MOSQUITO PESTICIDE SPRAYING

Another "Downwinder" Threat to Utahns

"New research suggests that the use of airplanes to spray anti-mosquito pesticides may increase the risk of autism spectrum disorder and developmental delays among children."

American Academy of Pediatrics, conference presentation 2016[1]

The Salt Lake City Mosquito Abatement District (SLCMAD)'s Environmental Assessment (EA) states, "[SLCMAD] was created in 1923 to protect Salt Lake City residents from the large numbers of these pestiferous mosquitoes." The EA seems to be using a mandate that is nearly 100 years old as part of their justification for their spraying strategy. Obviously much has changed in the last 100 years. This mandate, and the mere continued presence of mosquitoes in their natural habitat in the Northwest Quadrant is not enough to justify pesticide spraying.

For just about every human on earth pesticides now contaminate our air, drinking water, food and soil. They are detected on Mt Everest,[2] in the deepest parts of the ocean,[3] and in rainfall from the sky.[4] While contamination is global, for Salt Lake Valley residents, the most important place to reduce chemical usage in the Valley itself.

Utah Physicians for a Healthy Environment (UPHE) have had serious concerns about what might be done to control the mosquito population in the area of the inland port ever since the port first emerged from the legislature in 2018. Our concerns have grown significantly after learning that SLCMAD has been conducting massive aerial spraying of pesticides for many years over approximately 160,000 acres in the Northwest Quadrant of Salt Lake City within a few miles of

population centers on the West side, North Salt Lake, and West Bountiful. SLCMAD proposes to use known neurotoxins: permethrin, the synergist piperonyl butoxide (PBO), and the organophosphate pesticide (OP), naled.

In recent years numerous medical experts and entire medical societies have made strong position statements regarding the danger to humans of even small doses of chemicals, and their link to obesity, cancer, heart disease, birth defects, reproductive pathology, and neurologic and brain disorders such as Parkinson's, impaired intellect, autism and attention deficit disorder. The American Academy of Pediatrics, American College of Obstetricians and Gynecologists, American Society for Reproductive Medicine, International Federation of Gynecology and Obstetrics (FIGO), World Health Organization, and the Endocrine Society are all mainstream medical organizations that have, in one form or another, called for a sharp reduction in human exposure to chemicals broadly, and pesticides in particular.

A recent review by 12 of the most knowledgeable researchers on endocrine disrupting chemicals (EDCs), (chemicals that mimic or inhibit key human hormones at very small doses which includes most pesticides) concluded that, "Whether low doses of EDCs influence certain human disorders is no longer conjecture, because epidemiological studies show that environmental exposures to EDCs are associated with human diseases and disabilities," and that, "For every chemical that we looked at that we could find a low-dose cutoff, if it had been studied at low doses it had an effect at low doses"[5]

Toxicologist Linda Birnbaum, the director of the U.S. National Institute of Environmental Health Sciences (NIEHS), stated that "existing US regulations have not kept pace with scientific advances showing that widely used chemicals cause serious health problems at levels previously assumed to be safe."[6] This speaks to SLCMAD repeatedly invoking EPA approval of the pesticides it is using for spraying.

At the top of the list of chemicals of concern are pesticides (insecticides and herbicides) because they function as biological poisons to all living cells, from pest insects to humans and everything in between. Prominent researchers and medical societies have for years advocated a complete re-evaluation of the rationale for, and justification of, their use. It is increasingly clear that much of society's use of pesticides is cavalier, poorly reasoned, often counterproductive to the intended goal, and represents poor priorities by causing unacceptable public health risk for dubious benefit. That is particularly true of using pesticide spraying for mosquito control.

Executive Summary

1. Pesticides in general, including those used by SLCMAD, are a widespread risk to human health even at low doses, especially for fetuses and infants.

2. Decisions on the risks vs. benefits of exposing the public to dangerous chemicals should not be made by people with no expertise in public health, toxicology, or environmental toxins.

3. The VOCs from pesticide spraying is a significant contributor to local air pollution.

4. Pesticide spraying has created a chemical arms race, is not effective in reducing mosquito populations long term, is losing its effectiveness even in the short term, and can even be counterproductive.

5. We must not allow a cure worse than the disease. The incidence of severe outcomes from West Nile Virus is so low that preventing those outcomes should not be allowed to eclipse the long list of other health and environmental concerns from pesticide use.

6. Spraying does not re duce the incidence of WNV.

7. Claims of safety for pesticide spraying use faulty logic and outdated, faulty science. It makes no sense to expose hundreds of thousands of people to neurotoxic chemicals in an attempt to prevent a neurotoxic disease in a few dozen people.

8. Pesticide spraying has adverse impacts on beneficial insects, bird populations, wildlife, the ecosystem of the Great Salt Lake and beyond.

9. There are better ways to control mosquitoes.

10. SLCMAD does not need or deserve to raise your taxes to increase the spreading of neurotoxic chemicals.

Conclusion

The scientific and empirical evidence is overwhelming that spraying adulticides to kill mosquitoes, especially aerial spraying, is ineffective, and can be even counterproductive, over the long term, and even the short term, to both goals of controlling mosquito populations and preventing West Nile Virus. Furthermore, the medical literature strongly indicates that routine aerial spraying over Salt Lake City's airshed represents a broad-based danger to public health. Utah Physicians for a Healthy Environment (UPHE) implore SLCMAD to end all of their insecticide spraying for mosquitoes, whether from back packs, trucks, drones, or airplanes. This practice is an institutionalized relic of the 1950s and should be stopped immediately.

1. Pesticides Represent a Widespread Risk to Human Health

Almost regardless of where they are applied, through drift and volatilization, pesticides end up causing ubiquitous, global, environmental contamination. The logical extension is now well documented; pesticides are universally found in the blood and urine[7] of almost all human beings everywhere, in amniotic fluid, new-born babies, in mother's breast milk, [8] and in domestic drinking water worldwide. In studies dating back ten years, glyphosate, and 2,4-D, two of the most widely used herbicides globally, have been found in rain water and in air samples from the Midwest. "It is out there in significant levels. It is out there consistently," said Paul Capel, environmental chemist and head of the agricultural chemicals team at the U.S. Geological Survey Office, part of the U.S. Department of Interior.[9] Glyphosate was found in every stream sample examined in Mississippi in a two-year period and in most air samples taken." This speaks to the pervasive spread of pesticides throughout our environment. That the SLCMAD would contribute to this global problem would be difficult to justify unless there was a clear benefit. We believe there is no benefit to offset this concern.

In the early 1990s growing awareness of the toxicity of pesticides led to a National Academy of Sciences Report, *Pesticides in the Diets of Infants and Children* (NAS, 1993).[10] The report emphasized that children's pesticide burdens differ from adults quantitatively and qualitatively and questioned the validity of risk tolerances used by the EPA to declare "safety." (More about that in later sections). The report estimated that 50% of lifetime pesticide exposure occurs during the first 5 years of life. By the age of 6 the brain reaches 90% of its eventual adult volume and that corresponds to about 90% of its overall, permanent organizational and architectural structure,[11] obviously the most critical developmental window for organogenesis, especially for the brain.

The NAS report raised the profile of concerns about children being a uniquely susceptible subgroup to organophosphate pesticides (OP). The new concerns highlighted the vast physiologic differences between children and adults that go far beyond differences in size. Children at the pre-natal and early infant stages of development are much more chemically sensitive for multiple reasons, one of which is that they have a decreased ability to metabolize chemical toxins.[12,13,14] For example, children do not have the necessary enzymes to detoxify pesticides like OPs or permethrins.[15] In eight day old rats permethrin is nearly five times more acutely toxic than in adults because they lack permethrin-specific esterases.[16] In humans the blood brain barrier (BBB) doesn't mature for at least six months after birth.[17] That allows more of any toxic chemical to reach brain tissue.

Children consume far more food, calories, and water, inhale far more air, and have a greater surface area per unit of weight than do adults, significantly increasing their exposure. The same amount of exposure yields higher blood and brain levels in an infant for numerous reasons. It is no surprise that children 6-11 yrs. of age had levels of organophosphate metabolites twice as high in their urine as adults 20-59 yrs of age.[18] At the same time, organ growth is obviously rapid during the early stages of fetal and infant development, especially for the brain, yet the BBB is not completely formed allowing higher concentrations of toxic, exogenous chemicals to accumulate in the brain

at the worst possible time, with permanent consequences.[19] Pesticides can cross the placenta,[20] contaminate the intrauterine environment, including the embryonic and fetal brain, and impair development. This is far more than a theoretical concern. The most widely used insecticides work by attacking the nerve cells of insects. A nerve cell in a mosquito is almost identical to a nerve cell in a fetus. Furthermore, a nerve cell in a fetus can be just as critical to the fetus as it is to a mosquito and just as vulnerable to chemical attack. They can both be damaged by the same insecticide. Prominent researchers put it this way. "The molecular targets of pesticides are often shared between pest and non-target species, including humans. This is particularly true for the neurotoxic organochlorine, organophosphate, and pyrethroid pesticides."[21]

Last year researchers at NYU concluded that 81% of the cognitive loss in children from environmental neurotoxins came from exposures to polybrominated diphenyl ethers (PDBEs) and organophosphate pesticides, far eclipsing that caused by heavy metals like lead and mercury.[40]

Maternal exposure continues the threat to an infant in the postnatal period. Characteristics such as lack of ionization, small molecular weight, low volume of distribution, low maternal serum protein binding, and high lipid solubility all contribute to the transport of these chemicals into human breast milk. Human breast milk in the Salt Lake Valley no doubt already has contaminants, but aerial spraying by SLCMAD will add to that burden. A study of contaminants in human breast milk from multiple countries found every milk sample from every country was contaminated with pyrethroid pesticides,[22] and at levels high enough to cause neurologic and endocrine effects.[23] Other studies show contamination even in households where no pesticide is used.[24] Organophosphate pesticides have also been found routinely in breast milk.[25]

On the other end of the spectrum of health consequences, children have more future potential years of life. This allows more time in which to develop chronic diseases like cancer and heart disease known to be triggered by early exposure to environmental toxicants.

Researchers looking for reasons behind disturbing epidemiologic studies, have found evidence of auxiliary mechanisms of toxicity beyond the standard route recognized for lethality in insects.[26, 27, 28]

New research shows a link between OP exposure among children growing up in an agricultural area and loss of lung function.[301]

SLCMAD's environmental assessment (EA) of its pesticide use lists only six studies to support their statement that "several studies on this procedure have not found any negative impact on human health." [29,30,31,32,33,34] We assume this means these studies are the strongest evidence SLCMAD has to defend the practice. Three of the studies only looked at temporally related cases of hospital and outpatient visits for acute asthma and skin rashes. One study only sought to estimate dermal absorption of permethrin, and two studies used modeled exposure estimates combined with EPA toxicology risk assessments to declare no health impact. As a group these few studies simply

do not address the real health consequences of pesticide spraying and are almost irrelevant to the issue. Asthma attacks is not one of the most important disease outcomes from pesticide exposure and cannot be considered a surrogate or marker for other more serious outcomes. SLCMAD's director keeps using these studies to claim there is no health effects from the pesticides they use. But these studies do not claim that, in fact, have virtually nothing to do with the issue.

The first draft of this document was completed before SLCMAD abandoned the proposal to involve the US Air Force. SLCMAD's director replied to that draft with a rebuttal document listing a total of 12 references, but even those do not address the health consequences in any meaningful way either. The failure of traditional risk assessments (TRAs) to reflect human health hazards will be address later and in the Appendix.

Below we address the medical research on specific components of the spraying mix.

Pyrethroids

The primary and most consistent human effect of insecticide exposure is neurotoxicity. That is hardly a surprise given their origin as nerve agent chemical weapons. For over 40 years pyrethroid compounds have been the most commonly used insecticides for controlling adult mosquitos. "The main metabolites of pyrethroids have frequently been detected in urine samples from the general population, confirming widespread exposure of children and adults to one or more pyrethroids."[35,36]

For SLCMAD to repeatedly claim that exposure levels are so low as to avoid clinical consequence is contrary to robust research, overlooking multiple, important lines of evidence. Humans lack a critical enzyme, serum carboxylesterases, a primary avenue of pyrethroid detoxication through hydrolysis.[37] To that point, physiological differences and diffusion modeling shows that exposure to the pyrethroid deltamethrin was predicted to result in a two-fold greater peak brain concentration in humans compared to rats.[38]

The research showing the toxicity of pyrethroids to human health is direct and extensive. Pesticides in general, and pyrethroids in particular, are toxic to the brain, associated with a wide range of neurologic and brain diseases, especially impaired brain development, and loss of intellect and behavioral disorders in children. The damage to the nervous system from pyrethroids is comparable to that from the banned legacy pesticide, DDT.[39] Recently the mode of action of pyrethroids has been found to be similar to chlorinated pesticides which were banned in the 1980s because of their harm to human health and the environment. Implicating the risk of pyrethroids to fetuses at extremely low concentrations are studies showing toxicity to small invertebrates at concentrations of as little as two parts per trillion.[41] In 1999, after a heavy pyrethroid spraying campaign on Long Island in the wake of Hurricane Floyd, 10 million lobsters, 90% of the population, died off. The lobstermen sued the pesticide manufacturers for $125 million. This kind of devastation of marine life is a chilling warning about the destructive potential to humans, especially at the fetal stage.

Adverse outcomes are found among individuals whose exposure is comparable to what the general population experiences, and not limited to only highly or occupationally exposed individuals.

Experimentally, pyrethroid exposure in lab animals during development has a broad range of toxic effects on neurotransmitter systems, the BBB, and neurobehavior.[42] Experimental studies showed pyrethroids cause dopaminergic related neurodegeneration and alter mitochondrial function in vitro and in vivo.[43] Other studies showed pyrethroids cause neuroinflammation and damage to glial cell function, critical to the immune system that protects the brain.

The neurotransmitter acetylcholine, central to memory, learning, and attention span, is lower in parts of the brain in children with autism.[273] Interfering with acetylcholine is the primary mechanism of these insecticides. An in vitro study of cortical neurons found that pesticide exposure caused specific transcriptional changes similar to changes characteristic of the autistic human brain.[274] Low level pyrethroid exposure affects learning, motor activity and sexual behavior in lab animals.[47]

Consistent with this research, several epidemiologic studies have found an increased risk of autism with more pyrethroid exposure.[86,275,276] The clinical findings of specific studies are worth highlighting. Children with autism and developmental delay were more likely than a control cohort to have had agricultural--pyrethroids, OPs, or other pesticides--applied near their mothers' place of residence, especially during the third trimester.[86] In a swampy region in central New York, an observational study showed that children living in ZIP codes in which aerial pesticide spraying was conducted every summer since 2003, were 37% more likely to be diagnosed with autism or a developmental delay compared to those in ZIP codes where pesticides were used with other methods of distribution, such as manually spreading granules or using hoses or controlled droplet applicators.[44] This is particularly alarming because of the similarities with the circumstances in the Northwest Quadrant of SLC. More about this study later.

Pyrethroid pesticide exposure is inversely associated with performance on intelligence tests in six year olds.[45] Levels of pyrethroid breakdown products in the urine were proportionally associated with a higher level of behavioral problems in children.[46] Attention deficit disorder in children is also associated with pyrethroid exposure.[48,49] A study that found higher rates of autism with intellectual disability among children who lived within 2,000 meters of agricultural field that used pesticides, and the risk increased further if the children were also exposed as infants.[50] Among 11 pesticides examined in this study, the one with the highest risk was permethrin, which was even greater than chlorpyrifos, the one organophosphate that the EPA recently banned for almost uses and is widely banned in other countries. Pyrethroids are associated with increased risk of brain cancer.[51]

Exposure to common pyrethroids speeds the onset of puberty in boys.[52] A critically important study was just published in Jan. 2020 that showed a 56% increased rate of overall mortality among adults exposed to higher levels of pyrethroids over a period of 14 years.[53]

Most pyrethroid compounds are endocrine disruptor chemicals (EDCs) and as such can interfere with human reproduction and act as carcinogens.[54,55] One of the defining characteristics of EDCs is that they can interfere with normal organ development at very low doses, particularly at vulnerable stages, i.e. pre-and perinatal development. In 2009 The Endocrine Society, the medical organization with expertise most relevant to developmental chemical toxicity, publicly stated with regard to EDCs, "Even infinitesimally low levels of exposure indeed, any level of exposure at all,

may cause endocrine or reproductive abnormalities, particularly if exposure occurs during a critical developmental window. Surprisingly, low doses may even exert more potent effects than higher doses."[56]

Another characteristic of EDCs is that the adverse human health effects may not be apparent for many years after exposure and therefore cannot be assessed using traditional dose response models that are the key to toxicology risk assessments used for determining safe thresholds. Making matters worse, pyrethroid metabolites have greater endocrine disrupting activity than their parent compounds. Indeed, it is through endocrine disruption that many of the now documented adverse health outcomes occur from low dose exposure typical of what much of the human population is now exposed to. To quote the authors of one review paper, "their [pyrethrins'] structural resemblance to TH [thyroid hormone]s, with the fact that in vitro and in vivo animal studies demonstrate clear interference with TH homeostasis and action argues for more caution in their use and more intense scrutiny of their long-term effects."[57]

Organophosphates (OPs)

Given that SLCMAD is using naled, an organophosphate (OP), repeatedly via airplanes, it is important to examine the medical literature on the toxicity of this group of pesticides. OPs are progeny of nerve gas agents originally used in WWI that were debilitating or lethal by causing restlessness, seizures and respiratory arrest. OP are acetylcholinesterase (AChE) inhibitors, killing insects by the same biochemical process that sarin gas kills humans, provoking a marked acceleration of nerve impulses causes a buildup of acetylcholine, resulting in paralysis of the insect. AChE is essential in all mammals, including humans. OPs were adapted from chemical weapons to be lethal to insects at low doses via the same mechanism. Naled has the same method of action common to other OPs, including the recently banned chlorpyrifos. Acute toxicity in humans follows the same pattern as it does in insects, but the human effects of greatest concern are more related to chronic exposure to adults, and acute, low dose exposure to fetuses and infants.

Over 30 years ago, the Office of Technology Assessment (OTA) of the US Congress released an extensive report entitled "Neurotoxicity: Identifying and Controlling Poisons of the Nervous System." One of the two primary targets of the report was chemical pesticides. They stated, "Of particular concern are the delayed effects of some of the organophosphate pesticides."[58]

Naled leaves a breakdown product, dichlorvos, which is also an insecticide with similar acute and chronic effects as the parent compound which serves to prolong the toxicity. In fact, dichlorvos, is classified by the EPA as a group C (possible) human carcinogen, while naled itself is not. Dichlorvos exposure during pregnancy or childhood has been linked to an elevated incidence of brain tumors and leukemia.[59,60]

Naled is far more toxic by inhalation exposure than by ingestion, maybe as much as 20 times more toxic,[278] and the EPA has outlined numerous restrictions for potentially exposed applicators. However, the EPA has not calculated the potential for "bystander" exposure despite stating that there are no risks to bystanders, an inexplicable, critical omission. Another study found that small droplets of naled (the size produced by the ultra-low volume sprayers that SLCMAD boasts about using) were about four times more acutely toxic than larger droplets.[279]

There are numerous studies in animals showing that naled at low dose exposures causes a wide variety of adverse health outcomes, including diseases of the nervous, circulatory, reproductive, and immune systems.

OPs as a group, like pyrethroids, are endocrine disruptors. And like with pyrethroids, one of the mechanisms of endocrine disruption and therefore impact on brain development by OPs is inhibition of thyroid hormone production.[61] Other clinical outcomes associated with pre-natal OP pesticide exposure include abnormal primitive reflexes in newborns; mental and motor delays among preschoolers; and decreases in working and visual memory, processing speed, verbal comprehension, perceptual reasoning, and IQ among elementary school–age children. Prenatal exposures are also associated with elevated risks for symptoms or diagnoses of attention-deficit/hyperactivity disorder (ADHD) and autism spectrum disorder (ASD).[62]

Other systematic reviews and multiple epidemiologic studies have linked OP exposures during fetal development with poorer cognitive, behavioral, and social development in children.[63,64,65,66,67] In one review, adverse effects of OP pesticide exposure on neurodevelopment were seen in all but one of the 27 studies evaluated; the strongest associations occurred following prenatal exposures,[68] but it has also been found with post-natal exposure as well.[68] Another study showed that young mammals, including humans, may be at risk of impaired neurological development from organophosphate pesticides, even at low, commonly encountered environmental levels.[69]

More specific studies are worth mentioning. A meta-analysis by researchers at University College London found chronic, low-level exposure to organophosphate pesticides causes permanent damage to cognition, including information processing and working memory.[70] Urinary levels of organophosphate metabolites in pregnant mothers were measured, then the children they gave birth to were tested at age 7. Children from those mothers who were in the highest 20% of exposure, showed an average IQ deficit of a stunning 7 points.[71] The relationship between biomarkers of organophosphate exposure in pregnant mothers and neurologic tests at one year, two years, and 6-9 years, showed that more prenatal exposure caused a loss of perceptual reasoning as early as one year of age, and continued through childhood.[72] Prenatal exposure to organophosphates was associated with delayed mental milestones in 2 year olds.[73] In another study prenatal exposure to organophosphates was associated with decreased non-verbal IQ measured at age 6.[74] The US Congress produced a report back in 1990 that showed an organophosphate pesticide, in this case malathion, can damage the nervous system after just one exposure.[75]

A higher likelihood of an ASD diagnosis was observed for children born to women residing within 1.5 km of OP pesticide applications on agricultural fields.[76] Living in a residence close to an agricultural site with OP use during fetal development was associated with a reduction in children's IQs measured at age 7.[77] Risks for impaired neurodevelopment increased in children of farmworkers who experience higher exposures to OP.[78] Higher OP pesticide metabolite levels in the urine of pregnant mothers were associated with ASD traits in adolescence in those children.[79]

Some children are genetically more susceptible to organophosphates.[80] Children with genetic disadvantages that reduce capacity to detoxify OP pesticides have higher rates of neurodevelopmental disorders.[63] Prenatal exposure to one of the most potent OPs, chlorpyrifos, has also been shown to have an association with decreases in brain volume in the areas responsible for attention, receptive language processing, social cognition, and regulation of inhibition.[81]

OP pesticides can interfere with brain development at levels previously thought to be safe or inconsequential, and through mechanisms other than the lethal mode of action for insects.[76,82,83,84] This is an indication of an additional, as yet undetermined mechanism of toxicity.[85,86] Consistent with human studies, experimental animal studies also confirm the toxicity of early-life OP pesticides on neurodevelopment, resulting in impaired motor activity, behavior, and cognition, even at doses below the known mechanism by which they kill insects.

Within a population of 25.5 million children 0 to 5 years of age, researchers calculate a total loss of 16.9 million IQ points due to common background exposure to just organophosphates. This estimation does not take into account all the other known environmental neurotoxins like heavy metals, PCB, flame retardants, many other neurotoxic chemicals, including pesticides, which only add to that total.[87]

In Japan, between 1957 and 1971, school children saw a huge increase in impaired vision eventually tied to the use of an OP in agriculture. One town near an agricultural area now has an eye disease named after them, "Saku disease." In that town 98% of the children had visual acuity problems linked to the regular application of an OP on near-by agricultural fields.[88] In California a boy became blind after being outside while a helicopter was spraying an OP.[89] An ingredient in naled, trichlorfon, was found in a study to cause severely reduced brain weight in test animals exposed.[90]

In keeping with their potential to mimic or interfere with human hormones, exposure to OPs is a risk factor for acquiring hormone-related cancers, i.e. including breast, thyroid, ovary and lymphoma.[91] Recall that dichlorvos is classified as a group C carcinogen by the EPA. Exposure during pregnancy or childhood is linked to an increased incidence of brain tumors and leukemia.[60,92] Researchers found an association between exposure to dichlorvos saturated "no-pest" strips during pregnancy and childhood and the incidence of three types of childhood cancer: leukemias, brain tumors, and lymphoma.[282] A Missouri Department of Health study found similar results for childhood brain cancer.[283]

Pesticides attack the immune system. Both pyrethroids and organophosphates have been found to specifically inhibit a critical enzyme in white blood cells, and impair the growth and survival of those cells.[93,94] A World Resources Institute's report entitled "Pesticides and the Immune System: The Public Health Risks,"[95] concludes that immunosuppression by pesticides can provoke allergies, autoimmune disorders such as lupus, and cancer. It may also lead to increased susceptibility to viral infections (like COVID and WNV) and bacterial infections. The immunosuppressive effects of pesticides will be raised again in discussing whether pesticide spraying decreases the public health risk of WNV.

Animals studies have found that naled causes anemia, and birth defects in laboratory animals.[281]Labels on naled acknowledge it is a "severe" eye irritant, and "causes eye damage" and is "corrosive" to skin.[280]

The commercial preparation of naled, Dibrom, contains other ingredients that according to US pesticide law can be classified as "inert," but are hardly benign. They include the aromatic hydrocarbons naphthalene and 1,2,4- trimethylbenzene. Naphthalene is a toxin classified by the

EPA as a possible human carcinogen, and causes neurologic disorders and anemia in newborns. 1,2,4- trimethylbenzene is a tissue irritant and depresses brain function.

Naled has been banned in the European Union. The director of SLCMAD, Ary Faraji, has recently claimed that the EU didn't ban naled as we have publicly stated, rather that the manufacturer didn't apply for re-licensure for economic reasons. That claim is easily disproved. Official EU states in no uncertain terms that naled represented an "unacceptable risk" to human health and the environment, and that naled was to be removed from all European markets, Nov. 1 2012. [284, 285, 286, 287, 288, 289]

Naled can persist in the atmosphere for several days. Researchers at UC Davis measured both naled and its breakdown product dichlorvos in the air around a treated orange grove for three days after application.[299]

Low concentrations of naled celebrated by SLCMAD as "safe" also need to be viewed in this context: chlorpyrifos, a pesticide in the same category as naled, causes brain damage in humans at the lowest possible detectable dose. That's why the EPA recently banned 90% of uses for chlorpyrifos. A recent study found that naled, more than being just another organophosphate in the same category as chlorpyrifos, it joined chlorpyrifos in being the most neurotoxic of 30 organophophates.[300]

Naled is one of the most neurotoxic organophosphate pesticides in use

Piperonyl Butoxide (PBO)

Exposure to more than one pesticide at a time can cause synergistic toxicity.[96] But the SLCMAD's use of naled by airplanes and pyrethroids from the ground virtually guarantees it. The intentional role of PBO is to act as a synergist to pyrethroids, and it can magnify their toxicity by a factor of ten. There is every reason to believe that synergistic toxicity to mosquitos is matched by synergistic toxicity to humans. PBO's mode of action is the slowing down of metabolism of these compounds in the liver of the mosquitoes by interfering with cytochrome P450 monooxygenase enzyme. This enzyme is also the most important enzyme to humans' ability to metabolize toxic xenobiotics and endogenous compounds.

Not surprisingly, PBO also delays the metabolism of other toxins, which can obviously magnify their effect, including that of endogenous hormones. This further increases the risk to humans, especially with fetal exposure,[97,98] in precipitating impaired development and function of hormonal organs such the thyroid, pituitary, and adrenal glands. PBO is also classified by the EPA as a group C carcinogen. PBO is an immunosuppressant through its action of inhibiting lymphocytes.[99] Some studies in animals have shown PBO to have reproductive toxicity,[100,101,102] and it has been shown to cause liver tumors in rats and mice.[103]

There are several studies to suggest that PBO is, itself, also a neurotoxin,[104,105,106] which contradicts the official statement by the EPA that it is not. One of the most alarming studies on neurotoxicty from pesticides, published in the flag ship journal of the American Academy of Pediatrics, showed that children in the highest PBO exposed group, compared to the lowest exposed group, showed delayed neurodevelopment at 36 months, and loss of intelligence of about 4 points, comparable to that from lead toxicity.[107] This clearly identifies this "non-pesticide" ingredient as yet another potent neurotoxin in SLCMAD's pesticide inventory. The toxicity impact of PBO is likely enhanced by the fact that humans lack serum carboxylesterases, a primary mechanism of pyrethroid detoxication through hydrolysis.[37]

Other studies show that the toxic synergism of PBO when mixed with pyrethrins perpetuates its ecological impact.[108]

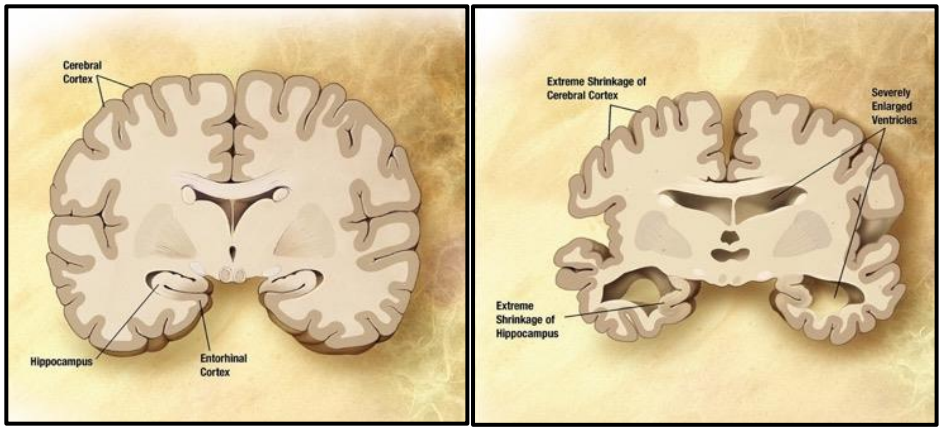
Other Pesticides

SLCMAD's EA also says that "other EPA approved adulticides with the same class of active ingredients may be used as well." Given the broad latitude that SLCMAD is allowing itself to use virtually whatever type and amount of pesticides it chooses, and without any meaningful medical scrutiny, and given that most pesticides have similar modes of action, it is also important to look at the medical literature on other pesticides used for mosquito control and pesticides in general.

Studies of pregnant women exposed to more pesticides, including organophosphates, gave birth to infants with smaller head size, thinner cerebral cortex, and other brain architectural abnormalities.[69,109,110,111] Prenatal and first year of life exposure to organochlorides, DDT, organophosphates, and other common agricultural pesticides increases the risk of intellectual disability, developmental delays, and autism.[112,113,114,115,116] A meta-analysis showed impairment of cognitive functions such as psychomotor speed, executive function, visuospatial ability, working and visual memory with low dose exposure to pesticides.[118] Neurodevelopmental scores at age 7 are lower with prenatal exposure to chlorpyrifos. Children in the top 25 percent of exposure levels scored 5.5 percent lower in working memory tests and 2.7 points lower in IQ.[71,72,119] One study even showed more behavioral problems in children during the peak of pesticide spraying season.[120] Numerous other studies show children growing up near agricultural pesticide use have higher rates of autism.[117,121]

Risks of Parkinson's Disease, Alzheimer's and adult cognitive decline are increased with pesticide exposure.[118,122,123,124,125] Individual's with a particular genetic make-up may be particularly sensitive to the neurodegenerative effects of certain pesticides.[123,125,126,127,128]

Maternal exposure to pesticides, including OPs, during pregnancy correlated with shorter gestational age, smaller length and lower birth weight.[129,130] Women exposed to higher levels of pesticides have higher rates of infertility,[131] and men have lower sperm counts. Pesticide exposure in women is associated with higher rates of endometriosis probably through their action as endocrine disruptors.[132]



Normal brain, frontal plane Alzheimer's brain, frontal plane

In addition to contributing to neurologic diseases, impaired intellect, and endocrine disruption, repeated spraying of neighborhoods in mosquito abatement programs creates problems for chemically sensitive individuals. Multiple chemical sensitivity syndrome can afflict as much as 15-20% of the adult population.[133,134] For them, chemical exposures can be a debilitating nightmare that includes profound fatigue, nausea, coughing, bronchospasm, rashes, severe headaches and impaired mentation. Insecticides are the most common triggers for the syndrome which can be precipitated by as little as one exposure event. "In a survey of 6,800 persons claiming to be chemically sensitive, 80 percent asserted they knew when, where, with what, and how they were made ill. Of the 80 percent, 60 percent blamed pesticides." Even a single spraying can provoke clinical symptoms in [genetically] predisposed individuals.[135] In 2008, the Center for Public Integrity analyzed 90,000 reports of adverse reactions to pyrethroids and found they had increased 300 times over ten years.[136] If the intent of pesticide spraying is to protect public health, the impact on chemically sensitive individuals is by itself enough of a reason to stop pesticide spraying.

Pesticide exposure can cause genetic damage.[137] Numerous studies have found that pesticides, including pyrethroids and OPs, can precipitate oxidative stress by increasing free radicals inside in the cell, damaging such critical macromolecules as RNA, DNA, DNA repair proteins and other proteins, causing genetic mutations, compromising antioxidant defense mechanisms and detoxification and scavenger enzymes.[138,139,140,141,142] This is not only a mechanism for causing a diseases like cancer, but is also a pathway for causing harm to subsequent generations.

Given the genotoxicity it is not surprising that numerous studies have found significant associations between pesticide exposure in general and several types of cancer, such as non-Hodgkin's lymphoma, multiple myeloma, soft tissue and lung sarcoma, pancreatic, stomach, liver, bladder, and gall bladder cancer.[143]

Genotoxicity and endocrine disruption are the likely mechanisms for reproductive pathology associated with pesticides. "Every class of pesticides has at least one agent capable of affecting a reproductive or developmental endpoint in laboratory animals or people,"[144]according to the author of a review article on the subject.

Disruption can occur in all stages of hormonal activity: hormone synthesis; hormone release and storage; hormone transport and clearance; hormone receptor recognition and binding; hormone post receptor activation; thyroid function; and the central nervous system.[145] In epidemiological studies, pesticides are linked with menstrual cycle disturbances, endometriosis, reduced fertility, prolonged time-to-pregnancy, spontaneous abortion, stillbirths, and birth defects.[146,147,148,149,150,302]

One of the researchers that helped discover the effect of pesticides on epigenetics and transgenerational harm, Dr. Paul Winchester, a pediatrician, said this, "Every one of the chemicals [pesticides] tested so far produces infertility, and the industrial world has reached the lowest level of fertility on record."[151]

Other research has found subsequent, multi-generational harm after exposure to just the first generation. Even more alarming, the pathologies were more extensive and more severe in subsequent generations.[152] Those diseases included prostate disease, obesity, kidney disease, ovarian disease, and birth abnormalities. Other studies showed not only does pesticide exposure significantly increase the risk of type II diabetes and obesity in humans and obesity in animals, but the effect can last through three generations.[153,154,155]

The insecticide pyriproxyfen used against mosquito larvae, causes reproductive abnormalities in keystone organisms persisting for multiple generations. Even 71 parts per trillion produced abnormalities.[156]

PFAS Contamination

Recent news reports have revealed that mosquito abatement pesticides are widely contaminated with one of the most toxic group of chemicals known, the C8 group, or PFAS. This family of compounds has been linked to a long list of disorders and disease, including: liver toxicity, immune disruption, developmental disorders, birth defects, thyroid disease, ulcerative colitis, and cancer of the kidneys, testicles, and pancreas.

The principal characteristic of the environmental and human toxicity of these compounds is that they do not break down either endogenously or exogenously, accumulating over time. Because of that their common nickname is "forever chemicals." They have been used to extend shelf life and increase dispersion of pesticides, and may also exist as a contaminant from the containers used to store pesticides. Manufacturers may get away with using the chemical because they claim it is inert, and therefore not required to be listed as an ingredient.

PFAS compounds are among the most toxic compounds ever produced by industry, yet they are completely unregulated by the EPA. This despite the EPA having concluded that anything above 70 parts per trillion (ppt) is not safe. Seventy parts per trillion is the equivalent of a few grains of

sand in an Olympic size swimming pool. Even that level is widely considered far too high.[157] For example, the Agency for Toxic Substances and Disease Registry has recommended a limit of 12 ppt. Michigan considers one of these compounds unsafe at concentrations greater than 6 parts per trillion.[158] PFAS compounds bioaccumulate in the food chain. Recently the fish in southeastern Michigan were declared unsafe to eat because they were contaminated with concentrations of PFAS thousands of times greater, i.e. in parts per billion.

Massachusetts recently discovered significant PFAS contamination of pesticides that were widely used in that state. Afterwards, elevated levels of PFAS were found in municipal drinking water in Massachusetts. PFAS contamination of the environment and public drinking water is already a nationwide, serious concern.

Adding more toxic compounds to the pesticide mix raises the possibility of further synergism. SLCMAD is merely relying on assurances from the pesticide manufacturers that the pesticides it is spreading all over the city do not contain PFAS. This is wholly adequate to address the concern. They should be doing their own testing to make sure. If contamination is found it would add yet another reason to reject the spraying program.

Health Risk for SLCMAD's Pesticide Workers

When weighing the risks and benefits of spraying, SLCMAD should also consider the extensive medical literature on the health consequences to those who are occupationally exposed. The health consequences just to their pesticide workers would likely more than offset any benefit to the community in theoretically reduced cases of WNV.

Certainly the many studies on the increased risk of neurologic disorders and neurodegenerative diseases like Parkinson's, Alzheimer's and cognition decline should be of serious concern to pesticide exposed employees.[70,122,159,160] Just one significant pesticide exposure "event" is enough to precipitate significant cognitive decline among pesticide applicators.[161,162] Research cited earlier in this report about a significant increase in all-cause mortality with pesticide exposure[53] is particularly relevant to the employees, as are the studies linking exposure to type II diabetes,[153] rates of depression and suicide.[163]

Pesticide applicators, including those that used permethrin, have shortened telomeres, chromosomal markers of shortened life expectancy.[164] Pesticides are toxic to the bone marrow, increasing susceptibility to aplastic anemia, a frequently fatal blood disorder.[165,166,167] Widespread media attention has been generated over the lawsuits regarding frequent use of glyphosate and cancer, especially lymphoma.[168] Women exposed to DDT before puberty, especially during infancy, had much higher rates of breast cancer.[169]

Men exposed to more pesticide residues from eating fruits and vegetables have lower sperm counts,[170] and women have more problems with endometriosis,[96,302] and reproductive toxicity, including birth defects and miscarriages.[171]

Looking for genetic toxicity and mutagenic damage in those occupationally exposed among agricultural and public health care workers, researchers from several studies found "a high rate of oxidative and DNA damage," compared to non-occupationally exposed controls.[143,172,173] Some studies actually showed that the disease burden increased among subsequent, unexposed generations.[17]

2. Decisions on the risks vs. benefits of exposing the public to dangerous chemicals should not be made by people with no expertise in public health, toxicology, or environmental toxins.

Decisions that affect public health should only be made by public health experts. Decisions about whether to expose hundreds of thousands of people to repeated applications of chemicals proven to harmful to public health, wildlife, and the environment in general should only be made by a panel with expertise in those disciplines. SLCMAD does not have that expertise. The director who seems to be the primary decision maker, has a PhD in entomology. That is not synonymous with expertise in the relevant disciplines. While there is a physician on SLCMAD's board, she does not demonstrate any real expertise on these issues.

3. Spraying Pesticides Increases Air Pollution

SLCMAD acknowledges that the spraying will release VOCs, but then dismisses the significance stating, "this activity would not exceed local standards for air emissions and would not result in nonconformance with the Clean Air Act" (CAA). This is irrelevant and is misleading regarding public health consequences. It reveals that the SLCMAD does not understand the significance of VOCs or how air pollution affects public health. It is another manifestation of their lack of public health/environment expertise.

Given the time of year of spraying(summer and early fall), warm temperatures enhance VOC volatilization. VOCs are a precursor of ozone in the summer and of particulate pollution (PM2.5) in the winter, both of which have been recognized as serious health hazards for over 50 years and regulated as such by the Clean Air Act (CAA). Utah is already struggling with high summer time ozone levels statewide, and the Wasatch Front has perennially been in violation of EPA's 24 hr. PM2.5 standard since 2006. Rural parts of Utah are experiencing higher summer time levels of

ozone than the Los Angeles Basin. Almost undoubtedly the VOCs from spraying pesticides increase ozone levels locally, as well as at sites remote from the sprayed area, as ozone can form thousands of miles away from where its precursors are released.

Most pesticide mixtures that contain solvents release high rates of VOCs.[175] In the San Joaquin Valley (SJV) of California, 65% of VOC emissions are from non-fumigant pesticides.[175] In the SJV, VOCs in the air and soil spike after pesticide spraying, and ozone spikes up to 15 ppb occur downwind immediately afterward, and don't return to pre-spray baseline levels for 1-2 days.[176] The study also detected predominantly high molecular weight aromatic hydrocarbons, the most toxic type of VOCs. With multiple rounds of spraying these ozone spikes will occur repeatedly throughout the season creating a significant source of summer time ozone.

Any presumption that short-term spikes in VOCs and ozone from pesticide spraying will not impact public health reflects a misunderstanding about the pathophysiology of air pollution. Virtually all of the components of air pollution--particulates, ozone, VOCs, toxic chemicals, NOx, and SOx-–precipitate an immediate inflammatory response that takes days or weeks to fully subside. The clinical consequences, including heart attacks, strokes, and increased mortality, are apparent within hours, and don't return to baseline for weeks or longer.[177,178]

Repeated episodes of elevated VOCs and ozone during a several month-period represent a risk to fetal development and viability for any exposed pregnant mothers during that time period.[179,180] Increases in ozone just in the few days prior to delivery are associated with increases in still birth.[181] Furthermore, the zone of exposure to spikes of ozone and VOCs will almost certainly be larger than that for pesticide drift.

Reducing VOCs has been a prominent part of the Utah Division of Air Quality's strategy to improve Utah's air quality.[182] Yet because the release of VOCs is from a mobile source, not a stationary source, the DAQ does not require a permit for the spraying. That is not because these VOCs don't matter, it is only because of a regulatory loophole that should be closed.

SLCMAD's statement in the EA, "this activity would not exceed local standards for air emissions and would not result in nonconformance with the Clean Air Act," needs to be addressed. There is no local standard for VOCs. Neither is there a state or federal standard for VOCs and the SLCMAD statement is not acute or informative on the issue of health consequences. In addition to their contribution to PM2.5 and ozone, there is a wealth of research showing that VOCs are also, themselves, one of the most hazardous components of urban and industrial air pollution. They are potent carcinogens, reproductive toxins, and neurotoxins. Unfortunately, this is unaddressed by the CAA and certainly not by SLCMAD's EA. Multiple events of aerial pesticide spraying would add to the already disproportionate toxic air pollution burden borne by the nearby residents of the West side, North Salt Lake and Bountiful.

Furthermore, VOC emissions from the spraying and the ozone spikes will occur during wildfire season, when we are now consistently experiencing some of the worst air quality of the year. The addition of VOCs to an already unhealthy backdrop of wildfire pollution (from both PM2.5 and ozone), is not a trivial matter, cannot be brushed off by SLCMAD.

4. Spraying pesticides has created a chemical arms race, is not effective in reducing mosquito populations long term, is losing its effectiveness even in the short term, and can even be counterproductive.

SLCMAD acknowledges that spraying is not effective. Their EA states, "In many instances, the numbers of mosquitoes collected in some of the traps do not decrease after aerial ULV adulticide operations are conducted." The obvious questions are: "Then why continue spraying?"

For effective mosquito control 90% of adults must be killed.[183] Only a few studies of the efficacy of spraying have been reported. Most of those studies do not suggest that kill threshold is achieved or that the strategy works. After ground spraying only a 34% reduction was achieved in Greenwich, CT, and in Houston only a 30% reduction. Little is accomplished unless spraying is done at least every 7 days. Zero mosquitoes will be killed upwind by an insecticide spray, and the spray will not eliminate larval mosquitoes.[184] The average upwind and downwind kill is only 21% to 45%.

Using ULV aerial equipment results in only 10% to 25% of the insecticide reaching the general target area, and up to 90% drifting away from the target into the environment at large, i.e. over the near-by communities and the Great Salt Lake.[185,186]

Depending on the study, the amount of pesticide that actually makes contact with a mosquito target can be less than 0.0000001% of the insecticide applied, but the average is probably around 0.0001% for flying insects (obviously more difficult than crawling insects).[187] Sprays must be composed of tiny particles so they will float in the air rather than fall to the ground, which is the purpose of the ULV technique. To ensure insects fly into the insecticide spray droplets, they must have a size in the range of 2-16 microns.[188] Even low winds easily blow mists of those size particles away from target areas.

Pesticide Resistance

Ecologist Garret Hardin has stated that "every biocide selects for its own failure." Continued use of pesticides creates resistant "super-mosquitoes" that require ever increasingly toxic chemicals to kill them.[189]

The resistance of insects like mosquitoes to pesticides is well established evolutionary science and has been observed in just about every country over several decades. Mosquitoes that survive pesticide exposure pass on genetic adaptations to their progeny that allow them to start developing resistance. Because of the short life cycle of mosquitoes that resistance can emerge very quickly. While it is common for mosquito numbers to drop for a few days after a spraying, the success is short lived because a new generation of more resistant mosquitoes quickly replace the deceased. A catch 22 is created. If spraying is done frequently enough to reduce the number of mosquitoes, it simultaneously accelerates the evolution of genetic resistance, and the resistance is passed onto to subsequent generations via epigenetic modifications such as DNA methylation, histone

modifications, and small RNAs, triggered by insecticide-induced stress.[190] Pesticide "priming" may even enable insects to be more tolerant of other types of induced stress, offering further survival advantages.[190] In fact it is worse than a catch 22. There is evidence that human disturbance of an area, including pesticides spraying not only increases the number of mosquitoes, it increases the number of disease-carrying insects.[191]

In hundreds of insect species, repeated application of pesticides on broad populations results in large numbers of resistant insects thriving in the sprayed areas. For example, the number of *Anopheles albimanus* mosquitoes in Guatemala that were responsive to DDT dropped from nearly 100 percent in 1959 to approximately 5 percent 20 years later.[192]

The headline of an article in Science Magazine from 2016 reads, "After 40 years, the most important weapon against mosquitoes may be failing."[193] It goes on to say that mosquitoes are developing resistance to pyrethroids "at alarming rates." The article quotes Maureen Coetzee, director of the Wits Research Institute for Malaria at the University of the Witwatersrand in Johannesburg, South Africa. "Nobody dreamt that insecticide resistance would spread the way it has spread throughout Africa." In Africa resistance to five mainstream insecticides increased dramatically between 2005 and 2017.[194] In Puerto Rico, which has been battling Zika cases since 2016, pyrethroids have become ineffective.[195]

Applying pyrethroids as surface sprays leads to a significant increase in the resistance of mosquitoes.[196] Mosquitoes from sprayed areas tolerated ten times the amount of chemical as did mosquitoes from non-sprayed areas.[197] Within a two-week lifespan, mosquitoes can develop resistance within a single season. A 2003 study found that WNV and malaria infected mosquitoes developed resistance to organophosphate insecticides as a result of a single genetic mutation.[198]

Damage to Mosquito Predator Populations

Of the numerous predators to mosquito larvae--dragonflies, ladybugs, lacewings, goldfish, guppies, fish, bats, and several types of birds—almost all have a longer life cycle than the mosquitoes. If a pesticide harms both mosquitoes and their predators, the later will take longer to restore their numbers giving the mosquitoes an advantage with each spraying.

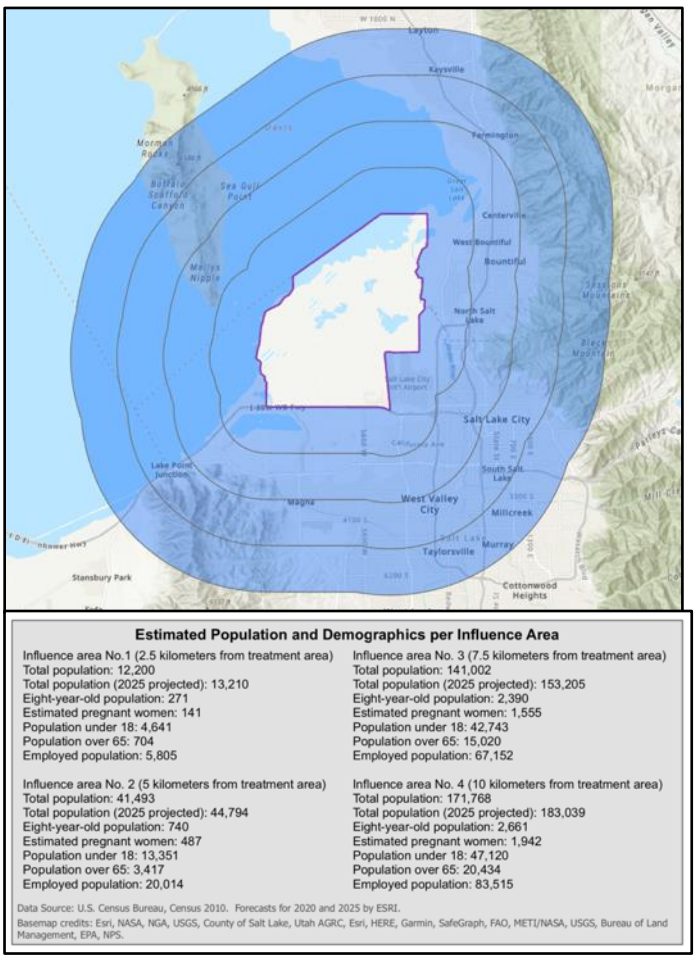
A headline in National Geographic reads, "How pesticides can actually increase mosquito numbers."[199] The article cites a study that shows mosquitoes evolve resistance to common pesticides but their predators, in this case damselfly larvae, have not developed that resistance. The end result is that pesticide spraying is actually counterproductive, doubling the number of mosquitoes in sprayed areas compared to unsprayed areas. The lead author of the study is Edd Hammill from Utah State University.[200]

Attempts to control medfly infestations in Florida and another in California, both using OPs backfired because beneficial insects and the pests' natural predators suffered greater damage. It took more than ten years for California agencies to acknowledge the pesticide spraying failure and replaced it with before preventative, nonchemical control program.

Damage to sympatric species

Spraying can adversely alter the balance with sympatric species (species that occupy the same ecological niche).

Over eleven years, Cicero Swamp in New York State was sprayed fifteen times with naled. During that time the mosquito population grew fifteen-fold.[201] When the populations of non-EEE [Eastern Equine Encephalitis] carrying mosquitoes were killed off, the EEE-carrying mosquitoes took their place. "In their research summary the authors of the study noted that the increase in disease- spreading mosquitoes 'discredits the rationale that preventive applications of naled reduce the risk of EEE."[202] The authors also concluded that "Ecological balance between the 2 species [of mosquitoes] may be restored by reducing naled applications to these swamps."



Data and graph courtesy of Vásquez Analytics Consulting

5. We Must Not Allow a Cure Worse Than the Disease.

The CDC states that adulticides should only be used as a last resort. "The underlying philosophy of mosquito control is based on the fact that the greatest control impact on mosquito populations will occur when they are concentrated, immobile and accessible. This emphasis focuses on habitat management and controlling the immature stages before the mosquitoes emerge as adults. This policy reduces the need for widespread pesticide application in urban areas."[203] We are hardly in a state where a "last resort" is needed or even helpful.

It is no small irony that the ostensible public health goal of spraying is to avoid significant neurologic disease and disability (and very rarely death) from WNV in a small number of people. But in doing so, the most common public consequence of spraying is to accept a risk for a small amount of neurologic disease in a large number of people.

While all the factors in the epidemic of autism and brain developmental disorders in children have not been completely determined, key insights have been established that are relevant to this issue. About two thirds of the risk for autism comes from exposure to environmental toxins at an early stage in brain development. The other third is from genetic vulnerability, much of which is sex specific.[204] That etiologic combination is undoubtedly also highly applicable to other brain developmental disorders.

Numerous studies show that typical urban air pollution is also a major risk for all brain developmental disorders. This combination means that those Salt Lake communities that are on the West side, near the airport, near the oil refineries and other industrial pollution, near the major freeways, and soon to be neighbors of the inland port, are already at significantly increased risk and are already victims of "environmental injustice." Pesticide spraying from SLCMAD in the Northwest Quadrant will make that even worse as pesticides are at the top of the list of environmental neurotoxins.

Attempting to quantify just one adverse public health end point, rates of autism, paints a very disturbing picture. Extrapolating from the observational New York study of aerial spraying and autism, for spraying in the Northwest Quadrant, this calculation can be made:

According to the Kem Gardner Policy Institute, the prevalence of autism is 1.7% of the Utah population.[205] Vasquez Analytics estimates that 12,200 people live within 2.5 kilometers of the proposed spray area, an area comparable in geographic positioning to that in the New York study. That suggests there are 207 cases of autism within the closest influence zone. If aerial pesticide spraying increased the rate of autism as much in Utah as it did in New York that means 77 cases could be attributed to pesticide aerial spraying. Even that is likely an underestimation of what may be happening in Utah for several reasons.

The New York exposure only involved one neurotoxic pesticide and the SLCMAD proposal involves two, plus a synergistic agent capable of greatly magnifying the toxicity. Furthermore, the control group in the New York study was not truly unexposed, they were still exposed to mosquito abatement pesticides (hydraulic spraying, manual dispensing of granules, or controlled droplet application) just not aerial spraying. The control groups also included zip codes as close as 2.5 km from the aerial spraying, so they were also likely exposed from chemical drift, just at lesser concentrations. The rate of autism is likely higher in influence area #1 than the Utah statewide average of 1.7%. The area is already exposed to numerous neurotoxic hazards, especially air pollution and lead that are known risk factors for autism, so the assumed 207 cases of autism is likely an underestimation. Finally, SLCMAD has acknowledged that they have been aerial spraying for decades, and routinely over an entire acreage of about four times the 43,000 acres proposed for Air Force spraying, and about which this calculation was made.

Even despite a rise in rates of West Nile Virus in 2021, cases have been dropping precipitously over the last 16 years and cannot be considered a health crisis severe enough to warrant exposing everyone to toxic chemicals

Even people bitten by an infected female mosquito, the carrier of WNV virus, run very little risk of serious illness.

SLCMAD offers a rationale for spraying stating that since 2003, there have been 422 documented cases of WNV in the state, with most of those in the Salt Lake Valley. If so, that is an average of only 23 cases a year, and 2021 had less than the average, and certainly not all those cases originated from mosquitoes from the Northwest Quadrant. That is hardly a serious public health threat. But looking deeper into the case history of WNV nationally and in Utah is even more revealing.

In Africa, where WNV was first found sixty years ago, very few human epidemics have been identified. WNV first appeared in the US in 1999. Northeastern communities like Boston and NYC that first responded to WNV with massive spraying have since scaled back their use of adulticides after demonstrating a lack of efficacy, and after three times more people became symptomatically ill from the spraying than from WNV.

Within a few years after 1999 there was every reason to believe that infection rates would subside as West Nile Virus becomes "endemic" to the United States, and will be characterized by low baseline infection rates interrupted by sporadic outbreaks. In fact that pattern appears to be the case. The number of cases in the US peaked in 2003 at around 10,000 and has declined significantly and steadily since. In 2020 there were only 557 total cases. Nationally, in 2002 there were 199 deaths, in 2020 there were only 38, and 40% of those were in Texas where the population increased 50% in those 20 years.[206] So far this year, nationwide, there have only been 725 WNV cases, and 35 deaths.

The case rate in Utah is following a similar step decline. The annual number of WNV cases peaked in Utah in 2006 at 158. Since 2008, the average annual number of cases in Utah has been 12, and in most years the number of cases is in the single digits. From 2008 to 2016 the average annual number of WNV case fatalities in Utah has been 0.2 cases. Adding in the two years of spikes in 2007 and 2017, that number increases only to 1.1 fatal cases per year.[207]

The Utah Department of Health (UDOH) stated that in 2018 11 residents contracted the virus and one person died.[207] In 2020 Utah recorded one case of "neuroinvasive WNV," and one case of non-neuroinvasive WNV, and no deaths.[208] As of October, the Utah Dept. of Health is reporting 21 human WNV cases in the state for 2021, and two deaths. Nationally, as of Oct. 5 the CDC has reported 725 WNV cases and 35 deaths.

While Salt Lake County often has the most cases of all the counties in Utah, the majority of Utah cases do not occur in Salt Lake County contrary to SLCMAD's claim. Nationally and in Utah, the number of cases of WNV is low, and for nearly 15 years has been trending lower despite occasional spikes. This is hardly a public health crisis in Salt Lake County warranting aggressive, repeated, wide spread spraying of biologic poison over large areas of the county.

The same trend has been observed in the United States with related infections, such as St. Louis encephalitis and Eastern equine encephalitis, where 30 or more years may pass between human outbreaks. The last outbreak of WEE (Western Equine Encephalitis) in Utah occurred in

1958. There has never been an outbreak of SLE (St. Louis Encephalitis) in Utah. It is disingenuous for the SLCMAD to list these two diseases as justification for spraying.

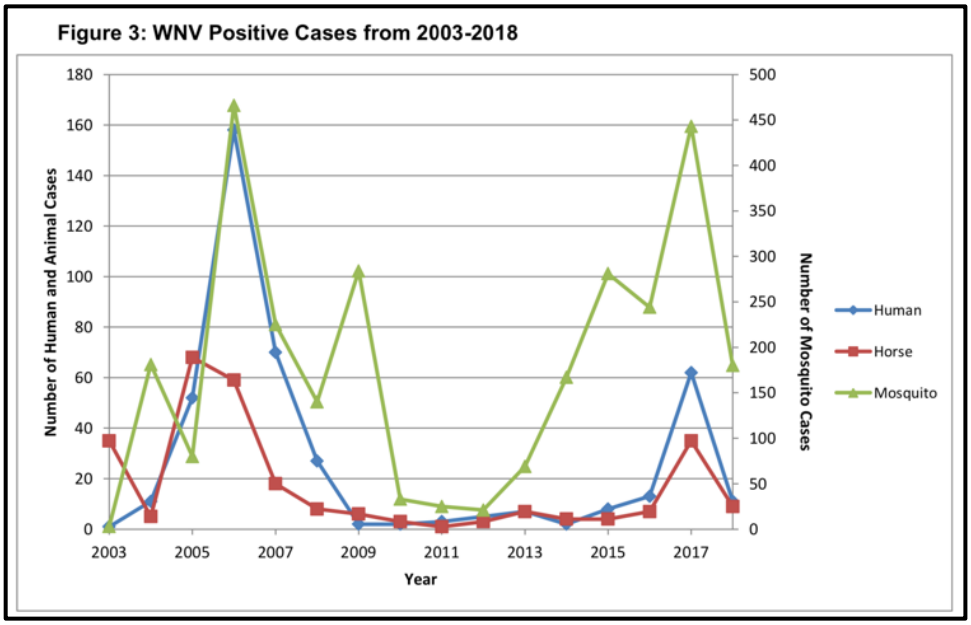


Chart of WNV cases in Utah 2003-2018.

Note the pattern on this chart that is consistent with a downward trend and low level in the number of WNV cases, despite a peak in 2017. Note also that after tracking closely with the mosquito counts in the mid 2000s, cases of WNV have not kept pace with the mosquito population. Indeed as hotter temperatures and extremes of precipitation (both floods and drought), signatures of the climate crisis, have led to increased mosquito populations, the number of WNV cases continues its steep decline. As of the w riting of this draft in October, the Utah Dept. of Health reports that the number of mosquito pools that are positive for WNV are at recording breaking numbers, about 8% of the total. The most recent year with that high of a percentage was 2017, and during that year there were 62 WNV cases. So the trend in human WNV cases is still downward despite a growing mosquito population. Further, although there are numerous reasons for an increasing mosquito population, the aggressive spraying campaign cannot be said to be suppressing that growth.

New York City, the epicenter of the 1999 WNV outbreak, found that out of more than 7 million, 62 people — or less than .0009% — became ill with the virus, and 7 died (one in one million). Putting this is context, that same year in New York City, 2,474 people died from influenza or pneumonia in 1999, 400 times the number of WNV mortalities.[209]

About 80% of people who are infected with WNV develop no symptoms, about 20% of people will develop mild symptoms. Less than 1% of individuals infected will develop a serious neurologic illness, such as meningitis.[210]

Dr. Gochfeld, Prof. of Environmental and Community Medicine at the Robert Wood Johnson Medical School and School of Public Health reported that, based upon his experience and other

West Nile Virus epidemics, typically less than one tenth of one percent of people bitten by infected mosquitoes develop any clinical signs of disease; in other words less than one in 1000 persons bitten by infected mosquitoes will develop some health problem. Gochfeld said:

> "*In weighing the risks and benefits of mosquito control, we should consider the disease itself and the risk to the human population. The media always paired the words "lethal" or "deadly" with "West Nile" or "encephalitis," reinforcing in the public's mind the danger from the disease. But it would be equally appropriate to characterize West Nile virus infection as "unapparent," "usually asymptomatic," or "occasionally serious." Seven deaths in a population of over 10 million people over a one month period is certainly tragic, but pales beside the number of deaths from many other diseases that are addressed less aggressively."*[211]

The U.S. Fish and Wildlife Services states, "Contrary to media descriptions of 'the deadly West Nile virus,' [it] is rarely fatal in humans. Less than one percent of people who acquire the disease will experience severe illness like encephalitis or meningitis. [214] Within this small proportion, the fatality rate is about 3-15%.[212] In other words the fatality rate is about one in 1,500 of those that become infected. Someone infected with WNV may have life-long immunity even if they show no symptoms. Most pregnant women infected with WNV give birth to normal, uninfected babies.

Pesticide exposure causes immunosuppression,[215] specifically inhibiting the survival and growth of leukocytes (white blood cells) by inducing apoptosis or cell cycle arrest and interfering with the function of each type of immune cells.[216] Those most at risk for a neurologic case or death from WNV are the immunosuppressed and the elderly, especially elderly males.[217] Pesticide exposure will only increase their risk. Those over 80 years old are at 43 times the risk for either one.[218] With such a low rate of death or serious disease for the population at large means that this is almost exclusively a disease of the elderly, especially elderly males, and everyone else must be at an almost imperceptibly low risk. A more complete public messaging strategy from health departments including this differentiation would itself go a long way to helping to protect the at-risk population while not creating unnecessary alarm and pressure for poor public policy.

6. Spraying Does Not Reduce the Incidence of WNV, WEE, or SLE

An analysis of rates of WNV showed that in 7 cities that used spraying programs for mosquito control, there were no lower rates of WNV compared to 7 comparable cities that did not use pesticide spraying. The communities that sprayed adulticides had an average of 1.37 people with West Nile virus per 100,000. The communities that didn't spray had 1.19 people with West Nile virus per 100,000.[219] Communities that did not spray put a strong emphasis on public education, selective larviciding and some employ seasonal help to locate breeding sites of mosquitoes.

The EA states that "90% of the mosquitoes" that are the intended target of the spraying are *Ae. dorsalis and Cx. Tarsalis*. But *Ae. dorsalis* do not carry pathogens, and therefore this species is only a nuisance mosquito, as are male mosquitoes of both species, because it is only female

mosquitoes that bite. High bite rates to humans are an indication that the relevant breeding areas are nearby, and very unlikely to come from an uninhabited area miles away.

The primary mosquito target relevant to public health is the female *Culex tarsalis*, the principal vector for WNV, and the EA does not give us any data on that specific species population. Adult Culex females live between 2-4 weeks, depending on multiple factors. Mosquitoes have a maximum flight range of between 50 m and 50 km, depending on the species. Few specimens survive long-distance flights "and do not relate to nuisance situations."[220] Female *Culex* mosquitoes are usually poor fliers and do not move far from their larval habitat. At the most they are capable of flying up to two miles away from their breeding grounds in uncommon circumstances. Even then they would have to find a new favorable breeding ground to present a meaningful risk for disease. So the key to preventing mosquitoes from infiltrating population centers is still eliminating standing water where people are.

In that case, SLCMAD's spraying 170,000 sparsely inhabited acres in the Northwest Quadrant would have little impact on the number of WNV infected mosquitoes in populated areas.

Pesticides can act as immunosuppressants not only to humans but to wildlife as well. Immunosuppression in wildlife makes them more susceptible to encephalitis if bitten by an infected mosquito, creating a feed-back loop due to more mosquitoes carrying the encephalitis pathogen and spreading the disease. Moreover, pesticide spraying can increase the biting aggressiveness of mosquitoes for up to two hours after a spraying.[221]

From an open letter written nearly 20 years ago, entitled, "Stop the Indiscriminate Spraying of 'Friendly Fire' Pesticides,"[222] the group Concerned Physicians and Scientists make these points. "There is a widely accepted erroneous belief that mass spraying of pesticides protects the population against mosquitoes. In fact, the opposite is true: the mass spraying will result in a deterioration of public health by exposing millions of people to "friendly fire" pesticides. Ironically, such spraying is especially dangerous to those with impaired immunity for whose "protection" such spraying is mainly being done."

7. Claims of Pesticide Safety Use Faulty Logic and Faulty Science

Because of the limited range of the female *Culex tarsalis*, SLCMAD cannot simultaneously claim their spraying is safe because it occurs only over unpopulated areas, but at the same time claim it is effective when it only occurs over unpopulated areas.

SLCMAD leans almost exclusively on EPA approval of the pesticides for their position that the program of spraying is safe for the public. American citizens, especially those in Utah, have a long and painful history of being victimized by the mistakes and the regulatory failures of government agencies. Public exposure to atmospheric radiation, nerve gas storage, lead, mercury, cigarette smoke, asbestos, Agent Orange, military burn pits, perchlorate, PCBs and countless other toxic chemicals, continued, in many cases, for decades after the science was clear that these were causing death and disease. And in many cases those exposures continue today, especially with pesticides.

It

It makes no sense to expose hundreds of thousands of people to neurotoxic chemicals in a vain attempt to prevent a neurotoxic disease in a few dozen people

seems we have learned nothing sixty years after Rachael Carson's *Silent Spring* should have transformed how we use chemicals that spread throughout our environment. Spraying against weeds and insects continues in Utah almost as a ritual conducted by every layer of government with little to no thought about the cumulative impact or the evidence for efficacy of the practice.

Several books written by EPA whistle blowers and extensive investigative reporting have exposed the disconcerting reality that the EPA has established protocols that protect chemical manufacturers at the expense of public health[223,224,225,226,227,228,229,230,231]The agency functions as much as a political agency as it does a scientific agency.

In the history of the EPA they have only completely banned eight substances and chemicals. The EPA still hasn't even completely banned lead in all gasoline, or all uses of asbestos. Under administrations of both political parties, the EPA has a long history of regulatory inertia lagging far behind the advancement of scientific evidence, as they are right now in not updating the national air quality standards. In most cases not only was the government far too slow in taking action, the pressure to act at all usually came from the public listening to the scientists.

We are in a similar situation now with the outdated and dangerous practice of pesticide spraying to control mosquitoes. Approval by the EPA of these chemicals for use on mosquitoes is no exoneration of the SLCMAD's strategy. Illustrating the point, the EPA has approved 85 pesticides that have been banned or are being phased out in other countries,[232] including some banned in countries like China and Brazil which are hardly known for robust environmental protection. In fact, recent documents reveal that the US EPA has been directly involved with chemical industry lobbyists in pressuring countries like Mexico to reverse their ban on certain pesticides.[233] The EPA has recently deliberately downplayed the extent of PFAS contamination in public drinking water.[234] The overwhelming number of pesticides ever withdrawn from the US market are done so by industry, not because of bans from the EPA.

Regarding the EPA approval process for chemicals, Dr. Winchester said, "…in the current paradigm…the only thing companies have to prove is that it doesn't kill you if you drink it or take a big dose of it."[151]

Furthermore, the EPA does little to evaluate mixes of multiple pesticides with synergistic agents, like the one SLCMAD uses, nor do they make any attempt to evaluate harmful epigenetic and genetic changes that are passed down to subsequent generations as mentioned earlier in this report, something toxicology risk assessments are not designed to detect.

Against this disconcerting backdrop it is all the more revealing that, nonetheless, the EPA did prohibit use of naled indoors or around the exterior of homes by anyone other than professional applicators, and it has been banned completely in the European Union.[235]

Many people who are likely to be exposed to the pesticides are also using mosquito repellants like DEET. A study from Duke University researchers has found that combination of DEET and permethrin exposure in lab animals causes the death of neurons and gross brain malformations.[236]

Claims of rapid breakdown of pyrethroids outdoors do not account for the pesticide seeping indoors within any residences near where spraying is conducted. Indoors they accumulate in dust and on household surfaces because they don't break down indoors like they do in direct sunlight outdoors. Children end up with 50% higher blood concentrations of these chemicals than adults do because they spend more time near the floor and have much more hand to mouth activity.[41] Human exposure also occurs because these chemicals linger on vegetation, vegetables and fruit.

At any one moment in time, between 40,000 and 50,000 women are pregnant in the state of Utah, many thousands of them will be exposed to some degree to known neurologic poisons from this aerial spraying proposal during the most critical stages of embryologic and fetal development. It has been universal advice from obstetricians for many years that their patients avoid any unnecessary pharmaceuticals, chemical exposures, and other contaminants to protect the integrity of fetal development, and that axiom certainly applies to pesticides.

Safety claims regarding pyrethroids do not adequately take into account cumulative exposures. It is one thing for SLCMAD to claim that one spraying event is benign. It is quite another to claim that multiple rounds of spraying, week after week, month after month, year after year are benign. Some residents will be exposed to repeated aerial spraying, and then any additional spraying that SLCMAD does in their neighborhood as a "service call." A pregnant mother living in one of the neighborhoods near the Northwest Quadrant could be exposed to repeated, and perhaps frequent doses of the insecticide by SLCMAD for weeks or months. Those same children could be further exposed after birth during each season of spraying, and again from the breast milk of its mother after birth. This cumulative and repeated exposure during the most critical stages of brain development in a person's life, represents a much greater level of risk than just one spraying event.

Because no one is exposed to just one toxic chemical, or even just one pesticide, the toxicologic studies on chemical exposures never adequately assess the clinical consequences of our total toxic exposures. Again, this is particularly relevant to fetuses and infants. An attitude on the part of decision-makers that justifies a specific source of toxins as being only a small part of the total problem is indefensible. The aphorism "death by a thousand cuts" shouldn't mean no one is responsible, it should mean everyone is responsible, and that everyone must stop contributing to the ultimate adverse outcome including the SLCMAD. And the most important "cuts" to eliminate are those closest to home.

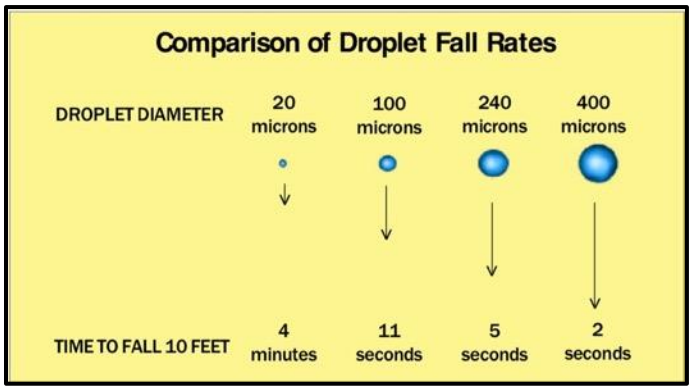
The aphorism "death by a thousand cuts" shouldn't mean no one is responsible, it should mean everyone is.

According to the Stockholm Convention,[237] an international treaty intended to minimize the environmental and global health consequences of persistent organic pollutants (POPs), organic compounds with an atmospheric half-life of over two days are considered POPs. By that definition these mosquito pesticides are considered POPs. The EPA states, "Because they can be transported

The more effective the spray, the more likely it is to drift away from the target area and into nearby neighborhoods.

by wind and water, most POPs generated in one country can and do affect people and wildlife far from where they are used and released. They persist for long periods of time in the environment and can accumulate and pass from one species to the next through the food chain."[238] Ten of the 12 chemicals banned in that treaty are pesticides.

Between 15 and 40% of applied chemicals are dispersed in the atmosphere by volatilization or droplet drift processes.[239,240] Pesticide off-gasing can continue for many hours or even days. When this occurs, the pesticide vapors may be subject to multiple, consecutive inversion cycles (more about that below), increasing the fugitive spread, and decreasing the dispersion predictability. Through either or both mechanisms, the pesticides can travel far from the site of release, and given that the breakdown times for OPs can range from several days to a few months, there is every reason to be concerned for the potential for spread of the actively toxic chemicals far off site.



In the EA, SLCMD cites difficult circumstances for effective spraying in the target area stating, "Some of the challenges to aerial adult mosquito control in the Salt Lake City area include extreme temperature inversions that occur at sunset and remain until sunrise." Indeed, Salt Lake City is ground zero for temperature inversions. North Dakota State University (NDSU) states, "… some of the seemingly best weather conditions for pesticide application are often the worst. That is because those conditions are caused by air temperature inversions. Air temperature inversions provide near-perfect conditions for tiny, aerosol-size droplets to drift away from their targets… Spraying during an inversion may cause increased lateral movement of fine drops and pesticide vapor." [241]

SLCMAD states that spraying only will happen when wind speeds are between 1 and 10 mph. But the NDSU advisory is, "Wind speeds 4-6 mph do not disrupt the inversion, and under these circumstances the smallest size droplets can drift laterally. "Smaller droplets (200 microns in diameter and less) fall as little as a few inches per second and may float along with the air for long distances."[241] "Even if the wind speed is only 1 or 2 mph, a small droplet can move a significant distance." Indeed, while the smaller the droplet size the more effective the spray will be against flying insects, but the more likely that spray will drift off target.

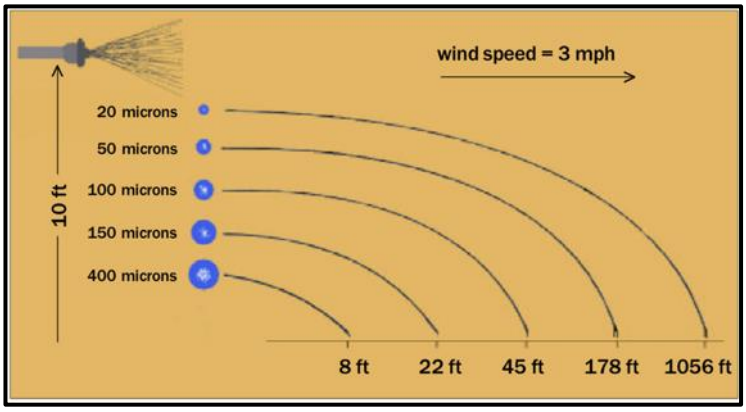
SLCMAD's airplane spraying is conducted from a height of between 100 and 300 ft. The Biomist label says that with aerial spraying below 200 ft. "the spray equipment must be adjusted so that the volume median diameter (VMD i.e. half of the total volume of spray droplets are larger than the specific size and the remaining half of the volume of spray droplets are smaller than that size), produced is less than 60 microns, and that 90% of the spray is contained in droplets smaller than 100 microns." For aerial applications above 200 ft. those stipulations change to 70 microns and 145 microns. In turn, the label for Dibrom states that for aerial application (it does not specify

height from the ground), the VMD must be less than 60 microns, and that "90% of the spray [must be] contained in droplets smaller than 115 microns."

However, the State of California's Department of Pesticide Regulation, Pest Control Aircraft Pilot Study Guide[242] goes to great length in discussing the inverse relationship between droplet size and the potential for drift (and from which both of the above diagrams were taken). For example, it states that "it takes approximately 4 minutes for a 20-micron droplet to travel a vertical distance of 10 feet, it takes only 2 seconds for a 400-micron droplet to travel the same distance." Further, "research also has proven those droplets smaller than 200 microns are very prone to drift. Those that are 100 microns or smaller are defined as driftable fines…The droplet size at which spray drift becomes a concern is 200 microns and below…Droplets smaller than 50 microns in diameter remain suspended in the air indefinitely or until they evaporate. Droplets of this size have no benefit to a pest control program because they are never likely to reach target surfaces." This suggests that aerial spraying cannot be simultaneously safely limited to a specific site and still be effective. The more effective the spray, the more likely it is to drift away from the target area and into nearby neighborhoods. We emphasize that this discussion assumes a height of 10 ft. from the ground, yet all these conflicting considerations would be far more applicable to a height of 100 to 300 ft., the height from which spraying airplanes typically fly.

Moreover, evaporation of the spray begins as soon as a droplet is released into the atmosphere. The longer it is exposed, the smaller the droplet becomes, and the more it will drift. Hot summer air temperatures and low humidity will accelerate evaporation and therefore drift.

Vapor drift can travel even further than particle drift.[243] A 2001 study by Texas A&M University found that pesticides can volatilize into the gaseous state and be transported over long distances rapidly through wind and rain.[244] A U.S. Geological Survey report reached similar conclusions.[272] A report from California in 2003, *Secondhand Pesticides*,[245] found that airbourne pesticides routinely exceed even far too lax health standards measured in the air, miles from where they are released. Concentrations of two different organophosphate pesticides were found near spray areas in concentrations that exceeded acceptable health levels by 184 and 39 times, respectively. The report also found that for almost half of the of pesticides applied in California, the concentrations in the atmosphere peak between eight and 24 hours after an application starts allowing plenty of time for further fugitive drift.



Drifting off target is not only an issue for pesticides leaving the targeted 170,000 acres, but also creating uneven distribution within that area, undoubtedly making some acreage receiving double doses and others receiving less or none.

NDSU emphasizes that, "…spray applicators need to use extreme caution in mountainous areas, protected valleys, basins, and the lower areas and shaded hillsides of some steeply rolling topography because cold air drainage can cause very intense inversions in these areas." And that, "Late afternoon spraying (two to three hours before sunset) has been found to be one of the worst times to spray. This is the time when inversions are beginning to form and often will intensify after sunset and continue all night…Based on these data and other observations, evening inversions pose a greater risk for spray drift…An inversion, plus low wind speed, is the best possible situation for long distance damaging drift of spray droplets."

Given these multiple conditions common to summer an early fall in the Salt Lake Valley, identified above as contrary to "safe" spraying, it is appropriate to ask if "safe" conditions actually ever exist. SLCMAD uses ULV nozzles, the purpose of which is to create extremely small droplets to prolong the suspension of the product in the air in order to intercept flying insects. But that also increases the drift of the product to non-target species and locations.

Several studies confirm that pesticides can become adsorbed to fine particulate matter, and as such can stay in the atmosphere between 3 and 10 days. Semivolatile pesticides, including permethrin, are mostly adsorbed to atmospheric particles, and "are very persistent with respect to the highly reactive hydroxyl radicals (OH) that is the self-cleaning agent of the atmosphere."[246] The authors of this study stated that the half-lives of this particulate phase for these chemicals can be several days to over a month, increasing the opportunity for these chemicals to travel long distances.[247,248] During that time the pesticide can travel thousands of miles, especially if released from aircraft up to 300 ft above the ground as indicated by the SLCMAD's EA.

Radioactively tagged pesticides have been shown to travel thousands of miles across oceans and to other hemispheres. For example, pesticides spread in the UK were found in the Southern United States 5-7 days later. Pesticides sprayed in the tropics have been traced to the Arctic. Naled is detectable in the atmosphere even in the absence of local naled use.[249]

As mentioned previously, pesticides are the chemical progeny of chemical weapons used in WWI. Few people are aware that the main reason why chemical weapons were banned after WWI is that it was widely recognized that it was impossible to control their atmospheric and geographic spread.[250,137] In fact in recent years chemical drift of the herbicide dicamba, with its high vapor pressure, has led to the chemical being banned in some states because of millions of acres of damage to non-target soybeans fields. Naled and its breakdown product DDVP also have relatively high vapor pressure which adds to its potential for fugitive drift.[251]

A pillar of the argument used to defend spraying is the contention that human exposure to the pesticides is low enough to be inconsequential. The validity of that assumption depends on whether traditional toxicology risk assessments (TRA)s by the EPA are valid expressions of human health harm. We join many other medical groups in the contention that they are not in and of themselves stand-alone determinants of disease potential and are often contradictory to research from the medical community. Please see Appendix A for a more detailed discussion of the scientific shortfalls of TRAs.

In 2011, the EPA made a statement supporting the supposed safety of pyrethroids based almost exclusively on experiments with adult rats. This type of narrow investigation is all too common a part of the EPA's reliance on toxicology risk assessments. For SLCMAD to de-emphasize or ignore all the other experimental research and the human epidemiologic research, is cherry picking the science, and guarantees a poor conclusion. Presumptions of safety of low dose exposure are no longer valid using this methodology, including for permethrin.[253]

Beyond poorly designed risk assessments, focusing on "extremely low" concentrations, is by no means the entire issue of human health risk. For the most vulnerable subset of the population, i.e fetuses and infants, what is most important is not the concentration of exposure as much as the timing of exposure in relationship to critical developmental windows. Conceptually it is fair to say if the chemical concentration is enough to be effective against mosquitoes after a single spraying, it is enough to be effective in harming fetal brain development after repeated spraying.

If that seems difficult to comprehend, consider this. A single drop of water, contaminated with just 1 part per billion of a toxic chemical, can contain 2.65 trillion molecules of that chemical, almost 30 molecules for every cell in a newborn baby's brain. In their action as endocrine disruptors, the health consequences may not be linearly related to the dose. More specifically, some harm can occur at barely measurable doses, putting at risk hundreds of thousands of residents of the Salt Lake Valley from this spraying.

There is an inherent contradiction in the contention that neighborhoods are far enough away to avoid clinically relevant exposure to the pesticides, but at the same time they are close enough to the breeding areas to be victimized by the targeted mosquitoes. Although a few mosquito species can become "tourists" traveling hundreds of miles, most prefer to stay within 50 to 100 meters laterally, or 10 meters vertically from their nest, as long as their food supply is adequate.[254] This is true of female *Culex tarsalis* mosquitoes, the species of greatest public health concern. They will not survive unless they are able to re-establish new breeding sites in human neighborhoods with food and water. The idea that the almost completely uninhabited (until the arrival of the inland port) Northwest Quadrant is a significant source of mosquitoes for humans miles away is not consistent with the evidence. If SLCMAD has evidence to the contrary they should offer that to the public. The real problem leading to human exposure lies in the neighborhoods themselves. In fact, most biting mosquito complaints from residents actually come from mosquitoes breeding on the residences' own property rather than from miles away.

The assumption that surrounding neighborhoods are far enough away to avoid clinically relevant pesticide exposure is further contradicted by studies that show significantly higher levels of pesticides in the indoor dust of homes near agricultural fields and in the concentrations of pesticide metabolites in children who lived in those homes.[252]

In a recent SL Tribune article the SLCMAD Director tried to make the case that the amount of pesticide used in this project would be much lower than is used in agricultural and implied that epidemiologic studies finding adverse health outcomes for communities near agricultural pesticide use were therefore not applicable. If that comparison is being used to dispute epidemiological studies from agricultural exposed populations, the Director is misinformed or being disenguous.

The EA says SLCMAD plans to use 0.5 to 1 fluid oz. of Dibrom (naled) per acre, but can use up to 2 fl. oz. every seven days, and further they can use 104 fl. oz. (10.73 pounds) per acre per year. It also opens the door to even more spraying. "More frequent treatments may be made to prevent or control a threat to public and/or animal health determined by a state, tribal or local health or vector control agency" (which we assume is the SLCMAD).

By comparison, the instructions on naled specify various limitations for agricultural use, including 0.9 to 1.9 lbs per acre per year for almonds: for beans and peas--4.2 lbs per season: for broccoli, cabbage, cauliflower, brussels sprouts, kale, and collards—9.4 lbs. per acre per season: for cantaloupes and muskmelons—1.9 lbs per acre per season. There was not a single crop listed for which the instructions on the use of naled, allowed as much pesticide to be used per acre as the SLCMAD has allowed for themselves in their EA.[255]

7. There are multiple oversights, inadequacies, omissions, inconsistencies, and errors in SLCMAD's Environmental Assessment of their pesticide use.

SLCMAD's EA states:

> *"Surveillance results by the Salt Lake City Mosquito Abatement District indicate that mosquito species present in the northwest quadrant of Salt Lake City are extremely numerous during summer months and capable of transmitting serious human diseases. The mosquito populations are large enough to cause human pain, discomfort, and stress. The nuisance caused by mosquitoes during the summer can decrease the overall morale and quality of life within Salt Lake City if the mosquito number are not controlled."*

The EA goes on to justify the spraying program with a two-fold "purpose and need." One, the mosquitoes are a nuisance. Two, to reduce "pathogen transmission." To use "nuisance" as a justification for exposing thousands of people to toxic chemicals is inappropriate and represents priorities that we believe much of the community would dispute. Likewise, "service requests," from the public cannot be considered justification for spraying environmental toxins. We contend that the community has a greater " purpose and need" to not be exposed repeatedly to neurotoxins.

The EA states, "In the many years of its use, there have been no reports of toxic effects of Biomist 30+30 to people, pregnant women, pets, or other mammals." The same could be said of just about any known toxins, radiation, lead, mercury, PCBs, etc. The toxicity to the population at large is primarily from chronic low-level exposure. The EA statement reflects a dangerous misunderstanding of the toxic effects of the product, as the previous part of our report provides in detail. We can only assume that the SLCMAD is alluding to acute, severe, life-threating reactions to the pesticide among those directly handling the product. Nonetheless, it reflects a completely inadequate appreciation of the public health consequences of the spraying program.

According to a letter from the EPA, the label on naled says "Do not apply over bodies of water (lakes, rivers, permanent streams, natural ponds, commercial fish ponds, swamps, marshes or estuaries)," and "This product is toxic to fish, aquatic invertebrates, and wildlife."[255] As close as the designated area is to the Great Salt Lake, this seems like it's use would be contradictory to what is allowed by even by the manufacturer.

That same letter says, "Applications must not occur during local, low-level temperature inversions. Temperature inversions restrict vertical air mixing, which causes small, suspended droplets to remain in a concentrated cloud. This cloud can move in unpredictable directions due to the light variable winds common during inversions." However, the label for Biomist (permethrin) says that best results are obtained with, "weather conditions conducive to keeping the spray cloud close to the ground. And inversion of air temperatures and a light breeze is preferable." It seems that the appropriate weather conditions are different and contradictory for each of the two pesticides.

The Biomist label also says,"This product is highly toxic to bees exposed to direct treatment on blooming crops or weeds," and the EA admits the same. However, the naled label says "Do not apply this product as an Ultra Low Volume (ULV) spray (>1/2 gallon per acre), or in any carrier other than water," and… "Aerial applications must not be made at a height greater than 10 feet above the top of the target plants unless a greater height is required for aircraft safety. Making applications at the lowest height that is safe reduces exposure of droplets to evaporation and wind."[255] All of these stipulations are contradicted by the plans detailed in SLCMAD's EA.

The naled label says, "Do not apply this product in a way that will contact workers or other persons, either directly or through drift. Only protected handlers may be in the area during application." SLCMAD does virtually nothing to notify the public prior to their spraying. The Director believes that it is ok to spray directly on anyone in the area because it is not forbidden by the EPA, stating, "If people prefer to stay inside when spraying takes place they can, but it is not necessary."

In Section 6 of the EA, Cumulative Impacts, there is no mention at all of cumulative impacts on human health which is a clear indication that the SLCMAD has no understanding of the issue, as detailed previously. In section 2.3 of the EA, various thresholds are listed to trigger aerial spraying on mosquito counts, but not on the number of mosquitoes infected with WNV. This again reflects an inappropriate priority by the SLCMAD. Public nuisance alone is not justification for spraying.

In section 2.5 after listing the products intended to be used, the EA then leaves the door wide open to use virtually any insecticide, "Because of operational, biological, ecological, or economic needs, other EPA-approved adulticides with the same class of active ingredients may be used as well." Given that the EPA has "approved" insecticides even more toxic than naled and permethrin, this phase must be stricken from the EA.

8. Pesticide spraying has adverse impacts on beneficial insects, bird populations, wildlife, the ecosystem of the Great Salt Lake and beyond

"Plants and insects are the fabric of this planet, and we're ripping it to shreds."

---Scott Black, director Xerces Society[273]

A recent headline in *Smithsonian Magazine* reads, "Insects Are Dying Off at an Alarming Rate"[256]Another from *National Geographic* reads, "You'll Miss Them When They're Gone."[257]

The increase in mosquito populations obscures the fact that globally the overall mass of insects and the number of insect species is in steep decline.[258] That this trend has been documented not just in agricultural areas, but also in the wild where insect populations have plummeted as much as 76%[259] over the last 27 years, speaks to the sensitivity of this part of the animal world, if not the entire animal world, to the damage being done by ubiquitous, low levels of pesticides found throughout the entire globe, including areas far removed from where spraying occurs.

Insects are irreplaceable in keeping the world's ecosystems and food chains viable, yet 40% of insects species are threatened with extinction.[260] Species higher in the food chain suffer population losses (like birds on the Great Salt Lake), waste is not broken down as efficiently, slowing the transition to nutrients, pest insects gain advantage over their predators, pollinator populations decline (some bumble bees are now endangered species [260]), and nutrition and water retention in soil declines which kills plant life and expands deserts.

While there are multiple causes, pervasive use of pesticides is a major contributor to this loss of biodiversity and insect life. Unfortunately, insects like mosquitoes, flies, cockroaches, and agricultural pests with faster breeding cycles appear to be thriving for multiple reasons, including pesticides killing their predators, and global warming increasing their geographic domain. Pressure for more and more pesticide use will likely be brought to bear, so it becomes increasingly important that SLCMAD not succumb to poor, counterproductive, unscientific policy. Poor local policy playing out in tens of thousands of locations has brought us to this global ecological precipice. Tunnel vision from the SLCMAD contributes to this much broader problem with its pesticide spraying program.

It seems we have learned nothing 60 years after Rachael Carson's *Silent Spring*.

We defer to other groups like the Audubon Society for comments on the impact of pesticide spraying on birds in the Great Salt Lake ecosystem. But from a larger perspective, a recent headline

in the *Scientific American* is chilling*; "*Silent Skies: Billions of North American Birds Have Vanished." Bird populations have declined by 3 billion since the 1970s, a decline of about 30%.[261] The cause of this alarming drop is multi-factorial, but there is no doubt that the loss of insects and the pesticides themselves have contributed significantly to this precipitous decline.
For more information and research on the impact on the Great Salt Lake ecosystem we refer you to the comments submitted by David Richards, Ph.D, OreoHelix Ecological.

9. There are Better Ways to Control Mosquitoes

Numerous cities and counties have adopted mosquito control strategies that do not include spraying adulticides. This group includes Lyndhurst, Ohio, Washington, DC, York County, Virginia, Dallas, Texas, Ft. Worth, Texas, Nassau County, New York, Marblehead, MA, Boulder, Colorado, Lane County, OR, and Seattle, WA.

Below are links to strategies in three communities that have been effective and avoided spraying adulticides: Boulder County, CO, Washington, DC, Madison, Wi., and an excellent control strategy from the Xerces Society.

https://www.publichealthmdc.com/environmental-health/pests/mosquitoes

https://storymaps.arcgis.com/stories/26548d1e7cae4b45b7f11c6c50e1aabc

https://doh.dc.gov/sites/default/files/dc/sites/doh/publication/attachments/Arbovirus%20Surveillance%20Mitigation%20and%20Prevention%20Plan_0.pdf

Conclusion

The scientific and empirical evidence is overwhelming that spraying adulticides to kill mosquitoes, especially aerial spraying, is ineffective, and can be even counterproductive, over the long term, and even the short term, to both goals of controlling mosquito populations and preventing West Nile Virus. Furthermore, the medical literature strongly indicates that routine aerial spraying over Salt Lake City's airshed represents a broad-based danger to public health. Utah Physicians for a Healthy Environment (UPHE) implore SLCMAD to end all of their insecticide spraying for mosquitoes, whether from back packs, trucks, or airplanes. This practice is an institutionalized relic of the 1950s and should be stopped immediately.

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Appendix A—Problems with Toxicology Risk Assessments

"The ultimate effect of these institutional defects is that chemical risk assessments in the US and the EU have a safety bar for approval that is so low that regulators virtually never decline to approve a chemical. In contrast, the exact same institutions use standards for taking any chemical off the market that are so high that such an event nearly never happens. Yet if both standards were based purely on science, as they claim to be, both bars would be the same height…
Chemical risk assessments themselves are the problem"

---Jonathan Latham, PhD,
the Cornucopia Institute

Virtually all toxicity data used by the EPA comes from a testing program called "toxicology risk assessments (TRAs)," the same approach that polluting industries, chemical manufacturers, and unfortunately regulatory bodies use to assess whether a certain project such as citing of a pollution

source, like a refinery or incinerator, or a chemical spraying program will create a public health risk. It is an antiquated, inadequate and scientifically deeply flawed process.

The science of toxicology is still tethered to a principle first advocated by the father of toxicology, Paracelsus (Theophrastus von Hohenheim), a 15th-16th Century Swiss physician, alchemist, and astrologer. His contribution to the Renaissance was the fundamental classic toxicology theory that "the dose makes the poison." In other words, there is a threshold at which low concentrations of toxic substances no longer present a threat to human health and above that threshold, the harm is proportional to the dose. That is still the basis of modern-day toxicology and government environmental regulations. But that assumption and the methodology based upon that assumption was flawed when it was adopted in the 1970s and it is all the more so now.

Regulatory toxicology testing primarily has consisted of high dose testing on adult animals, usually mice or rats, and subsequent testing at progressively lower doses in an attempt to establish a dose response model from which a threshold can be derived below which effects, usually death or cancer, could no longer be identified.

This methodology is deeply flawed for numerous reasons, and has contributed greatly to a barrage of unsafe and inadequately tested chemicals being unleashed upon the public and the environment.

How TRAs originated is instructive. They were first used by the EPA in 1975 under pressure from industry to dismiss health risks of one of the earliest known toxins, vinyl chloride. The EPA felt like they could respond authoritatively if they could calculate the number of cancers that would be caused by exposing the population to vinyl chloride. Federal regulators then started adopting that technique with many other environmental controversies and disputes with industry, and that methodology continues today. The makers of toxic chemicals and industrial polluters are content to see it continue because with supposedly hard, mathematical data, the risks of chemicals and contaminants are easily obscured. Below are eleven reasons why TRAs fail to identify many health risks, and usually underestimate the toxicity of chemicals.

1. Most of the research used in risk assessments is supplied by the chemical manufacturers themselves or from contract labs hired by the companies. The potential for corruption of that system is obvious, but starkly illustrated by the history of Industrial Bio-Test Labs (IBT). For three decades IBT performed one third of all the toxicology testing in the United States. Several of its executives were convicted in 1983 of extensive scientific misconduct for fraudulent research favoring industry.[262,263] IBT had performed over 2,000 key product safety tests resulting in the release of 212 pesticides. After an extensive review in 1983, EPA determined that only 16% of IBT's testing results were valid.[264] Only a tiny number of the invalid studies have been replaced, the agency admits, and most of those pesticides continue to be marketed, sold, and used.[262]

As disturbing as this scandal is, the EPA process created in the wake of that scandal is even more so. EPA has established a research protocol called Mutual Acceptance of Data (MAD), whose effect is to exclude truly independent, peer-reviewed scientific research, like from academia, in favor of industry produced research.

Jonathan Latham, PhD, writes for the Cornucopia Institute,…"no EPA employee ever sets eyes on the original experiments or the primary data, and only a handful can access even the summarised results. This system has the consequence of excluding any formal possibility that whistleblowing on the part of Federal employees or FOIA requests (from outsiders) might reveal fraudulent or otherwise problematic tests. EPA calculatedly turned a blind eye to any potential future wrongdoing in the full knowledge that the chemical regulatory system it oversaw was systemically corrupt."

2. TRAs overlook the presence of non-monotonic dose-response curves, i.e. the relationship between dose and outcome is not linear. There are hundreds of studies that show non-linearity is also true for other thoroughly researched toxins like air pollution and lead. Non-linear dose response curves open the possibility that adverse health outcomes occur at concentrations below the classic toxicological no-observed-adverse-effect level (NOAEL). Indeed, numerous studies have shown that some toxins are actually more dangerous at lower concentrations than higher ones. Furthermore, these magical thresholds of safety don't really exist even in those cases where the relationship is linear.

3. There are obviously numerous health outcomes beyond death and cancer that are more difficult or impossible to identify in animals, like infertility, immunosuppression, endocrine disruption, neurodegenerative diseases, altered behavior and subtle, reduced intellectual capacity, which are exactly the health outcomes of greatest concern with pesticides in general, and the specific chemicals that SLCMAD intends to use.

4. The impact of toxins on genetic and epigenetic integrity may show up in subsequent generations, even if they are not exposed. But examining subsequent generations are seldom part of the research.

5. There is increasing evidence that neurotoxicity can be very sex specific, something that is increasingly apparent in human studies, but seldom part of the animal studies done for risk assessments.

6. This strategy ignores critical differences in physiology, metabolism, microanatomy, hormones, liver function, and enzyme production between fetuses, infants, children and adults. Chemicals crossing the placenta and affecting fetal development has been an after-thought at best.

7. Toxicology risk assessments are basically mathematical calculations. These calculations dramatically oversimplify very complex biologic, physiologic and molecular processes that don't lend themselves to mathematical equations.

8. The assessments do not pass the acid test of scientific reproducibility. Two different "assessors" can come up with widely disparate results given the same input data, especially if they have different motivations—subjectivity rather than objectivity. William Ruckelshaus the former EPA director wrote in 1984, "We should remember that risk assessment can be like the captured spy: If you torture it long enough, it will tell you anything you want to know."[265] In 1991, the National Academy of Sciences wrote, "Risk assessment techniques are highly speculative, and almost all rely on multiple assumptions of fact -- some of which are entirely untestable."[266]

9. Through biological processes many toxic substances accumulate in organisms, concentrate in certain tissues of those organisms, and increase in concentration as they move up the food chain. Humans are at the top of the food chain and because our capacity to break down toxic chemicals is very limited, concentrations in our bodies usually increase over time. Lipophilic (fat-like) toxins will concentrate especially in human breast milk increasing the risk to progeny but that kind of exposure consequence is not addressed by the EPA's methodology.

10. Classical toxicology assessments almost never attempt to evaluate more than one toxin at a time, and the regulations derived from those risk assessments are similarly singularly focused. The default assumption is that the hazard posed by each individual compound, tested out of context and in isolation, can predict the hazard of the entire complex mixtures of chemicals. Obviously no one on earth is exposed to merely one chemical. No children on the West side, in North Salt Lake or West Bountiful are exposed to just one neurotoxin. Furthermore, the potential for toxicity may be much greater than just the additive sum of all these chemicals individually. Specifically, synergistic effects from chemical interactions are quite possible, if not likely, which would significantly increase the potential for health hazards.[267] In fact SLCMAD intends to use a synergistic agent that may increase the lethality of the chemical mix to insects, and the toxicity to humans by a factor of ten.

Under the absurd methodology of only considering one toxin at a time, a safe dose of aspirin, combined with safe doses of ibuprofen, Oxycontin, Toradol, Celebrex, acetaminophen, three glasses of wine, and a pint of Jack Daniels would all be considered individually "safe," but in reality might very well add up to be lethal. When a supposedly tolerable exposure to diesel exhaust is combined with a tolerable exposure to cadmium, fluoride, lead, arsenic, permethrin and naled, the end result can be an intolerable health consequence. But the source of each of these toxins can claim to the community that their toxin is safe.

Furthermore, it is routine for toxicology experiments to only use exposure to the "active ingredient" in a product, when the "inactive" compounds, like adjuvants and surfactants, are often toxic as well, and sometimes more so.

11. Traditional toxicology assessments wrongly assume that we have a comprehensive understanding of the complexity of biological processes and chemical toxicity when in reality there are vast information gaps. Lack of knowledge cannot be equated with safety; it can only be equated with lack of knowledge. Much of that lack of knowledge is deliberately orchestrated by the manufacturers. The most basic information is not available on over 90 percent of the industry inventory of chemical products. The Government Accountability Office says that 95 percent of the information given to the EPA by companies on their new chemicals are wrapped up in confidentiality claims, which makes them unavailable to regulators. Almost all the data that is available is supplied by the manufacturer whose obvious self-interest obscures objectivity. And the cumulative health impact of exposure to all these chemicals is ignored.

Manufacturers don't even have to reveal enough of the chemical mix of their products to allow government agencies to track their spread in the environment or to independently test for toxicity. The TSCA requires manufacturers to submit safety data only if they have it. Most don't, so the

EPA is left with computer models to predict whether chemicals will pose health problems, and no one believes that is adequate. In those rare instances where evidence stacks up implicating a chemical as a serious hazard, industry response follows a well-worn path. "By the time the scientific community catches up to one chemical, industry moves on to another and they go back to their playbook of delay and denial," said Deborah Rice, a former EPA toxicologist who now works for the Maine Center for Disease Control and Prevention.[268]

Despite all this, the anachronistic premise that the "dose makes the poison," established almost exclusively in lab animals, is still the basis of toxicology risk assessments. This testing frame work is now increasingly recognized within the medical community as a pillar of misunderstanding and misinformation routinely used by industry, especially chemicals corporations to claim their products are safe and which leads to regulatory agencies approving their use. It is this regulatory framework that the SLCMAD relies upon to exonerate their pesticide spraying program as a "safe," or "acceptable" risk to public health.

Because of the obvious short comings of risk assessments, countries in Europe began embracing a more protective approach to toxic chemical exposures guided by the "precautionary principle." The principle can be expressed this way. When there is reasonable evidence of harm, society is compelled to act to protect the public, rather than wait for scientific proof. This adoption of the precautionary principle was even written into the Maastricht Treaty that formed the European Union. Journalist Peter Montague describes this difference between European and American regulation of chemicals this way. The European model asks, "How much is avoidable?" The American model asks, "How much harm is acceptable?"[269] The United States' approach prioritizes protection of corporations and chemicals rather than public health. The precautionary principle has not been allowed to enter the arena of government regulation under either Democratic or Republican Administrations because of corporate pressure.

The result has been frank regulatory failures, and the failures started with original chemical safety law, the Toxic Substances Control Act (TSCA), passed in 1976, virtually written by the chemical industry. It allowed all 62,000 chemicals that were on the market at the time to remain so, unless the EPA found at some later time that they represented unacceptable risk.

Only undeniable health consequences and apparent toxicity after consumer use and wide spread environmental contamination of the products needed to be reported to the EPA. If this seems like closing the barn door after the horses have already escaped, bear in mind that before 1976, there was no barn door at all preventing or regulating chemicals that were unleashed upon the public.

Furthermore, the law only allowed testing if information from the chemical makers themselves suggested it was likely to be dangerous. Moreover, the law allowed chemical corporations to claim just about everything was a trade secret allowing them to hide critical information from the public, regulators and even health care providers. As a result, the EPA has only tested about 200 of all 140,000 industrial chemicals produced since WWII. For chemicals entering the market since 1976 they are also allowed unless the EPA again determines they constitute unreasonable risk.[270] But they only have 90 days to make that determination and hardly ever do they have all the data they need. The end result is virtually all the chemicals made by corporations will make it into the bodies

of consumers. For example, despite it being an undeniable killer of tens of thousands of people, it is still legal to manufacture and sell asbestos.

When the EPA tried to ban asbestos in 1991, the asbestos industry went to court, and a judge overruled the ban because they hadn't considered the costs of the ban as required by the deeply flawed TSCA. While asbestos use has plummeted, it is still allowed in brake pads and to manufacture chlorine. The EPA in its entire history has only succeeded in completely banning eight chemicals--PCBs, dioxins, fully halogenated chlorofluoroalkanes (ozone depleters), hexavalent chromium (the Erin Brockovich chemical), and four metal working chemicals. The last time the EPA banned anything completely was in 1984.[271] However it should be noted that the EPA did ban indoor use of one of the chemical groups that SLCMAD intends to use (organophosphates) which, given the EPA's history, speaks volumes about their toxicity.

Regulatory impotence flows from the default assumption that industrial chemicals are safe, unless or until, unequivocally proven unsafe. Essentially human consumers are today's guinea pigs of the chemical industry, just like the previous generation was for DDT, PCBs, asbestos, radiation and lead. Once a dangerous product is put on the market, it is only withdrawn after it has already caused large scale, undeniable damage and/or death.

Appendix B

A close examination of the literature cited by SLCMAD in their defense of pesticide spraying

Carney RM, Husted S, Jean C, Glaser C, Kramer V. 2005. Efficacy of aerial spraying of mosquito adulticide in reducing incidence of West Nile Virus, California. Emerging Infectious Diseases. 14:747-54.

Data from 2005--spraying works.

Currier M, McNeill, M, Campbell D, Newton N, Marr JS Perry E, Berg SW, Barr DB, Luber GE, Kieszak MA, Rogers HS, Backer LC Belson MG Bubin C Azziz-Baumgartner E, Duprey ZH. 2005. Human exposure to mosquito-control pesticides- Mississippi, North Carolina, and Virginia, 2002 and 2003. MMWR. 54: 529-532.

Not a study

Davis RS, Peterson RK. 2008. Effects of single and multiple applications of mosquito insecticides on nontarget arthropods. Journal of the American Mosquito Control Association. 24: 270-280.

Data from 2005—on "benign effect" non-target arthropods.

Duprey Z, Rivers S, Luber G, Becker A, Blackmore C, Barr D, Weerasekera G, Kieszak S, Flanders WD, Rubin C. 2008. Community aerial mosquito control and naled exposure. Journal of the American Mosquito Control Association. 24: 42-46.

2004 Study that showed less naled metabolites in urine after spraying than before, although metabolites till present 40 hours after.

Elnaiem DEA, Kelley K, Wright S, Laffey R, Yoshimura G, Reed M, Goodman G, Thiemann T,Reimer L, Reisen WK, and Brown D. 2008. Impact of aerial spraying of pyrethrin insecticideon Culex pipiens and Culex tarsalis (Diptera: Culicidae) abundance and West Nile virus infectionrates in an urban/suburban area of Sacramento County, California. Journal of MedicalEntomology. 45: 751–757.

2008 published date. An area aerially sprayed three consecutive night showed a 50% reduction in two species of mosquito that carry WNV 2 weeks later, but was not statistically significant for the species that is the primary vector for WNV in Utah, Culex tarsalis. No information on long term mosquito populations and no correlation with human WNV cases. Note that it required three consecutive nights of spraying to achieve even that temporary reduction.

Holcomb KM, Reiner RC, Barker CM. 2021. Spatio-temporal impacts of aerial adulticideapplications on populations of West Nile virus vector mosquitoes. Parasites and Vectors. 14:

While this modeling study is the most recent of the ones cited by the Director, citing data up to 2017, aerial spraying only reduced Culex tarsalis by 30.7%

Karpati AM, Perrin MC, Matte T, Leighton J, Schwartz J, Barr RG. 2004. Pesticide spraying for West Nile virus control and emergency department asthma visits in New York City, 2000. Environmental Health Perspectives. 112: 1183-1187.

SLCMAD's director keeps citing this study as evidence for no effect on human health. In addition to the comments made earlier in this report, please note this physician written editorial below that was published in the same journal in response to this study, mentioning many of the same critiques that we have. Also note that this critique was written in 2005 when the incidence of WNV was much greater than it is now.

Ziem G. Pesticide spraying and health effects. Environ Health Perspect. 2005;113(3):A150-A151. Doi:10.1289/ehp.113-a150a

"I noticed with interest the article "Pesticide Spraying for West Nile Virus Control and Emergency Department Asthma Visits in New York City, 2000" by Karpati et al. (2004). I am a physician who treats hundreds of patients with chronic illness from chemical overexposure. Many of these patients have toxic encephalopathy, reactive airway disease, and other chemically induced organ system damage. When my patients become ill from pesticide spraying, they usually do not head for an emergency room, where they typically experience long waits in an environment containing germicidal residue, scented products, carbonless copy paper, hospital linens with heavy fabric softener, and other exposures. In addition, they have learned from experience that emergency department personnel often do not understand their condition and do not know how to treat it. Thus your survey, while with admirable intent, greatly underestimates the problem of respiratory exacerbation from West Nile virus pesticide use. Many of my patients have

experienced severe neurologic and respiratory exacerbations as well as other organ system damage, such as significant increase in liver enzymes, from exposure to residue from pesticide spraying for West Nile virus. In addition, it is my understanding that these pesticides are not effective for controlling adult mosquitoes and that the Centers for Disease Control and Prevention and other authorities recommend larvae control. The extent of exacerbation of illness caused by pesticide use for West Nile virus control is likely greater than the number of cases of West Nile virus.

Persons who are at increased risk for symptom exacerbation from pesticide spraying such as that used for West Nile virus control include individuals with migraines, chronic sinus problems, asthma, reactive airway disease, autoimmune diseases (many of which are exacerbated by pesticide exposure), and conventional allergies (Kipen et al. 1994). There is increased respiratory inflammation with conventional allergies, and pesticides more readily enter the body because the barrier function of the respiratory tract is further compromised. In addition, Karpati et al. (2004) failed to take note of the U.S. Environmental Protection Agency (EPA) final report "Principles of Neurotoxicity Risk Assessment" (U.S. EPA 1994). This document confirmed the lack of a blood–brain barrier between the nose and the brain, so that pesticides readily enter the body through the nose and pass directly to the brain. This report further confirmed the unusual vulnerability of the brain to neurotoxicants: pesticides are lipophilic and therefore seek out lipid tissue such as the brain, and because the brain has unusually long neurons, repair of damage in the neurons occurs much less readily than in other body cells."

Other groups at increased risk of pesticides are those with chronic obstructive lung disease, toxic encephalopathy, and neural degenerative diseases. Pyrethroid pesticides are significant neurotoxins (Eells et al. 1992; McDaniel and Moser 1993; Tippe 1993; Vijverberg and van den Bercken 1990), and because they are increasingly replacing organophosphates, they now account for a large proportion of the pesticide-induced chronic illness among my patients. Emergency room visits are merely the tip of the iceberg, and patients with many of these disorders usually avoid the emergency room. Thus, the use of emergency rooms is not a sensitive indicator of body damage from pesticides.

Lothrop HD, Lothrop BB, Gomsi DE and Reisen WK. 2008. Intensive early season adulticide applications decrease arbovirus transmission throughout the Coachella Valley, Riverside County, California. Vector Borne and Zoonotic Diseases. 8: 475-490.

This study describes a spraying strategy from 2006 in California. Mosquitoes worldwide have demonstrated increased resistance since then. This study reported the results of extreme strategy of intense aerial spraying, 26 "treatments" over 40 nights. Still, the overall mosquito kill rate was still only 61%. We cited previously researchers that found in order to achieve effective mosquito control, kill rates needed to be above 90% (reference #183). Furthermore, the authors state that, "Too few dead birds or human cases were detected for meaningful statistical analysis." This study provides weak if any support for SLCMAD's current spraying strategy.

Macedo, PA, Schleier, III JJ, Reed M, Kelley K, Goodman GW, Brown DA and Peterson RKD. 2010. Evaluation of efficacy and human health risk of aerial ultra-low volume applications of pyrethrins and piperonyl butoxide for adult mosquito management in response to West Nile virus

activity in Sacramento County, California. Journal of the American Mosquito Control Association. 26: 57-66.

SLCMAD cites this study as an indication that no human health risk occurs from spraying. This study does not provide any evidence to make such a claim. The study's conclusions are based on modeled, estimated concentrations of exposure, not actual measured external exposures, or internal tissue or blood concentrations. And it uses EPA risk assessments (TRAs), the deficiency of which are thoroughly explained in Appendix A. Furthermore, the model used multiple assumptions that were not correct, such as exposure was limited to 24 hours.

This study too estimated kill rates at 57% and 41% for pathogen carrying mosquitoes, well below the 90% threshold required for effective mosquito control. Furthermore, this study only included kill rates up to 12 hours after spraying, hardly a meaningful time frame in community mosquito control.

In defending pesticide spraying SLCMAD's director has stated that strategies from other cities that successfully do not use pesticides, cannot be extrapolated to SLC because of different topography, weather, wind patterns, surface water, etc. But at the same time SLC uses studies in done in other areas with where are these features are different to provide support that spraying pesticides work. SLCMAD is obviously starkly inconsistent, and cherry picking when they are willing to invoke evidence from other areas.

Peterson RKD, Macedo PA, Davis RS. 2006. A human-health risk assessment for West Nile virus and insecticides used in mosquito management. Environmental Health Perspectives. 114: 366-372.

This 15 year-old study purports to evaluate the health consequences of mosquito pesticide spraying modeled, not actual exposures, and plugging those into traditional TRA methodology, compared to the risks of poor health outcomes from WNV. Again, TRAs do not come close to evaluating the actual consequences of pesticide exposure, nor does it address how or if pesticides may actually decrease the risk of WNV infections.

Reisen, W, Brault AC. 2007. West Nile virus in North America: perspectives on epidemiology and intervention. Pest Management Science. 63: 641-646.

This article has nothing to do with pesticide risk, in fact, if anything supports a no-spray strategy, the authors state, "because of the low prevalence of human infection during epidemics (<3%) and the relatively low rate of clinical illness in infected individuals, vaccination of the US population has not been considered a viable intervention option, even in high-risk areas."

Staples JE, Shankar MB, Sejvar JJ, Meltzer MI, Fischer M. 2014. Initial and long-term costs of patients hospitalized with West Nile virus disease. American Journal of Tropical Medicine and

This review article does not provide any useful information on the issue.

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