people will conclude from the term "inert" that such ingredients could not possibly have any adverse health or environmental effects. This is not the case at all. The chemicals used as inerts include some of the most dangerous substances known. Some of these chemicals are suspected carcinogens and have been linked to other long-term health problems like central nervous system disorders, liver and kidney damage and birth defects. They can also cause short-term health effects like eye and skin irritation, nausea, dizziness and respiratory difficulty. The U. S. Environmental Protection Agency (EPA), the agency responsible for regulating the use of pesticides, has categorized inert ingredients into four groups: substances known to cause long-term health damage and harm the environment, chemicals suspected of causing such health or environmental damage, chemicals of unknown toxicity, and those of minimal concern. Although EPA has published a list of chemicals used as inerts, this list does not tell consumers which products contain these inerts. Furthermore, pesticide manufacturers are not required to list all inerts on the product label. Thus, people must play blind man's bluff when it comes to which inerts might be in the pesticides they buy or are used where they live, work or play.

Inerts usually make up at least half if not most of consumer pesticide products. For instance, 99.1 percent of Raid's Ant and Roach Killer is inert ingredients and Ortho Diazinon Dust is 96 percent inerts. Of the 85 pesticide products examined by the Attorney General's office, 75 percent contained over 90 percent inert ingredients (see Table 1 on page 7 for a list of these products). Despite the health effects EPA associates with inerts, people do not know to which chemicals they may be exposed since inerts are not identified on the label. Health effects of some inert ingredients are

listed in Table 2 on page 9.

Pesticides are widely used throughout the United States in non-agricultural settings--in homes, outside homes, in offices, schools, and recreational areas. Over 70 million pounds of pesticides are applied on lawns alone every year. The use of lawn care pesticides is increasing at about 5 to 8 percent annually. In fact, four times as many pesticides are used on home lawns as are used to grow food crops. Commercial lawn care is now a \$1.5 billion industry. In addition, according to a 1985 study, pesticides used on golf courses accounted for nearly 12 million pounds nationwide. And all these pesticide products--whether used in lawn care, household fumigation, pet care or in personal-care products like insect repellents--contain substantial amounts of inert ingredients.

Who knows what the secret inert ingredients are? Obviously, the pesticide manufacturers and formulators know. Under the Federal Insecticide, Fungicide and Rodenticide Act (FIFRA) pesticide registrants (primarily manufacturers and formulators) must report the identity of inert ingredients to EPA. So one might assume that EPA knows the identity of the inert ingredients in every registered pesticide product. Unfortunately, that does not appear to be the case.

In 1987, EPA announced an "Inerts Strategy" designed to eliminate the most toxic inert ingredients from use, require improved label disclosure of inert ingredients, increase disclosure of inert ingredients, and increase the toxicity testing required for inerts. That strategy, if effectively implemented, could have enhanced the level of protection afforded to the public. In 1991, the EPA Inspector General reported on an investigation of EPA's implementation of its own "Inerts Strategy." The Inspector General reported that:

"EPA has not... enforced the 1987 Inert Strategy requirements for inerts with toxic effects... EPA identified 68 inerts as potentially toxic, and assigned them to a high priority for testing... EPA has no specific procedures or timetables for insuring that these inerts are reviewed."

"EPA is not sure how many chemicals registrants are using as inert ingredients because the inerts were not accurately coded into... [the EPA database]... there were about 600 registrations for which ... the chemical name was not available."<sup>(1)</sup>

How has EPA responded to this criticism of its implementation of the Inerts Strategy? Not very effectively. According

to a 1993 internal memo from the EPA Inspector General's office, corrective actions originally scheduled for completion in 1992 or 1993 had been delayed until 1995 or beyond. Attempts to develop a computer database for inert ingredients had failed, and further development of the system was contingent upon further funding.<sup>(2)</sup>

Thus, the EPA does not necessarily know the identity of the inert ingredients in the pesticide products sold to the general public, and that situation may not be resolved for years to come.

Even when EPA knows the identity of the inerts, FIFRA instructs it to keep that information secret if the manufacturer requests confidentiality *unless* the agency decides "that disclosure is necessary to protect against an unreasonable risk of injury to health or the environment".<sup>(3)</sup> Enacted almost half a century ago, the "trade secrecy" section of FIFRA was intended to protect manufacturers from any competitors who might copy the recipe for a successful product. Today, inert pesticide ingredients are still considered confidential under this obsolete regulation even though "trade secrets" are not necessarily secrets within the industry. Companies can now use commonly available "reverse engineering" techniques to find out the inert ingredients in their competitors' products. Now, this information is secret only to the public.

The Attorney General's office went directly to pesticide manufacturers to ask them to name the inert ingredients in some of their products sold in New York State. Many of the companies contacted refused to provide such information. Others agreed to identify inert ingredients only with an expectation of confidentiality. However, a few companies did cooperate without reservation. Thus, the survey shows that inerts information is generally not available to the public; most companies continue to withhold the identity of inert ingredients under a claim of confidentiality.

Ironically, many *non-pesticide* products containing the same chemicals used as inert ingredients in pesticides are governed by various laws, regulations, standards or guidelines because of their potential toxicity. There are limits for many of these chemicals in air, water and the workplace. There are restrictions on disposal of these chemicals, penalties for spills and special requirements for their transportation. These laws include the Toxic Substances Control Act, the Clean Water Act, the Clean Air

Act, the Resource Conservation and Recovery Act (RCRA), and regulations issued by EPA and the Occupational Safety and Health Administration, as well as guidelines from the National Institute for Occupational Safety and Health. (See Table 3 on page 11 for a selected list of regulations, laws, and advisories concerning chemicals used as

inerts.)

Even though some laws limit human exposure to these chemicals by restricting their release into air, water or the workplace, there is no way of knowing when the same chemicals are released as inert ingredients in pesticides. As long as pesticide ingredients are kept secret, people cannot even take steps to avoid exposure. And if an individual experiences a health problem in reaction to a pesticide, precious time can be lost while the doctor tries to obtain information concerning the chemicals to which the patient has been exposed.

The Federal Insecticide, Fungicide and Rodenticide Act should be amended to require pesticide manufacturers and formulators to disclose the total composition of pesticide products sold to the public. Product labels should identify each inert ingredient in the formulation. This information is simply too important to keep secret any longer because what the public does not know now about pesticides may very well hurt them some day.

**Credits**

This report was originally prepared in June 1991 by Michael H. Surgan, Ph.D., Chief Scientist and Assistant Attorneys General Deborah Volberg, Nancy Stearns and James A. Sevinsky, with assistance from other members of the Environmental Protection Bureau.

**Tables**

**Table 1: Percent Inerts in a Sampling of Pesticide Products<sup>(A)</sup>**

HOUSEHOLD PESTICIDE	MANUFACTURER	PERCENT INERT
Ant, Roach, and Spider Killer	Dexol Industries	99.5
Aphid and Mite Attack	Ringer Corp.	97.96

Crawling Insect Attack	Ringer Corp.	99.56
Flea Kill Fogger	The d-Con Co. Inc.	98.35
Hyponex Bug Spray	Hyponex Corp.	99.78
Insecticidal Soap for Indoor Plants	Safer, Inc.	98.0
Mite Killer	Safer, Inc.	98.0
No-Roach	Gaston Johnston Corp.	82.034
Ortho Flea-B-Gon	Chevron Chemical Co.	99.17
Ortho Hi-Power Ant, Roach & Spider Killer	Chevron Chemical Co.	95.11
Ortho Hornet & Wasp Killer	Chevron Chemical Co.	99.50
Raid Ant & Roach Killer	S.C.Johnson & Sons Inc.	99.10
Raid Flying Insect Killer	S.C.Johnson & Sons Inc.	99.2
Raid Fogger II	S.C.Johnson & Sons Inc.	85.0
Raid Fumigator	S.C.Johnson & Sons Inc.	87.4
Raid House and Garden Bug Killer	S.C.Johnson & Sons Inc.	97.504
Spectracide Garden, Rose & Household Plant Spray	Kenco Chem. & Mfg. Corp.	99.5
Spectracide Home Insect Control	Kenco Chem. & Mfg. Corp.	99.17
Spectracide Indoor Fogger	Kenco Chem. & Mfg. Corp.	99.40
Spectracide Wasp and Hornet Killer	Kenco Chem. & Mfg. Corp.	99.3664
Wasp and Hornet Attack	Ringer Corp.	99.56
LAWN CARE FUNGICIDE		
Lawn and Turf Fungicide	Faesy & Besthoff, Inc.	92.0
Lawn Disease Preventer	Glorion Corp.	95.0

Lawn Fungicide	Lebanon Chemical Corp.	99.945
<b>LAWN CARE HERBICIDE</b>		
2-Way Green Power	Lebanon Chemical Corp.	96.52
Balan 2, 5G	Elanco Products Co.	97.5
Expel Dandelion Killer	Lebanon Chemical Corp.	97.92
Longlife Weed and Feed	Frank's Nursery & Crafts	99.9845
Preen'n Green	Lebanon Chemical Co.	99.26
Spectracide Grass and Weed Killer	Kenco Chem. & Mfg. Corp.	99.7
Step 1 Crab Grass Prevention	O.M.Scott & Sons Co.	99.85
Step 2 Weed Control	O.M.Scott & Sons Co.	97.205
Super Turf Builder Plus 2	O.M.Scott & Sons Co.	97.66
Super Turf Builder Plus Halts	O.M.Scott & Sons Co.	98.97
Surety Weed and Feed Plus	Howard Johnson Ent. Inc.	99.063
Team 2G	Elanco Products Co.	98.0
XL 2G	Elanco Products Co.	98.9
<b>LAWN CARE INSECTICIDE</b>		
Bugout	Lebanon Chemical Corp.	98.86
Chinch Bug & Grub Preventer	Glorion Corp.	97.28
Deluxe Weed and Feed	Glorion Corp.	97.28
Grub Buster	Free Flow Fertilizer	98.5
Insect Control	O.M.Scott & Sons Co.	96.40
Lawn Insect Control	Glorion Corp.	98.86
Lawn Insect Control	O.M.Scott & Sons Co.	94.16

Lawn Insecticide	Free Flow Fertilizer	95
Lawn Insecticide	Greensweep Household Products	58.5
<b>LAWN CARE INSECTICIDE</b>		
Longlife Lawn & Garden Insecticide	MANUFACTURER Frank's Nursery & Crafts	PERCENT INERT 95.000
Oftanol	Glorion Corp.	98.5
Spectracide Lawn & Garden Insect	Kenco Chem. & Mfg. Corp.	95
	Control (granular)	
Spectracide Lawn & Garden	Kenco Chem. & Mfg. Corp.	18.7
	Insect Control (liquid)	
Step 3 Insect Control	O.M.Scott & Sons Co.	96.40
<b>GENERAL HERBICIDES</b>		
2 in 1 Crabgrass Preventer	Glorion Corp.	98.78
Aatrex 4L	CIBA-GEIGY Corp.	57.0
Arsenal	American Cyanamid Co.	72.4
Chopper	American Cyanamid Co.	72.4
Ortho Kleenup Super Edger	Chevron Chemical Co.	99.50
Prowl	American Cyanamid Co.	57.7
<b>GARDEN FUNGICIDE</b>		
Dexol Bordeaux Mixture	Dexol Industries	87.35
Garden Fungicide	Safer, Inc.	99.6
Pipron L.C.	Elanco Products Co.	17.6

Rubigan E.C.	Elanco Products Co.	87.5
GARDEN INSECTICIDE		
Liquid Sevin	Faesy & Besthoff, Inc.	77.5
Ortho 3-Way Rose & Flower Care	Chevron Chemical Co.	98.85
Rose & Flower Spray or Dust	Bonide Chemical Co. Inc.	84.5
Spectracide Rose & Garden Insect Killer	Kenco Chem. & Mfg. Corp.	99.88
OUTDOOR INSECTICIDE		
Abate 1-SG	American Cyanamid Co.	99
Amdro	American Cyanamid Co.	99.12
Cygon 400	American Cyanamid Co.	56.5
Gypsy Moth Biological Control	Acme Burgess Inc.	99.14
Mosquito Attack	Ringer Corp.	50
Ortho Diazinon Soil & Foliage Dust	Chevron Chemical Co.	96
Ortho Diazinon Plus Insect Spray	Chevron Chemical Co.	75
Ortho Home Orchard Spray	Chevron Chemical Co.	62.5
Ortho Isotox Insect Killer	Chevron Chemical Co.	90.6
Ortho Orthene Systemic Insect Control	Chevron Chemical Co.	90.6
Ortho Rose & Flower Insect Killer	Chevron Chemical Co.	99.70
Ortho Sevin	Chevron Chemical Co.	95
Raid Yard Guard	S.C.Johnson & Sons Inc.	99.125
Yard and Garden Insect Attack	Ringer Corp.	99.56
PET CARE		
Hartz 2 in 1 Flea and Tick	The Hartz Mountain Corp.	99.332

INSECT REPELLENT		
Cutter Insect Repellent	Miles Laboratory	67
Off	S.C. Johnson & Sons Inc.	85.00
Ortho Outdoor Insect Fogger	Chevron Chemical Co.	91.385
MOLLUSCICIDE		
Deadline	Pace National Corp.	96
Ortho Slug-geta	Chevron Chemical Co.	98

Table 2. Some Adverse Health Effects Of Certain Inert Pesticide Ingredients	
Chemical	Effects
Carbon tetrachloride*	Irritation of skin, eyes nose, throat; dizziness, vomiting, abdominal pain; diarrhea; damage to kidneys, liver; central nervous system depression; suspected carcinogen.
Chlorobenzene*	Eye and skin irritation, burns and inflammation; chest pain, slow heart rate, ECG irregularities; lung, liver and kidney damage; central nervous system depression; coma.
Chloroform*	Irritation to eyes and gastrointestinal tract; damage to liver and kidneys; central nervous system depression; nausea, dizziness, fatigue, respiratory distress; gonadal atrophy; fetal resorption; mutagen; coma and death by cardiac arrest; suspected carcinogen.
Chloroethane	Irritation of eyes; abdominal cramps, nausea, vomiting; liver and kidney damage; nervous system dysfunction; blood cell disorders; suspected carcinogen.
Cresols	Skin irritation, burns, and inflammation; irritation of eye, permanent damage and blindness; pneumonia; pancreatitis; central nervous system disorders; kidney

	failure.
Dibutylphthalate	Irritation of eyes and throat; photophobia, conjunctivitis, nausea, dizziness.
Diethylhexylphthalate*	Eye, nose and throat irritation; liver damage; testicular damage; central nervous system depression; suspected carcinogen.
Dimethylphthalate	Irritation of eyes, mouth, nose, throat; dizziness, abdominal pain, nausea, vomiting, diarrhea; central nervous system depression; reduced respiratory rate; paralysis, coma.
Epichlorohydrin*	Skin and eye irritation, conjunctivitis, corneal clouding; nausea, vomiting, fatigue; liver and kidney damage; inflammation of lungs, chronic bronchitis; death by respiratory paralysis; mutagen; fetotoxic.
Ethylbenzene	Irritation of eyes, nose and throat; skin irritation, inflammation, blisters and burns; liver and kidney damage; central nervous system disorders; headache, sleepiness, difficulty in breathing; unconsciousness and coma.
Ethylene dichloride*	Nausea, vomiting, diarrhea; damage to liver and kidneys; central nervous system depression; death due to circulatory and respiratory failure.
Isophorone	Irritation of skin, nose, throat, respiratory system; lung congestion and degeneration; central nervous system disorders; kidney and liver damage; suspected carcinogen.
Methyl bromide*	Eye and skin irritation; blurred vision, headache, dizziness, nausea, abdominal cramps; anorexia; bronchopneumonia, pulmonary edema; brain damage, convulsions, coma; kidney and respiratory failure.
o-Dichlorobenzene	Eye irritation and cataracts; skin irritation and lesions; headache, nausea, vomiting, drowsiness; respiratory depression; anemia, kidney and liver damage; chromosomal breaks.
p-Dichlorobenzene	Irritation of skin, eyes, respiratory system; headache, dizziness, hyperactivity, weakness, weight loss; liver and blood disorders; kidney damage; lung congestion, difficulty in breathing; mutagen.

Phenol	Irritation of eyes, nose, throat; headache, dizziness, fainting, abdominal pain, nausea, vomiting, diarrhea; damage to liver, kidney and heart; chromosomal aberrations and damage; mutagen.
Propylene dichloride*	Eye and skin irritation; dizziness, disorientation, nausea, vomiting; liver and kidney damage; central nervous system damage; coma; hemolytic anemia; suspected carcinogen.
1,1,2-Trichloroethane*	Gastrointestinal inflammation and congestion; liver and kidney damage; immune function disorder; central nervous system depression; suspected carcinogen.
Toluene	Skin, eye and respiratory irritation; abdominal pain, headache, nausea, dizziness, drowsiness, hallucinations; anemia; liver disorders and enlargement; central nervous system dysfunction; coma and death.
Trichloroethylene*	Eye irritation, visual distortion; abdominal pain, nausea, diarrhea; anorexia; liver and kidney damage; peripheral nerve damage, numbness and paralysis; blood disorders; cardiac arrhythmia; suspected carcinogen.

\*This chemical was identified as an Inert Ingredient by EPA in 1991, but is absent from the most current list of Inert Ingredients released in May, 1995.

Sources:

- U. S. Environmental Protection Agency, *Chemical Profiles, Interim Guidance, Chemical Emergency Preparedness Program, 1985*
- U. S. Environmental Protection Agency, Office of Health and Environmental Assessment, *Health Assessment Documents*
- U. S. Public Health Service, Agency for Toxic Substances and Disease Registry, *Toxicological Profiles*
- New York State Department of Health, *Chemical Fact Sheets*





**2. Maximum Contaminant Level Goals**

Establishes non-enforceable Maximum Contaminant Level Goals (MCLG) which are set at a level at which no known or anticipated adverse health effects occur and which provide an adequate margin of safety.

**3. Monitoring Requirements**

Requires monitoring for contaminants likely to be found in the system's drinking water, including contaminants not regulated under National Primary Drinking Water Regulations. Results must be reported to both the State and EPA and made available to the public.

**4. 1986 Amendments/Statutory Contaminants**

Requires EPA to regulate 83 contaminants by publishing MCLG's and promulgating National Primary Drinking Water Regulations for each of the 83 listed contaminants.

**5. 1986 Amendments/Drinking Water Priority List**

Requires EPA to establish a priority list of contaminants which may have adverse health effects and are known or anticipated to occur in public water systems.

**TOXIC SUBSTANCE CONTROL ACT (TSCA)**

**6. Section 4(a), Final Test Rules**

Requires EPA to test substances which meet certain criteria, such as those which may present an unreasonable risk or injury to health or environment, in order to develop health or environmental data.

**7. Section 8(d), Health and Safety Data Rule**

Requires manufacturers, importers and processors of listed substances to submit to EPA copies and lists of

unpublished health and safety studies on the listed substances with which they deal.

**8. Section 4(a), Dioxins/Furans Rule**

Requires manufacturers and importers of certain organic chemicals to test for the presence of halogenated dibenzodioxins (HDD) and halogenated dibenzofurans (HDF) as contaminants. Results and existing test data must be submitted as well as additional information if HDD and/or HDF concentrations exceed designated levels.

**9. Section 12(b), Export Regulations**

Requires exporters of chemical substances to notify EPA of such exportation if any exported substances are affected by TSCA Sections 4, 5, 6 or 7.

**COMPREHENSIVE ENVIRONMENTAL RESPONSE, COMPENSATION AND LIABILITY ACT (CERCLA)/SUPERFUND AMENDMENTS AND REAUTHORIZATION ACT (SARA)**

**10. CERCLA. Hazardous Substances**

Establishes a list of substances which must be reported to the National Response Center when released in quantities exceeding a specified reportable quantity.

**11. SARA. Title III Section 302 and 304, Extremely Hazardous Substances**

Requires facilities handling substances named on the list of extremely hazardous substances to notify the State of the presence of these substances in excess of their Threshold Planning Quantities and must notify local authorities of their release in excess of their Reportable Quantities.

**12. SARA. Title III Section 313, Toxic Chemicals**

Establishes a list of toxic chemicals. Manufacturers, processors and users of these chemicals must submit release reporting forms.

**13. CERCLA Section 104(i), Priority list of CERCLA Hazardous Substances**

Requires EPA and the Agency of Toxic Substance and Disease Registry to 1) prepare a prioritized list of hazardous substances commonly found at National Priorities List sites which pose the greatest potential health risk, 2) to develop Toxicological Profiles of these substances, 3) establish a research program to fill data gaps.

**RESOURCE CONSERVATION AND RECOVERY ACT (RCRA)**

**14.** Requires notification of EPA by anyone who generates, transports, treats, stores or disposes of wastes specified under Section 3001 of RCRA.

**15. Hazardous Constituents for Groundwater Monitoring**

Requires groundwater monitoring at RCRA land-based hazardous waste disposal units for all constituents listed in Appendix IX to 40 Code of Federal Regulations (CFR) 264.

**16. Land Disposal Prohibitions - Halogenated Organic Compounds**

Restricts land disposal of waste containing halogenated organic compounds above specified concentrations.

**17. Land Disposal Prohibitions**

Lists the hazardous wastes identified in 40 Code of Federal Regulations 261 which were scheduled for restricted/prohibited land disposal after enactment of the Hazardous and Solid Waste Amendments to RCRA in 1984.

**CLEAN WATER ACT (CWA)**

**18. Section 304, Water Quality Criteria; Section 307, Priority Pollutants**

Requires EPA to publish and periodically update ambient water quality criteria. Criteria are to reflect latest scientific knowledge on the identifiable effects of substances on public health and welfare, including but not limited to aquatic life, aesthetics and recreation. Establishes a list of toxic pollutants for which EPA is required to publish ambient water quality criteria. Under Section 304, these chemicals shall be subject to effluent limits resulting from the application of best available technology.

**19. Section 311, Hazardous Chemicals**

Requires EPA to publish a list of substances that are considered hazardous if spilled in navigable waters.

**OCCUPATIONAL SAFETY AND HEALTH ADMINISTRATION (OSHA)**

**20. Air Contaminants (29 CFR 1910.1000)**

Establishes a list of air contaminants and sets exposure limits for the workplace.

**NATIONAL TOXICOLOGY PROGRAM**

**21. Report on Carcinogens**

Identifies carcinogens and subdivides them into Known and Anticipated. Categorizes 162 substances on this basis.

**AMERICAN CONFERENCE OF GOVERNMENTAL AND INDUSTRIAL HYGIENISTS**

**22. Threshold Limit Value Chemicals**

Provides various workplace exposure limits (time-weighted average, short-term exposure limits, ceiling limits) for each covered substance.

**INTERNATIONAL AGENCY FOR RESEARCH ON CANCER**

**23. Human Carcinogens (Groups 1, 2A, and 2B)**

Identifies carcinogens and classifies them as: Group 1 (sufficient evidence of human carcinogenicity); Group 2A (probable human carcinogen); Group 2B (possible human carcinogen).

**DEPARTMENT OF TRANSPORTATION**

**24. Hazardous Materials**

Regulates interstate commerce of hazardous materials, including all CERCLA hazardous substances. Specifies requirements for description, shipping names, class, labeling and packaging, as well as spill notification.

**CLEAN AIR ACT**

**25. Section 111, Potential Human Health Hazards**

Lists substances published by EPA pursuant to Section 111 of the Clean Air Act, which pose a potential health hazard and for which specific control techniques are defined.

**NATIONAL INSTITUTE OF OCCUPATIONAL HEALTH AND SAFETY (NIOSH)**

**26. Criteria Documents**

Specifies a NIOSH Recommended Exposure Limit and appropriate preventive measures to reduce or eliminate adverse health effects.

1. "Inert Ingredients in Pesticides," USEPA, Office of the Inspector General, Audit Report E1EPF1-05-0117-1100378, Sept. 27, 1991.
2. Memo from Michael Simmons, Associate Assistant Inspector General for Internal and Performance Audits to Victor J. Kimm, Acting Assistant Administrator for Prevention, Pesticides and Toxic Substances, September 17, 1993. See Foreword for an update on the computer database.
3. FIFRA, Section 10(d)(1)(C), entitled, "Protection of Trade Secrets and Other Information".
4. Based on a market survey conducted during the spring and summer of 1990.

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