CENTRAL Life Sciences®
Central Garden & Pet Company

February 25, 2013

Environment Committee
Connecticut General Assembly
Room 3200, Legislative Office Building
Hartford, CT 06106

Dear Senator Meyer, Representative Gentile, Senator Chapin, Representative Shaban and members of the Environment Committee,

I am writing, as a representative of Central Life Sciences, to express our opposition to HB 6438, which would restrict the use of certain products used for mosquito control in coastal areas of the state. I respectfully request you to vote no in the interest of the public health of Connecticut citizens.

Of particular concern is the restriction of methoprene, the active ingredient in Altosid®, a product manufactured by Central Life Sciences and trusted since it was first registered with the U.S. Environmental Protection Agency (EPA) in 1975 as a valuable tool in mosquito abatement programs. These programs are vital to maintaining and protecting the public from the spread of serious mosquito-borne disease like West Nile virus (WNV) and Eastern Equine Encephalitis (EEE).

The people of Connecticut have entrusted you as members of the General Assembly to use sound judgment and vigor to protect the lives of their children and families. With that charge, I know this committee will scrutinize and examine this issue completely and avoid making decisions based on incomplete studies or speculative anecdotal evidence that could endanger public health.

Last year, I testified against a similar piece of legislation. I said that the cornerstone of public health is prevention. I still stand by those words today. Without a doubt, proactive mosquito control protects the people of Connecticut – and our nation – from deadly diseases like WNV and EEE.

As we examine this issue, it's important look at why prevention is so important. In Texas last year, more than 1,700 residents contracted West Nile virus – 76 of whom died. Dallas County alone accounted for nearly 22 percent of those cases. There's a critical lesson to learn here. Dallas County authorities failed to use any larvicide to control mosquito populations until late July – after it was too late – and only after the public was at the highest possible risk for the disease.[1] Preventive measures like larviciding earlier in the season would have reduced the mosquito population the spread of disease befell the state of Texas last summer.

Since 1999, when WNV entered this country, more than 100 people in Connecticut have gotten sick – 21 last year alone. Three have died. Last year, cases included victims in senate district 12 and house districts 97, 104, 117, 122, and 127 – all represented by members of this committee. And the representation within the General Assembly goes far beyond the walls of your committee room. All but two of the state's cases of West Nile virus occurred in coastal districts. This bill would threaten preventive mosquito control in those areas ***where*** it is needed most, ***when*** it is needed most – in advance of an outbreak.

Last summer, our nation experienced the highest incidence of WNV since 1999. While the number of infections has doubled in a single year in this state, it pales in comparison to the seven and half times increase in human infections (712 in 2011[2]

[1] Friedman, Scott. Sept. 13, 2012. "Dallas Revisits West Nile Virus Attack Plan: Birds not tested for virus; little larvicide purchased prior to epidemic."NBC-5 (Dallas). Available: http://www.nbcdfw.com/investigations/Dallas-Revisits-West-Nile-Virus-Attack-Plan-169256236.html.
[2] Centers for Disease Control. "Final 2011 West Nile virus Human Infections in the United States." Available: http://www.cdc.gov/ncidod/dvbid/westnile/surv&controlCaseCount11_detailed.htm

vs. 5,387 in 2012[3]) and five and a half times increase in deaths (43 in 2011[2] vs. 243 in 2012[3]) nation-wide. Included later in this document, you will find a guidebook that highlights scientific and statistical evidence that supports our conclusion – that the use of methoprene in Connecticut has saved lives, and that its use does not endanger lobsters.
West Nile virus is a vicious, life-altering illness. Survivors will tell you of the devastating disease that left them crippled, unable to walk, with debilitating headaches, in a coma for several months, or even facing their own death. Just from one mosquito bite. Families of victims who lost their battle will tell you about the weight of unexpected loss they have suffered from loved ones that were taken too soon. Just from one mosquito bite. And that notion gets even scarier when you consider that a single female mosquito has the potential to produce more than 50 billion offspring over one season. **One mosquito. One bite. That's all it takes.**

Preventive tools like methoprene are critical to keeping mosquito populations down to prevent massive outbreaks of these kinds of diseases. Larvicides like methoprene, when applied to standing water – the place where mosquitoes tend to breed – stop them from becoming flying, biting adults that spread disease and threaten human lives.

Independent and available science tells us methoprene is one of the least toxic and most effective tools available to control mosquito populations. In fact, it's been used for more than 40 years with no environmental issues. It begs the question – after such long-standing safe and effective use, why here and now is this becoming an issue? As I've said in my previous testimony, at recommended application rates, methoprene can rarely, if ever, be detected in the outflow areas from treated catch basins. It would be like trying to find the tip of a needle in a haystack. Not to mention that once in the environment, methoprene breaks down in sunlight. The argument that methoprene is affecting lobsters just doesn't hold much weight.

Can't we agree that the sanctity of public health and well-being should outweigh an unproven threat to the lobster population in the Long Island Sound, especially when shell disease, water temperature and plastics have been shown by many independent researchers to be far more likely culprits? Repeated, reliable evidence clearly shows that methoprene poses no threat to lobsters.

I hope this committee turns its focus to the real causes of the reduction in the lobster population and stops using Methoprene as a scapegoat. Focusing on the wrong cause will only delay finding the solution. You can look at the attached evidence: a guidebook on methoprene, a study by study review of evidence previously submitted by the lobstermen, and news coverage from last summer showcasing the effects of WNV in Connecticut and in Texas.

Again, thank you for giving us the opportunity to present this information. I look forward to continuing to work with you in the future, and our company stands ready to provide any additional information you might need.
Sincerely,

Mark Newberg
Director, Corporate Affairs and Communications
Central Life Sciences

Enclosures

[3] Centers for Disease Control. "West Nile Virus (WNV) Human Infections Reported to ArboNET, by State, United States, 2012 (as of December 11, 2012)." Available: http://www.cdc.gov/ncidod/dvbid/westnile/surv&controlCaseCount12_detailed.htm

CENTRAL Life Sciences®
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METHOPRENE: A TESTED, TRUSTED TOOL IN PROTECTING PUBLIC HEALTH

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SUMMARY: METHOPRENE AND MOSQUITO CONTROL

More than 40 years ago, Central Life Sciences discovered insect growth regulators (IGR) which paved the way for biorational products, i.e. products derived from a variety of biological sources. These products prevent the development of nuisance and disease-carrying insects — like mosquitoes — before they can cause economic damage and spread dangerous illnesses like West Nile virus and Eastern Equine Encephalitis (EEE). IGRs are the active ingredients in many of the products Central Life Sciences manufactures today.

Altosid®, a larvicide from Central Life Sciences, is commonly used by mosquito abatement districts and public health department officials, and contains methoprene. The methoprene in Altosid stops mosquitoes from becoming flying, breeding, biting adults. Methoprene is target-specific and will not affect fish, waterfowl, mammals or beneficial predatory insects. As a result, vector control professionals can feel comfortable and confident using Altosid in their district's most sensitive areas — and feel even better about the effective, long-term control provided in return. Furthermore, due to the effectiveness and residual larvicidal activity, the use of Altosid reduces adult mosquito populations and the need for wide-spread spraying of products into the environment.

First registered by the Environmental Protection Agency (EPA) in 1975 as a biorational product, in later years, after the EPA reorganized divisional branches, Altosid fell under the jurisdiction of the Biopesticide Division.

Several formulations of Altosid (liquid, pellet or briquette) have been developed for specific mosquito larvicidal applications depending on the particular usage. All of the formulations are designed to maintain a low, yet effective concentration of the active ingredient at the desired treatment site through the sensitive late fourth larval instar, or developmental stage, of the mosquito. Applications of Altosid to larval mosquito habitat through this stage prevent the development of and subsequent emergence of adult mosquitoes.

The EPA classifies methoprene in toxicity categories III and IV, which means slightly toxic to practically nontoxic. The EPA's Methoprene Fact Sheet states that study data indicate "oral, dermal or inhalation exposure to methoprene for an extended duration is not likely to cause adverse health effects in humans."

Extensive research has clarified methoprene's effects on non-target aquatic organisms, including crustacea. Studies of "immature and adult anthropods demonstrate 24- and 48- hour LC_{50} values [that is, the median lethal concentration — the concentration predicted to kill 50 percent of the organism] — is greater than 900 ppb [parts per billion]." Additional studies demonstrate no sensitivity of larvae to methoprene at concentrations up to 1,000 ppb. These concentrations are approximately 500 times higher than the concentration of methoprene (0.2 ppb) needed to control mosquitoes.

The EPA concluded in its review that "all the environmental fate data requirements for methoprene have been satisfied." Furthermore, methoprene degrades rapidly in water, is metabolized rapidly in soil, and does not leach; therefore it "is not expected to persist in soil or contaminate ground water."

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SECTION I: METHOPRENE IS TRUSTED AND HAS BEEN TESTED

Many laboratory and field studies have been performed to study impact of methoprene on the environment. Studies consistently demonstrate that methoprene is either undetectable or present at extremely low concentrations when used at recommended application levels.

Since it was first registered with the EPA in 1975, and reviewed again in 1992, methoprene has been repeatedly tested for safety and effectiveness under EPA guidelines, which includes testing on a number of crustaceans.

METHOPRENE IS TARGET SPECIFIC TO MOSQUITOES AND A FEW OTHER INSECTS

The favorable environmental properties of methoprene have been established by the work of many different research groups. If used as recommended, there is little to no effect on non-target species like mammals, fish, crustaceans, birds, protozoa, annelids, mollusks, amphibian, damselflies, mayflies, water beetles or waterfowl.

The toxicity of methoprene to mammals has been studied extensively. The mammalian LD_{50}, or median lethal dose, for methoprene is greater than 34,560 mg/kg body weight. Methoprene does not cause gene mutations or cancer. It does not cause birth defects and thus, is considered a less toxic approach for insect control. Methoprene and other IGRs are used to control pests in stored grain, fleas on companion animals, flies in cattle production and other areas where insects can spread disease and cause economic damage.

Methoprene is exempted from the requirement of food tolerances by the EPA; therefore it may be applied to all raw agricultural commodities for human consumption. It is also approved by the World Health Organization for application to drinking water where mosquitoes may breed and spread disease.

METHOPRENE DOES ITS JOB THEN GOES AWAY

Methoprene does not persist in the environment after use and is subject to degradation from UV light and metabolism in plants, animals, aquatic microorganisms and soil microbes[4]. It is stable in water for only a few hours and in cases of heavy microbe populations or full sun exposure, the persistence is further reduced.

This lack of persistence is one reason that slow release formulations were developed to provide the methoprene concentration necessary to control mosquitoes. As methoprene degrades, the slow release formulations provide a continuous source of active ingredient at the desired effective rate into the water column until the formulation is exhausted. Even in these slow-release formulations, methoprene does not bioaccumulate in fish and does not persist in the environment.

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[4] Schaeffer and Dupras Jr. 1973, Quistad et al. 1974a, 1974b, 1975a, 1975b, 1975c, 1975d, 1976a, 1976b; Chamberlain et al. 1975, Schooley et al. 1975a, 1975b; Schooley and Quistad 1979.

SECTION II: CLAIMS AGAINST METHOPRENE HAVE BEEN WIDELY DISPROVED

CLAIM: Methoprene is highly toxic to lobsters at low concentrations.
TRUTH: Methoprene is not toxic to lobsters at concentrations found in waters surrounding treated areas. Conflicting data have been reported on the toxicity of methoprene to lobsters.

- In the *Journal of ShellfishResearch*, Zulkosky et al reported that methoprene at 10 ppb caused no toxicity in Stage I-II larval lobsters.[5]

- Hans Laufer, as detailed in a speech at the 2003 Long Island Sound Lobster Health Symposium, said that 1 ppb, 10 ppb and 100 ppb concentrations of methoprene did not delay stage transitions in Stages I-III, though higher concentrations did.[6]

- Walker et al reported in the *Journal of Shellfish Research* "that Stage IV larvae exposed to methoprene concentrations of 50 ppb experienced a greater than 90 percent mortality rate after three days." [7] A similar study found that methoprene was toxic to State II larvae at 1 ppb and Stage IV larvae at 5 ppb. In context, the recommended rate of application for methoprene in vector control programs is 0.2 ppb.

CLAIM: Maine does not use methoprene and they have abundant lobster populations.
TRUTH: According to Maine's Supervisor of Mosquito and Tick Control, both larvicides methoprene and BTI are used to control mosquitoes in standing water in Maine. Additionally, there is no scientific basis for any connection between Maine's use of methoprene and their abundant lobster population, which can be linked to various environmental factors, including water temperature.

- In discussions with the Maine's Supervisor of Mosquito and Tick Control, both larvicides methoprene and Bti are used to control mosquitoes in standing water. Their use is restricted to contained waters that do not move into streams or creeks to avoid movement of chemicals from one private property to another.

- Environmental conditions in Maine make waters much more hospitable to lobsters. Water temperature is much colder north of Nantucket Sound. Robert Glenn, a senior marine fisheries biologist with the state Division of Marine Fisheries said in a 2010 article in the *Cape Cod Times,* that "Although temperature increases have been noted in Boston and in Maine, the water is so much colder north of Nantucket Sound that it would take a huge increase in the average temperature, of 15 degrees or more, to reach levels that lobsters can't tolerate."[8]

CLAIM: Mosquitoes are just like lobsters and methoprene will cause mortality in the same way.
TRUTH: If mosquitoes and lobsters are alike, then blue crabs (another related species) would experience the same problems in the presence of methoprene. They do not.

- In "*Estuary News*" (Vol. 16, No. 3, Spring, 2006), a publication of the EPA's *Delaware Bay National Estuary Program,* there appeared an article titled "*Protecting Horseshoe Crab and Human Health*" written by David Bushek of Rutgers University's Shellfish Research Laboratory. In the subsection boldly titled "Impact of Mosquito Control," it states that horseshoe crabs could be susceptible to adverse impacts from contaminants, and that "one contaminant of concern is the mosquito larvicide known as methoprene." The supposition was that methoprene prevents mosquito larvae from molting, and since horseshoe crabs are arthropods too, then according to the

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[5] Zulkosky, A. et al. "Acute toxicity of resmiethrin, malathion and methoprene to larval and juvenile American lobsters (Homarus americanus) and analysis of pesticide levels in surface waters for Scourge, Anvil and Altosid application.Journal of Shelfish Research 24.3 (October 2005).
[6] Laufer, Hans, et al. "Hormonal responses of lobsters to stress, an Interim Report" Third Long Island Sound Lobster Health Symposium Program: 32-37.
[7] Walker, Anna, et al. "Metabolic effects of acute exposure to methoprene in the American lobster, Homarus americanus." Journal of Shellfish Research 24.3 (October 2005).
[8] Fraser, Doug. Cape lobster industry faces crisis (June 13, 2010) Cape Cod Times. http://www.capecodonline.com/apps/pbcs.dll/article?AID=/20100613/NEWS/6130340. Retrieved 02.27.12.

researchers' wisdom it would seem that methoprene might possibly contribute to the Bay's declining horseshoe crab population. The researchers exposed developing and newly hatched larvae of horseshoe crabs to "environmentally relevant levels of methoprene." They reported, "After following development and survival through the first larval molt, we were unable to detect any macroscopic effects. That is the eggs and larvae appeared to develop normally, even at levels well above those applied to control mosquitoes along Delaware Bay."

CLAIM: Methoprene flows out of catch basins and into lobster-bearing waters.
TRUTH: Studies have consistently shown that methoprene, applied at recommended rates, is not flushed into natural waters in detectable amounts.

- Butler and Gettman investigated methoprene concentrations from the application of Altosid pellets into catch basins. The methodology included placing a known amount of pellets (3.5gm) into catch basins and subsequently determining the level of methoprene by chemical analysis. Three sampling points were used; in catch basins prior to flooding, at the outfall of the catch basins into Point Judith Pond, and 10 feet away from the outfall following flooding. A mesh trap was placed over the outfall pipe to determine if any of the pellets had washed out. Multiple replicate water samples were sent to two different laboratories for independent analysis.

  "Our results suggest that methoprene applied to catch basins in 30-day slow release pellets is not flushed into natural waters in detectable amounts (≥ 0.2 ppb). Furthermore, our results suggest that the use of methoprene is not likely to affect non-target organisms in natural environments when used according to label instructions. Since methoprene concentrations were below detectable levels in Point Judith Pond, our study area, it is extremely unlikely that it would be found in larger bodies of water such as Long Island Sound or Narragansett Bay at levels anywhere near detection. Our data show that the only organisms likely to be exposed to methoprene at detectable levels (≥ 0.2 ppb) are those that share catch basin environments with mosquito larvae."[9]

- In October 2009 and June 2010, the Rhode Island Department of Environmental Management performed additional studies to monitor the waters downstream from catch basins for the presence of methoprene. The 2009 study addressed Altosid levels (in ppb) from samples taken at an outfall following treatment and flushing of a series of 13 connected catch basins. None of the 11 samples yielded detectable levels of Altosid. These results are, again, indicative of the dilution factor, even though the experiment was designed to maximize the potential for acquiring detectable levels. The following year, in June 2010, an experiment addressed Altosid levels (in ppb) from samples taken downstream from an application in a tidal creek. Thirty-five grams of pellets (representing the amount in 10 catch basins) were placed in a mesh pouch and secured at the bottom of a creek. One week later, during a gradual tidal flow, samples were taken adjacent to the pouch and at distances of 10, 20 and 30 feet downstream. None of the 12 samples yielded detectable levels of Altosid, again, indicative of the dilution factor.

- In a study conducted by Environment Canada in the Hamilton Ontario area, waters were sampled in areas where methoprene was actively used. Data shows that a low level of methoprene was seen in some samples closest to the treatment areas, but quickly disappears as it gets into streams and other bodies of water. Water samples were taken from inside the catch basins, and in only 50 percent of the basins were detections confirmed. The highest detection level was only sample at 4.35 ppb in a catch basin and the remaining detections were 0.65 ppb and lower. Receiving water samples from the catch basins were also collected and, generally, were determined to be below the concentrations of ecological concern as set by the interim Ontario provincial water quality objective (0.2 ppb). No methoprene was detected in Hamilton Bay.[10] Similarly, Fletcher, et al., did not detect methoprene in the Ontario receiving waters they tested.[11]

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[9] Butler, Ginsberg, LeBrun, Gettman. Evaluation of non-target effects of methoprene applied to catch basins for mosquito control. (August 2010) *Journal of Vector Ecology*.
[10] Struger, John, et al. "Occurrence and Fate of Methoprene Compounds in Urban Areas of Southern Ontario, Canada." *Bulletin of Environmental Contamination and Toxicology* 79.2 (2007): 168-171.
[11] Fletcher, R., et al. "The Widespread use of Methoprene for the Prevention and Control of West Nile Virus in Ontario, Canada: Is it Impairing our Streams?" American Geophysical Union 86.18 (2005).

- Des Lauriers and colleagues concluded that rainfall flushed methoprene from the studied Toronto catch basins (the methoprene application sites) to the storm sewer outfall and into the receiving waters at concentrations lower than the level that may cause ecosystem damage.[12]

- The U.S. Geological Survey and U.S. Department of the Interior monitored surface waters in Suffolk County NY over a three-year period for methoprene. Methoprene in excess of 1 ppb was found in a single sample at 9 ppb measured within an hour of application. No methoprene was found in four of the seven sites treated with methoprene.

- In a Washington State Department of Ecology study, methoprene was only detected in 6 percent (4 of 68 total) of the Grant County, Washington surface water samples Johnson and Kinney collected directly after methoprene treatment. Concentrations of methoprene ranged from 0.1 ppb to 0.6 ppb.[13]

- In response to discovery of West Nile Virus Beaufort County, SC, a costal county with more water acreage than land, initiated a treatment regime. In response to environmental concerns about the harm to shrimp, crabs and oysters, the Beaufort County Mosquito Control initiated a study to determine the potential for catch basin treatment to result in methoprene concentrations sufficient to disrupt the development of non-target species. Methoprene was found in 4 of 65 post-treatment water samples. The maximum concentration found was 0.285 ppb. The researchers concluded, "Even with conditions believed to represent a worse-case scenario, catch basin treatment … did not result in methoprene concentrations sufficient to adversely impact non-target species. Further as methoprene degrades rapidly in the environment, accumulation over time to toxic concentrations is unlikely."[14]

CLAIM: Methoprene causes deformities in frogs.
TRUTH: Most of the concern adding fuel to this claim came from studies conducted with extremely high doses of methoprene (in some cases, 15,000 times the maximum recommended application rate).

- Methoprene was investigated as a possible cause for the reported increase in amphibian deformities. Most of the concern was generated by a study (La Clair et al. 1998) that reported that methoprene and its degradation products cause deformities in Xenopus laevis (frog embryo) . However, not only were the studies conducted with extremely high doses of methoprene (ca. 15,000 times the maximum recommended application rate of Altosid) that had little, if any, relevance to methoprene in the environment, but the findings could not be replicated by other researchers. [15, 16]

- The extensive environmental studies that had already been carried out with methoprene, including specific studies on anurans (frogs)17 indicated that it was unlikely that methoprene or any of its degradation products were involved in the high rates of deformities observed.

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[12] Des Lauriers, Angelune, et al. "A field study of the use of methoprene for West Nile Virus mosquito control." Journal of Environmental Engineering and Science 5.6 (2006): 517-527.
[13] Johnson and Kinney. "Methoprene Concentrations in Surface Water Samples from Grant County Mosquito Control District No. 1." Washington State Department of Ecology Publication 06-03-001: (2006).
[14] Warren, D.A., et al. Assessment of Methoprene in Marine Waters after Catch Basin Treatment with Altosid XR Briquets.
[15] LaClair JJ, Bantle JA, Dumont J. Photoproducts and metabolites of a common insect growth regulator product developmental deformities in Xenopus Environ Sci Technol 32, 1453-1461 (1998)
[16] Degitz, S.J. et al. Devemopmental toxicity of methoprene and several degradation products in Xenopus laevis. Aquatic Toxicology 64. 2003
[17] Miura and Takahashi 1973, Simonin et al. 1992, U.S. Environmental Protection Agency 2001.

- A study conducted by the EPA, (Degitz et al. 2003) evaluated the aqueous stability and developmental toxicity of methoprene and its degradation products in X. laevis embryos (FETAX). They found that methoprene exposure did not result in developmental toxicity at concentrations up to 2 ppb. They concluded that methoprene and its degradation products are not potent developmental toxicants in X. laevis and are not a factor in the occurrence of deformities in the environment.

- Ankley et al. (1998), working for the EPA, conducted a study to evaluate the effects of both methoprene and ultraviolet light (UV) on the northern leopard frog (Rana pipiens) embryos. They found that UV light did cause limb abnormalities, whereas in the groups treated with methoprene only (up to 125 ppb), no increased mortality or developmental abnormalities were detected.

- In another study, Henrick et al. (2002) carried out a careful evaluation to address these concerns. Chemical analyses of water samples were carried out which demonstrated that methoprene and its degradation products could not be detected in the Minnesota water bodies where frog deformities were observed. A comparison of the use of Altosid in different counties in Minnesota with reported frog deformities showed no correlation between frog deformities and use of methoprene. This study also concluded that methoprene has negligible or no effect on anurans even at 100 times the maximum recommended field rate.

CLAIM: Methoprene was involved in the initial lobster die-off in Long Island Sound in 1999.
TRUTH: Methoprene was not used in the New York City mosquito program in 1999 since the effort that year focused on use of adulticidal controls. Altosid was not introduced in New York City until 2000.

- In 1999, West Nile virus was affecting the New York City area and as a result, the City of New York organized a concentrated mosquito control effort. This initial mosquito control effort was centered on the use of insecticides for adult mosquitoes. It was not until the following year, 2000, that Altosid was used in the New York City mosquito control program.

- A 2005 study in the *Journal of Shellfish Research* estimated the concentration of methoprene in Long Island Sound in 1999 based on the actual amount of methoprene used in the area. The model predicted extremely low levels, 0.0005 ppb methoprene, in the Sound. In this model, *all* of the methoprene applied in the catch basins of the watershed was predicted to reached the waters of Long Island Sound with no attenuation or decay — a highly unlikely scenario in practice. They concluded "the Phase I run for methoprene showed that even if all of the pesticide applied reached the water and never decayed, the concentration would not have been lethal to either larval or adult lobsters". [18]

- The surface waters of Long Island Sound were actually monitored in 2003 when methoprene was actively used in storm drains surrounding the Western Sound. No methoprene was detected. [19]

CLAIM: In 1999, lobsters were killed by mosquito pesticides; other factors had no effect.
TRUTH: A 2005 study published in the *Journal of Shellfish Research* points to a combination of factors that upset the balance of the lobster population in the Long Island Sound, including water temperature, disease, and increasingly hostile environmental conditions.

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[18] Landeck Miller, Robin, et al. "Application of water quality modeling technology to investigate the mortality of lobsters (Homarus americanus) in western Long Island Sound during the summer of 1999." *Journal of Shellfish Research* 24.3 (October 2005).
[19] Abbene, I.J. et al. Concentrations of Insecticides in Selected Surface Water Bodies in Suffolk County, New Your, Before and After Mosquito Spraying, 2002-04.U.S. Department of Interior US Geological Survey Open-File Report 2005-1384.

- "There is overwhelming evidence that in the absence of any pesticide application, a confluence and succession of factors pushed the balance of the WLIS lobster population far out of equilibrium with its environment in 1999. Faced with disease (paramoebae), and subjected to sustained and increasingly hostile environmental conditions, the immune systems of the lobsters were unable to compensate, and many lobsters succumbed. While the main mortality event occurred in 1999, the problem may have been building for several prior years.

- Sustained, above average water temperature was a driving force behind the snowball effect of environmental and oceanographic factors that stressed lobsters to the point at which their physiology and immune systems could not cope with these sustained and increasingly lethal environmental conditions in conjunction with, or in addition to, mounting an immune defense against the parasitic amoebae. Based on the body of evidence, the scenario leading to the 1999 mortality event is proposed as follows:

    - Scattered reports of atypical lobster mortality began as early as 1997, however , it is unknown when paramoebiasis began to affect the lobster population.

    - In 1999, water temperature remained several degrees warmer than average for a duration of many months. For much of the summer and continuing into early fall, bottom water temperature remained at or above the temperature threshold that, in lobsters, induces a significantly enhanced respiration rate. The warm waters also likely accelerated the growth of natural populations of Neoparamoeba pemaquidensis in LIS waters.

    - Lobsters in western LIS concentrated in numbers as they moved to deeper waters to escape warm shallower waters and moved away from hypoxia-affected areas towards those with slightly higher DO concentrations. This displacement, exacerbated by the abundant lobster population, caused crowding that, in the presence of less-than-optimal conditions, increased susceptibility to disease.

    - In late August, winds from a front moving through the region completely mixed the water column in a matter of hours, brought warm surface waters to the bottom and raised warm temperatures several more degrees. While lobsters typically can endure abrupt changes in temperature, these lobsters were already in a weakened physiological state.

    - Pesticides were being applied to combat the spread of WNV by mosquitos. Exposure of some lobsters in near-coastal waters to these pesticides could have resulted in sublethal effects, which may have further weakened those lobsters.

    - As summer hypoxic conditions dissipated, low dissolved oxygen at the water sediment interface coupled with high water temperatures facilitated the release of sulfides and ammonium from the sediments, conditions shown to lead to a significant increase in the rate of mortality.

    - Affliction with paramoebiasis sealed the fate of these lobsters. Either the energy used in mounting a sustained immune response to the disease took its toll on the lobsters, or the lobsters were unable to mount an adequate response as their immune systems were potentially compromised by the stressful conditions presented by this "perfect storm" of lethal synergistic effects. Mortality became rampant and the commercial lobster fishery in western LIS largely collapsed."[20]

[20] Pearce, Jack; Balcom, Nancy. The 1999 Long Island Sound Lobster mortality event: findings of the comprehensive research initiative. (2005, October 1). *Journal of Shellfish Research.*

CLAIM: Methoprene hasn't been studied in the Long Island Sound environment.
TRUTH: Multiple studies were conducted in the Long Island Sound in 2005 to examine the effects of methoprene on juvenile lobsters.

- Studies by Zulkosky et al (2005) [21] and Miller et al (2005) [22] evaluated pesticide levels in the Long Island Sound following mosquito applications. Zulkosky evaluated effects of methoprene and adulticides on stage one and two larvae and juvenile lobsters. They also evaluated surface water concentrations following aerial applications of mosquito control products. Methoprene was found to have no effect on lobster larvae and juveniles at the highest dose tested of 10 ppb.

CLAIM: The problems with lobster populations in Rhode Island were related to methoprene.
TRUTH: Detailed analysis of abundance and mortality rate for several life stages of lobster in the Rhode Island area failed to reveal any evidence that the methoprene used in mosquito control programs has had any impact on the local lobster population.

- Methoprene is a growth disruptor for invertebrates that grow through molting and metamorphosis. It has the potential to kill and alter molting of larval lobster and may interfere with chitin deposition in shells of juveniles and adults (Walker 2005). The capable concentrations however are far above that likely to occur near the outfall of treated storm drains (Butler 2005). Moreover, the relatively short half-life of methoprene in water (Schaefer and Dupras 1973) and the enormous dilution factors operating on spatial scales of the fishery make it extremely unlikely that adverse impacts have occurred. In Black Point Narragansett, there was no evidence that abundance of settler, juvenile or adult lobster was lower during years of methoprene usage (2000-2007) than years prior to usage (1990-1999).

- Lobsters in southern New England have experienced an epizootic outbreak of bacterial shell disease. It is likely that mortality rate in lobster has been increased by the disease (Gibson and Wahle 2005, Dominion 2008, ASMFC 2006). The outbreak however began in 1996, accelerated in 1997, and was well underway by 1999, before any methoprene treatments began in 2000. It is highly unlikely that methoprene had anything to do with a disease that still affects over 20 percent of the animals in the area. That is not to say that shell disease has not impacted the lobster population. It has likely killed some animals outright, induced abortive molts in some egg bearing females and reduced growth rate in other animals. Still, with tens of thousands of animals examined, the number dying remains relatively low at about 3 percent (Dominion 2008) and abortive molts are very rare and show no pattern with regard to methoprene usage (Figure 10). Continued study of the disease is warranted but it does not appear that methoprene is a strong candidate for new research. Altogether, the results do not support a methoprene impact hypothesis. Methoprene was similarly indicted in Long Island Sound as cause for a lobster die-off but ultimately no evidence was found and increasing water temperatures were deemed more likely responsible (Dove et al. 2005).

- It is clear that the collapse of the Rhode Island inshore lobster stock and fishery in 2002-2004 and continued low abundance is not due to methoprene application. It occurred as a result of a "perfect storm" of failed settlement by the 1995-1996 cohorts, an acute mortality event (1996 oil spill) that winnowed extant juvenile populations, chronic shell disease and overfishing (truncated age structure). The first factor was unavoidable, fishery managers cannot control long-term forcing by climatic events. The second event, while technically avoidable, was a result of human error that happens from time to time. The magnitude of shell disease as a mortality agent has yet to be fully understood but is believed low. Overfishing in the lobster fishery is given short thrift among all the factors

[21] Zulkosky, A. et al. "Acute toxicity of resmiethrin, malathion and methoprene to larval and juvenile American lobsters (Homarus americanus) and analysis of pesticide levels in surface waters for Scourge, Anvil and Altosid application.Journal of Shellfish Research 24.3 (October 2005).
[22] Miller, Robin Landeck, et al. "Application of water quality modeling technology to investigate the mortality of lobsters (Homarus americanus) in western Long Island Sound during the summer of 1999." *Journal of Shellfish Research* 24.3 (October 2005).

that can be blamed. Despite the warnings of three peer-reviewed stock assessments (ASMFC 1996, 2002, 2006) it is still not appreciated how vulnerable a recruitment based fishery is.[23]

- An astonishing amount of methoprene would have to be applied in Narragansett Bay in Rhode Island to cause an effect in the lobster population. Narragansett would take an extraordinary quantity of methoprene applied to a watershed's storm sewer catch basins to reach harmful levels of the biopesticide in receiving waters. For example, the lobster harvest area in Rhode Island is approximately 87 square miles and would require more than 4400 metric tons of methoprene to be applied (assuming a water depth of only three feet). However, in 2007, only approximately 1.2 metric tons were sold in Rhode Island.[24]

CLAIM: It would be better to abandon the use of methoprene and just kill adult mosquitoes.
TRUTH: The opinion of the Suffolk County, New York superintendent responsible for mosquito control states, "By controlling mosquitoes in the larval stage, the use of methoprene products in Suffolk County prevents infestations that could adversely impact people while not exposing residents to pesticides.
Preventing these infestations means there is less need for the use of the more toxic materials, adulticides, used to control adult mosquitoes. Not only are adulticides far more toxic to lobsters than methoprene, but their use can result in the exposure of County residents to these pesticides. Based on historic data, adulticiding by the County could increase 10-fold to achieve the level of control now achieved through larviciding with methoprene. In addition, pesticide applications by homeowners and other private interests could be expected to increase and these applications would involve doses 64x those used by Vector Control. This bill would eliminate a negligible risk to lobsters at the cost of increasing mosquito infestations, increasing the use of demonstrably more toxic materials and increasing the exposure of human beings to pesticides."

CLAIM: It would be better to only use Bti and avoid methoprene altogether.
TRUTH: In the Suffolk County Environmental Impact Study it states, one reason for the County to use multiple larvicide products is to allow for resistance management. The County tends to alternate between Bti and methoprene in salt marshes. Bti is effective on the younger growth stages of mosquito larvae, it is an ingestion toxicant meaning that larvae must eat it to receive a toxic dose. It provides another tool for mosquito control professionals to use but it cannot be relied on as a total larvicidal control method, especially in permanent water sources. Bti is susceptible to UV light degradation and competing food sources. It is less effective in higher organic matter environments and will only be effective for 48-72 hours in the water. As the season progresses, higher organics will develop in mosquito habitats which will lessen the effectiveness of the product. In situations with synchronous broods of mosquitoes, applications of Bti will be ineffective if applied to later stage mosquito larvae since these larvae do not feed.

Methoprene prevents larvae from developing, and is a contact pesticide and is effective on late stages. Since it works on the late stages, the younger mosquito larvae are left in the water column as a food source for beneficial species. Suffolk County records indicate large improvements in larval control effectiveness when methoprene was introduced in 1995.

CLAIM: Methoprene use endangers other organisms.
TRUTH: "Methoprene degrades rapidly in water so the use of most formulations in estuaries is generally not of concern. However, concern has in fact been raised in recent years with respect to methoprene's potential impact on shrimp, crabs and lobsters. These concerns stem from the fact that a shared evolutionary past, as well as resultant similarities in biology, exist between crustaceans and dipteran species (including mosquitoes).

[23] Gibson, Mark. Lobster Settlement and Abundance in Rhode Island: An Evaluation of Methoprene Application and Other Factors Potentially Influencing Early Survival. (June 2008) RI Department of Environmental Management, Division of Fish and Wildlife
[24] Based on data from "Rhode Island Commercial Fishing Areas" map produced by the University of Rhode Island Environmental Data Center, <http://www.edc.uri.edu/fish/imagemaps.html> (accessed June 16, 2008).

Studies have indicated environmental and not chemical causes. Most of the recent studies of estuarine invertebrates have used shrimp, Atlantic oysters, amphipods, copepods and mud crab. In general, impacts to these species are not anticipated to occur at expected environmental concentrations. The Caged Fish experiment found no impact to exposed fish and shrimp, for example. The risk analysis found no risks for impact to ecosystems because exposures were much less than the levels required to cause impacts to organisms. The lowest concentrations found to cause impacts to lobsters, when the exposures were sustained for days, were only recorded immediately after applications in local sampling.

This reinforces the conceptual understanding that the effectiveness of methoprene on mosquito larvae occurs at concentrations below those necessary to cause significant non-target organisms effects.
Methoprene has been used by Suffolk County Vector Control SCVC since 1995, and is particularly useful in the salt marsh, where Bti is not always effective."[25]

CLAIM: Some scientists' work shows that methoprene is harmful to lobsters.
TRUTH: The primary concern with the use of methoprene (in salt marshes) appears to revolve around the work in a single lobster study.

Dr. Michael Horst's 1999 research with crab larvae used concentrations up to 500 times higher than those levels present in real-world vector control applications. Horst, Walker, et al. more recent work in 2005 with lobster larvae suggested that there was increased mortality utilizing concentrations of 1 to 2 ppb continuously during a 72 hour exposure. (These results were not confirmed in concurrent Stony Brook University analyses). In any case, 1 ppb methoprene for 72 hours is an extremely unrealistic exposure. The Caged Fish Study, conducted as part of the Suffolk County, New York's Long-Term Plan, clearly demonstrated that concentrations of methoprene rapidly decrease to 0.005 ppb within two hours of application.

Methoprene is applied in wetland areas, not where larval crabs and lobsters are found. Blue claw crabs hatch offshore and only arrive in estuaries when they are close to being fully developed. Lobsters hatch offshore, develop offshore and live offshore. A modeling exercise, made to estimate the maximum amount of pesticides that could have been in Long Island Sound when the 1999 lobster die-off occurred, found the maximum amount of methoprene that could be present in the near offshore waters of the Sound was measured in the parts per quadrillion, and the lowest concentration linked to effects are in the parts per billion.

Studies conducted by Anne McElroy, PhD, Associate Professor and Graduate Program Director at the Marine Sciences Research Center at Stony Brook University, did not find methoprene lethal to larval lobsters at concentrations up to 10 ppb. In addition, she collected water from surface water bodies following vector control spraying by Suffolk County, New York and did not find the concentrations of methoprene to be acutely lethal in controlled laboratory setting. These studies by McElroy's laboratory are consistent with other published studies, which contradict the results of Horst's work.

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SECTION III: OTHER SUGGESTIONS FOR LOBSTER DECLINE IN NORTHEAST STATES

HOSTILE ENVIRONMENTAL CONDITIONS

"A spokeswoman for the New York Department of Environmental Conservation, Aphrodite Montalvo, said research shows the lobsters had been stressed by hostile environmental conditions and that individual pesticides were not enough to cause the die-off. She said the agency has upgraded sewage treatment plants and taken other steps toward improving water quality."

Conn. lobstermen seek other work as stocks dwindle. (2011, October 29). *Associated Press.*

PLASTICS

Researchers at the University of Connecticut have discovered waterborne chemicals leached from plastics and detergents, may contribute to the decline in population and to shell disease in lobsters in the Sound. Chemicals such as bisphenol A can slow the lobster's molting pattern and interfere with regular development.

Buckley, Christine. Lobster Dieoffs Linked to Chemicals in Plastics. (2010, August 10). *UConn Today Blog.*

"Hans Laufer, a research professor in the Department of Molecular and Cellular Biology in the College of Liberal Arts and Sciences, has found that by interfering with hormones crucial to young lobster growth, chemicals such as bisphenol A can slow the lobsters' molting patterns and interfere with regular development, leading to body deformations, susceptibility to disease and potential death."

Buckley, Christine. Lobster Dieoffs Linked to Chemicals in Plastics. (2010, August 10). *UConn Today Blog.*

"Hans Laufer, a professor at the University of Connecticut, has spent the last four years investigating the link between the plastic by-products, called alkyphenols and shell rot. In recent years, the disease has become an epidemic in Long Island Sound (located between Connecticut and Long Island, N.Y.) affecting up to 70 per cent of some lobster populations at its peak. 'There seems to be a direct relationship between plastic compound breakdown and shell disease,' Laufer said. 'The lobsters try to molt out of the old shell,' Laufer said. 'If it's just mild enough, they recover. If it's serious, of course, it kills them.'"

Manzocco, Natalia. Steps taken to protect lobster; Fishing Group starts education program to warn of dangers of plastic in Bay of Fundy (2008, June 3) *The Telegraph-Journal.*

CLIMATE CHANGE/WATER TEMPERATURE/CALCINOSIS/SHELL DISEASE

"If Long Island Sound is becoming inhospitable for lobsters or other animals because water temperature is too high, that means they're not going to stay there and there's nothing anybody is going to do about it,'' said Gordon Colvin, the director of marine resources for the New York State Department of Environmental Conservation."

Johnson, Kirk. Warming Waters and Dying Lobsters; Scientist Links Climate to Population's Decline in the Sound. (2002, November 9) *The New York Times.*

''The correlation is very strong,'' [Dr. Alistair D. M. Dove] said. ''Not proven, but strong. Climate is the killer here.''

Johnson, Kirk. Warming Waters and Dying Lobsters; Scientist Links Climate to Population's Decline in the Sound. (2002, November 9) *The New York Times.*

''There are researchers who are sure it was pesticide – and the lobster fishermen are sure – but personally I now think there's very little chance,'' said Jack Mattice, the director of the New York Sea Grant Institute, which is helping oversee the lobster research. ''I believe that it was primarily temperature, and I think most people would probably say that temperature was a direct or indirect cause.''
Johnson, Kirk. Warming Waters and Dying Lobsters; Scientist Links Climate to Population's Decline in the Sound. (2002, November 9) *The New York Times.*

"Two summers ago, yet another lobster disease turned up. At first, it seemed just to add to the confusion. Orange grit was clogging and sometimes petrifying the gills of lobsters around eastern Long Island Sound. Under study, it proved to be deposited calcium, not unlike kidney stones in humans. It was called calcinosis. Then, its discoverer had a eureka moment. Alistair Dove, at the State University of New York-Stony Brook, got to thinking about what could drive a lobster's metabolism and, by extension, cause such a metabolic disease. 'That was the first time we thought of temperature,' he says."
Donn, Jeff. Northeast lobster decline tied to warming. (2004, August 24). *Associated Press.*

"Other scientists showed higher temperatures strain lobsters and may make them more susceptible to infection and pollutants. Maybe too much warmth is weakening their immunity to disease while making harmful microbes flourish, they reasoned. It could be luring warm-water predators into the Northeast and chasing cold-water ones from Maine. It could explain much of what lobstermen have encountered."
Donn, Jeff. Northeast lobster decline tied to warming. (2004, August 24). *Associated Press.*

"Two scientific reports have shown that warming waters in the western Sound may have seriously contributed to the die-off. One report released this summer and associated with the Washington-based Union of Concerned Scientists (UCS) stated that "although a number of factors played a role in this die-off, warmer water temperatures seem to have set the stage."
Failoa, Anthony. What's Killing the Lobsters Of Long Island Sound? (2007, October 7) *The Washington Post*.

"What we found was that the concentrations of pesticides in the water could not have been high enough to be lethal to lobsters," said Sylvain De Guise, director of Connecticut's Sea Grant program and a lead researcher on a major 2005 study of the die-off published in the Journal of Shellfish Research. "Instead, we're probably looking at a combination of factors." He continued: "What you can say is that the western Sound is at the southernmost range for [coastal] lobsters, and it's very likely that the impact of warming waters would be seen here first. I'd have to say that global warming, based on common sense, is the strongest argument.""
Failoa, Anthony. What's Killing the Lobsters Of Long Island Sound? (2007, October 7) *The Washington Post*.

"'(The lobster decline) is a combination of factors that are all related back to changes in water temperature,' said Robert Glenn, a senior marine fisheries biologist with the state Division of Marine Fisheries.

Glenn said the expansion in the number of days with high water temperatures is probably a bigger factor than the temperature rise itself. While lobsters can tolerate the occasional warm day, prolonged exposure to water over 68 degrees wreaks havoc on their respiratory and immune systems and leads to outbreaks of shell disease and other lobster diseases."
Fraser, Doug. Cape lobster industry faces crisis (2010, June 13) *Cape Cod Times.*

"Last year, a federal effort to coordinate research, the U.S. Global Change Research Program, found ocean warming already was forcing a migration of some species.

"The northward shifts we have seen in the area are due in part to climate change. We are starting to see some of the effects of global climate change in our area," said Janet Nye, a NOAA researcher working out of Woods Hole, Mass. She studied historical fish records and found that of 36 northwest Atlantic species, almost half had moved northward in 40 years as water temperatures warmed."
Struck, Doug. "Warming Waters Exacerbate Dwindling New England Fisheries." (2010, July 13). *Scientific American.*

"It is not possible to draw a direct relationship between the decline of the Southern New England lobster stock and increased water temperatures. However, the strong coincidence in the timing of the increase in water temperature with the timing of the decline in landings, spawning stock biomass and recruitment, coupled with overwhelming experimental evidence of increased physiological stress, immunosupression and increased rates of disease in lobster exposed to prolonged periods of temperatures ≥ 20 °C, strongly suggest that increasing water temperatures have played a primary role."
American Lobster Technical Committee Atlantic States Marine Fisheries Commission. "Recruitment Failure in The Southern New England Lobster Stock." (2010, April 17) *Accessed via Connecticut Department of Energy Environmental Protection website*.

"In August 1999, the temperature differential between surface and bottom water temperatures in the Long Island Sound was as much as 5 [degrees]C and both nearshore waters and deep bottom waters continued to warm (CTDEP 1999, Wilson & Swanson 2005). On August 29th, the effects of Hurricane Dennis to the south of Long Island Sound, coupled with strong winds associated with a fast moving cold front from the north that passed through the LIS region, caused a complete vertical mixing of the water column, raising bottom water temperature several degrees to >22[degrees]C (Wilson et al. 2004, Wilson & Swanson 2005).

Following the passage of this front, there was, according to temperature and salinity data, limited restratification until September 16th, when Tropical Storm Floyd passed through the region, resulting in a substantial rainfall event and causing additional mixing of the water column. Water monitoring data for August through October indicate that bottom water temperatures remained elevated above 20[degrees]C (CTDEP 1999), a condition which Powers et al. (2004), Chang (2004) and Draxler et al. (2005) report induces respiratory stress in lobsters."
Pearce, Jack; Balcom, Nancy. The 1999 Long Island Sound Lobster mortality event: findings of the comprehensive research initiative. (2005, October 1) *Journal of Shellfish Research.*

"Dr. [Carmela]Cuomo, [a marine ecologist and geologist at the University of New Haven] contended that even without the mixing event, the lobsters' long exposure to high water temperatures, low dissolved oxygen, ammonia and sulfides were enough to account for the 1999 die-off. She also warned that those conditions in Long Island Sound were far from rare. 'The probability of it happening again is very high,' she said."
Rather, John. Warm Water Killed the Lobsters. (2004, October 10). *The New York Times.*

"These warm water conditions may also have accelerated the growth of natural populations of the parasitic paramoeba, identified as Neoparamoeba pemaquidensis, found infecting the lobsters in 1999 (Mullen et al. 2005). In late October, more than 90 percent of lobsters examined were infected with paramoebae; those collected later in the fall had infection rates of about 29 percent (P. Howell, CTDEP, pers. comm.). This paramoeba inhabited LIS prior to 1999 (Mullen et al. 2005), therefore, it is possible that paramoebiasis was beginning to infect the lobster population prior to 1999.

Unfortunately none of the lobsters that died in 1997 or 1998 was set aside for a pathologic work-up. Strains of N. pemaquidensis are facultative pathogens of sea urchins (affecting the nerve ring), salmon and, now, American lobsters (Mullen et al. 2005). The presence of paramoebae in lobsters was monitored for 3 years following the 1999 die-off; more than 800 lobsters collected systematically throughout the Sound by CTDEP and examined by pathologists at the University of Connecticut exhibited infection rates of 0 percent to 14 percent (P. Howell, CTDEP, pers. comm.)"
Pearce, Jack; Balcom, Nancy. The 1999 Long Island Sound Lobster mortality event: findings of the comprehensive research initiative. (2005, October 1) *Journal of Shellfish Research.*

HYPOXIA (i.e., LACK OF OXYGEN)

"Since the late 1980s, the CTDEP Water Management Bureau has monitored water quality throughout the Sound. Dissolved oxygen data are used to assess the severity and extent of hypoxic conditions that develop in late summer in western LIS, which in 1999 were not especially severe (Gates 2000). Hypoxic conditions were present from July 2nd through August 21st, covering about 120 sq. miles (http://dep.state.ct.us/wtr/lis/monitoring/ summer99.htm). Hypoxic conditions were most severe during the first week of August. Data also showed a strong correlation between elevated bottom water temperatures and low bottom water dissolved oxygen levels in a west-to-east gradient across the Sound.

Three years of survey work by CTDEP in the early 1990s produced a response curve for lobsters with respect to hypoxic areas. Lobsters were found to "herd" or crowd in greater numbers near margins of hypoxic zones where dissolved oxygen (DO) concentrations were >2 mg/L, and leave areas where DO was [less than or equal to]2 mg/L (Simpson 2000). This suggests that as the lobsters moved from shallow to deeper waters to find cooler water in summer 1999, they were at the same time avoiding severely hypoxic areas, with the result that the lobster population was further concentrated in certain regions of western LIS."
Pearce, Jack; Balcom, Nancy. The 1999 Long Island Sound Lobster mortality event: findings of the comprehensive research initiative. (2005, October 1) *Journal of Shellfish Research.*

OVERFISHING

"By official standards, lobstermen are overfishing Northeastern waters. They are leaving too few lobsters to breed later generations, even in Maine, according to calculations of government biologists. Some predict drastic decline in Maine's future."
Donn, Jeff. Northeast lobster decline tied to warming. (2004, August 24). *Associated Press.*
"Biologists on a multi-state Fisheries Commission committee have found that warmer waters, disease and fishing have depleted lobster stocks, and they recently recommended a five-year ban on lobstering from Cape Cod to Virginia."
Struck, Doug. "Warming Waters Exacerbate Dwindling New England Fisheries." (2010, July 13). *Scientific American.*

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REVIEW OF MATERIALS BROUGHT FORTH BY CT LOBSTERMEN ON 2012 METHOPRENE LEGISLATION

In 2012, the Connecticut Lobstermen submitted a series of studies as evidence that methoprene was causing harm to lobsters in the Long Island Sound. It is vital to call out the fact that many of these studies had already been presented, discussed and shown by the scientists at Central Life Sciences during their testimony at the hearing to be unrealistic in terms of exposures and concentrations of methoprene used during the research. In their materials you'll find rates at 250 times the application rate, abstracts of studies but no data or conclusions, six studies repeated, four studies not having anything to do with methoprene, and, four studies that actually conclude methoprene is recommended due to low toxicity or lack of negative environmental effects.

The objectives of academic researchers do not always coincide with investigations of the safety of a chemical to the environment. Therefore, the designs and exposure concentrations of these studies do not translate to real-world conditions and the results are not beneficial for use to assess the impact of a chemical on the environment and the ecosystem. This is the case with a number of the laboratory studies done with methoprene. High concentrations were selected to try and elicit effect and conclusions drawn without understanding what, if any effects are present at environmentally relevant concentrations. The results simply do not translate to a real-world setting.

As Central Life Sciences has communicated previously, at recommended application rates, methoprene can rarely, if ever, be detected in the outflow areas from treated catch basins when it is distributed into waters associated with fish, lobsters and other organisms. If it is detected, it is found in rates far below what would be required to cause harmful effects to crustaceans.

In the following pages, we have provided additional insight into each publication brought forth by the lobstermen.

Study 1

Pesticide induced alterations in gene expression in the lobster, Homarus americanus
Horst, Walker, et al. (2007)

The laboratory testing for this study was done at a single dose which is far in excess of what has been found in the environment and draws conclusion that are not supported by the data presented.

This publication reports the results from a study of gene express by methoprene in adult lobsters exposed to methoprene at 50 ppb (over 250 times the effective rate) for 24 hours. The authors conclude that the data show that "a wide variety of cellular targets are altered by methoprene" based on their data on gene expression. At the request of Central Life Sciences, Dr. Carl Thummel, Professor, Department of Human Genetics at the University of Utah, an expert in the study of gene expression, reviewed the article. Dr. Thummel concluded that based on the data presented "I do not think they can conclude that these genes are significantly affected by methoprene." (Personal communication C. Thummel to M. Mizens, June 21, 2007)

The publication also draws erroneous conclusions that methoprene may have played a role in the 1999 lobster die-off. See study 7 for a description of the role methoprene had in the 1999 lobster die-off.

Study 2

Metabolic effects of acute exposure to methoprene in the American lobster,Homarus americanus
Walker, Bush, et al. (2005)

The laboratory testing was done at doses which are in far in excess of what has been found in the environment and draws conclusion that are not supported by the data presented.

This study claims that methoprene bioaccumulates in lobsters. The conclusions from this study need to be approached with some skepticism. The study design does not evaluate bioaccumulation of methoprene in lobster tissue according to EPA or internationally (OECD) established methods. The lobsters were only exposed four hours at an exaggerated concentrations of 50 ppb (250X the effective concentration of methoprene) and equilibrium between the lobster and the test solution was not established, a necessary condition for establishing bioaccumulation.

Study 3

Effects of pesticides of lobster health and trace level measurements and toxicological at environmentally realistic concentrations
Clemetson, Balcom, et al. (2001)

No data is presented here. This is description of the subject of a future presentation.

Study 4

Acute effects of methoprene on survival, cuticular morphogenesis and shell biosynthesis in the American lobster, Homarus americanus
Horst, et al.

No data is presented here. This is a description of proposed research.

Study 5

Metabolic effects of acute exposure to methoprene in the American lobster, Homarus americanus
Walker, Bush, et al. (2005)

This is a duplicate of Study 2 where laboratory testing was done at doses which are in far in excess of what has been found in the environment and draws conclusion that are not supported by the data presented.

Study 6

Acute toxicity of resmethrin, malathion, and methoprene to larval and juvenile lobsters, Homarus americanus and analysis of pesticide levels in surface waters after Scourge, Anvil and Altosid application
Zulkosky, Ruggieri, et. al.

Only the abstract of the publication was presented to the committee. Because the publication was available and reviewed by Central Life Sciences, the following comments are relevant to the toxicity of methoprene to lobsters in Long Island Sound.

The data presented in this publication show that methoprene in the environment after spray applications is below levels that have been shown to be toxic in the laboratory and that it does not persist in the environment.

The objective of the investigations presented here was to access the acute toxicity to larval and juvenile lobster and to measure methoprene (and other pesticide) levels in surface waters on Long Island and in Long Island Sound after pesticide applications in 2002 and 2003.

No toxicity was reported in larval lobsters at concentrations up to 10 ppb (50 times the effective concentration for methoprene) for 48 hours. The authors indicate that these findings are not consistent with those of Walker et al. (2005), but in agreement with those of investigations with other crustaceans.

Finding from the environmental monitoring were:

- Methoprene was detected after each of two spraying events at concentrations ranging from 0.0074 ppb to 0.631 ppb.
- In no cases were pesticides detected other than those applied in an actual spray event, indicating that none of those compounds are persistent in the environment.

Study 7

Application of water quality modeling technology to investigate the mortality of lobsters, Homarus americanus in Western Long Island Sound during the summer of 1999
Miller, et al. (2005)

Only the abstract of the publication presented to the committee. Because the publication was available and reviewed by Central Life Sciences, the following comments are relevant to the toxicity of methoprene to lobsters in Long Island Sound.

The objective of this study was to develop numerical models to quantitatively address the question whether application of methoprene (and three other pesticides) alone could have caused the massive die-off of lobsters in Long Island Sound in 1999.

From data supplied by NY and CT on the use of pesticides in 1999, between June and November 9.4 kg of methoprene were used in the area. The modeling demonstrated that if the methoprene had been able to get into the Sound (which it did not because it was used in a contained area) it was not a causative agent in the lobster die-off in LSI in 1999. The maximum concentration of methoprene calculated by the modeling was 0.0005 ppb, well below any lobster stress endpoint. The authors concluded, "It is likely that environmental conditions in Long Island Sound during the fall of 1999 other than West Nile related pesticide concentrations are responsible for the lobster mortality"

Study 8

Toxicity of insecticides form control of freshwater culex annu……..[text not legible]
Brown, Watson, et al. (2000)

Only an abstract of publication is presented to the committee. The study evaluated the toxicity of methoprene and other insecticides used for mosquito control in Australia to non-target shrimp species and concluded that due to the low toxicity of methoprene it is recommended for use for control of mosquito larvae.

Study 9

Projecting population-level responses of mysids exposed to endocrine disrupting chemical
Raimondo and McKenney (2005)

Only the abstract of the publication is presented to the committee. The publication proposes a model for estimating population-level effects on mysid shrimp using methoprene as the model compound. Data from previous research by the authors is used in this exercise. The relevance of this modeling to predicting effects on shrimp populations is unknown.

Study 10

The influences of insect juvenile hormone agonists on melamorphosis and reproduction in esturine crustaceans
McKenney (2005)

Only the abstract of the publication is presented to the committee. This article reviews data published on estuarine crustaceans of three compounds, methoprene, fenoxycarb, and diflubenzuron. No actual data on methoprene is presented. The focus is on using the published data to propose a 2-generation study in mysids to evaluate toxicity of chemicals.

Study 11

Sublethal effects of mosquito larvicides on swimming performance of larvivorous fish…[text not legible]
Hurst, Kay et al. (2007)

Only an abstract of publication is presented to the committee. The study evaluated the toxicity of methoprene and other insecticides used for mosquito control in Australia to crimson-spotted rainbow fish and **concluded that due to the low toxicity of methoprene it is recommended for use for control of mosquito larvae.**

Study 12

Pulse exposure effects of selected insecticides to juvenile Australian crimson-spotted rainbow fish
Brown, et al. (2002)

Only an abstract of publication is presented to the committee. The study evaluated the toxicity of methoprene and other insecticides used for mosquito control in Australia to juvenile crimson-spotted rainbow fish and **concluded that due to the low toxicity of methoprene it is recommended for use for control of mosquito larvae.**

Study 13

Effects of single and multiple applications of mosquito insecticides on nontarget arthropods
Davis and Peterson (2008)

Only an abstract of publication is presented to the committee. **The study evaluated the toxicity of a number of insecticides, including methoprene, on nontarget aquatic and terrestrial anthoprods in Montana and concluded that the insecticides had few, if any, deleterious effects.**

Study 14

Ecdysteroid responses of estuarine crustaceans exposed through complete larval development to juvenile hormone agonist insecticides
Tuberty and McKenney (2005)

This publication is not about methoprene.

Study 15

Evaluation of insecticide impregnated baits for control of mosquito larvae in land crab burrows on French Polynesian Atolls
Lardeux, Sechan, Faarula (2002)

This publication is not about methoprene.

Study 16

Effects of salinity stress on survival, metabolism, limb generation, ad ecdysis in UCA pugnax
Shock, Foran, Steuckle

This publication is not about methoprene.

Study 17

Toxicity of insecticides form control of freshwater culex annu……..[text not legible]
Brown, Watson, et. al.

This is a duplicate of Study 8. Only an abstract of publication is presented to the committee. The study evaluated the toxicity of methoprene and other insecticides used for mosquito control in Australia to non-target shrimp species and concluded that due to the low toxicity of methoprene it is recommended for use for control of mosquito larvae.

Study 18

Projecting population-level responses of mysids exposed to endocrine disrupting chemicals
Riamondo and McKenney (2005)

This is a duplicate of Study 9. Only the abstract of the publication is presented to the committee. The publication proposes a model for estimating population-level effects on mysid shrimp using methoprene as the model compound. Data from previous research by the authors is used in this exercise. The relevance of this modeling to predicting effects on shrimp populations is unknown.

Study 19

Pulse exposure effects of selected insecticides to juvenile Australian Crimsom-spotted rainbow fish
Brown , et al. (2005)

This is a duplicate of Study 12. Only an abstract of publication is presented to the committee. The study evaluated the toxicity of methoprene and other insecticides used for mosquito control in Australia to juvenile crimson-spotted rainbow fish and **concluded that due to the low toxicity of methoprene it is recommended for use for control of mosquito larvae.**

Study 20

Long term monitoring of grass shrimp Palaemoneisis app. population metrics at sites with agricultural runoff influences
Leight, et al. (2005)

Only an abstract of the publication is available. Methoprene is not mentioned.

Study 21 (Comments from EPA Meeting)

Transcript from meeting of Pesticide Program Dialog Committee (2005)

These are excerpts of the meeting transcript. The dialog appears to be about the issue of spray drift. With no context, a Dr. Fry states that methoprene goes downstream and starts killing lobster larvae. This is an isolated comment with no substance to back the statement.

Study 22

Lobster settlement and abundance in Rhode Island: An evaluation of methoprene application and other factors potentially influencing early survival
Gibson (2008)

This study, an evaluation of lobster populations and the use of methoprene at effective rates concludes in the Discussion section that, "Detailed analysis of abundance and mortality rate for several life stages of lobster in the Rhode Island area failed to reveal any evidence that the methoprene used in mosquito control programs had had any impact on the local lobster population."

Study 23

Acute toxicity of resmethrin, malathion, and methoprene to larval and juvenile lobsters, Homarus americanus and analysis of pesticide levels in surface waters after Scourge, Anvil and Altosid application
Zulkosky, Ruggieri, et. al.

This is a duplicate of Study 6. Only the abstract of the publication was presented to the committee.

Study 24

Morphologic effects of in vivo acute exposure to the pesticide methoprene on the hepatopancreas fo a nontarget organism, Homarus americanus
Walker, Golden, Horst (2010)

While effects were reported in this publication, the laboratory testing was done at doses which are far in excess of the efficacious concentration of methoprene for mosquito control as well as in excess of concentrations found in the environment.

The purpose of this study was microscopic examination of the hepatopancreas of adult lobsters exposed to 25ppb (125 times the effective rate) and 50 ppb (250 times the effective rate) methoprene for 24 hours. The details of the experimental design are sketchy, including the number of lobsters examined. Microscopic changes were observed at the doses tested. Whether the observed changes are transient was not examined. The study did not examine lower, more environmentally

relevant doses, to try to establish a no effect level. In the discussion the authors state “methoprene appears to cause both immediate and delayed morbidity in target and non-target animals, including the adult lobster.” While methoprene has been shown to have immediate effects on lobsters at high doses, delayed morbidity to lobsters has not been demonstrated.

Study 25

Bioaccumulation and metabolic effect of the endocrine disruptor methoprene in the lobster, Homarus americanus
Walker, Bush, et. al.

This is a duplicate of Study 2. The laboratory testing was done at doses which are in far in excess of what has been found in the environment and draws conclusion that are not supported by the data presented.

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SUMMER-FALL 2012 WEST NILE VIRUS NEWS COVERAGE BY STATE

CONNECTICUT

> First Human West Nile Virus Infection of 2012
> Two New West Nile Virus Cases Confirmed
> West Nile virus found in Eastern Connecticut; experts say don't panic
> Mosquitoes test positive for West Nile virus in 13 Conn. towns
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TEXAS

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> West Nile Virus Victim Questions Whether Health Department Delays Added to Epidemic
> From hospital bed, DeSoto resident advocates for West Nile spraying

CONNECTICUT

First Human West Nile Virus Infection of 2012

The person a New Haven resident between 30 and 39 years old.
NBC-Connecticut
Thursday, Aug 16, 2012

A New Haven resident is the first human case of West Nile virus infection in Connecticut this year.

The person is between 30 and 39, became ill during the first week of August and was hospitalized the following week with meningitis related to the infection, according to the
Connecticut Department of Public Health.

The person has been discharged from the hospital and is recovering, health officials said.

Symptoms of illness from West Nile virus included headache, stiff neck, nausea and muscle weakness.

The City of New Haven Health Department posted tips on protecting yourself on its Web site.

“August and early September is the time of the year when people are at greatest risk of illness associated with West Nile virus infections,” Dr. Randall Nelson, State Public Health Veterinarian with the Department of Public Health, said in a news release. “DPH urges everyone to take the warnings of the risk of mosquito-transmitted illness seriously and take precautions to prevent mosquito bites.”

Infected mosquitoes have been repeatedly trapped in New Haven and surrounding communities from July 12 through Aug. 8.

From June 27 to Aug. 8 the Connecticut Agricultural Experiment Station has identified WNV positive mosquitoes in 40 towns: Bethel, Bridgeport, Cheshire, Chester, Danbury, Darien, East Haven, Fairfield, Farmington, Glastonbury, Greenwich, Groton, Hamden, Hartford, Killingworth, Meriden, Milford, Monroe, New Britain, New Canaan, New Haven, Newington, Newtown, North Branford, North Haven, Norwalk, Old Lyme, Shelton, South Windsor, Southington, Stamford, Stratford, Wallingford, Waterbury, West Hartford, West Haven, Westbrook, Westport, Wethersfield, and Wilton.

Mosquitoes positive for eastern equine encephalitis were trapped in Chester on Aug. 8.

Between 2000 and 2011, there were 89 human cases in the state and three were fatal.

Two New West Nile Virus Cases Confirmed

One victim is a Stamford resident. Nationally, it has been a very busy season with many cases of the West Nile virus reported.
By Patch Staff
August 24, 2012

The most recent cases of West Nile virus are residents of Stamford and Bridgeport, both between the ages of 50-59. The Stamford resident became ill during the first week of August. The Bridgeport resident became ill during the second week of July. Both were hospitalized for encephalitis.

“August is when people are usually infected with West Nile virus in Connecticut," said Dr. Jewel Mullen, commissioner of the state Department of Public Health. "While most people do not become severely ill from West Nile virus, people over the age of 50 are more likely to become ill and develop serious symptoms when infected.”

The Centers for Disease Control say the West Nile virus, which is spread by mosquitoes, has been most prevalent this year. The most cases have been reported since the infection was first found in the U.S. in 1999.

Commissioner Mullen added that, so far this year, West Nile virus activity in the state is a couple of weeks earlier than usual but otherwise within the range of what has been identified in Connecticut over the past decade. Nationally, the 1,118 cases reported thus far represent the highest number of cases reported through the third week in August. Nearly half have been reported from east Texas.

Last year, there were nine reported cases of the infection in Connecticut residents and infected mosquitoes identified in 30 towns. So far this season, the Connecticut Agricultural Experiment Station has identified West Nile virus positive mosquitoes in 41 Connecticut towns.

For information on West Nile virus and what you can do to prevent getting bitten by mosquitoes, visit the Connecticut Mosquito Management Program Web site at www.ct.gov/mosquito.

West Nile virus found in Eastern Connecticut; experts say don't panic

By JIM KONRAD
Norwich Bulletin
Posted Aug 25, 2012 @ 09:52 PM

West Nile virus, which is responsible for at least 43 deaths across the nationthis summer, has reached Eastern Connecticut. But how concerned should you really be?

Don’t be afraid. Instead, local experts say , be smart, cautious and proactive, and you should be safe.

The mosquito-borne illness has been in Connecticut for some time — mostly in or near Fairfield and New Haven counties, reaching as far east as Glastonbury . On Thursday , the state Mosquito Management Program announced that two residents had tested positive for the virus in Stamford and Bridgeport. That doubled the state’s human West Nile cases in 2012 to four.

On the same day , the state also announced that of the four mosquitoes trapped Aug. 13 at Mount Misery in Voluntown, one tested positive for West Nile.

The state traps mosquitoes once a week at each of its 91 sites across the country . When the virus is found, trappings are done two to three times a week to see how prevalent it is in that region.

“We haven’t detected since (Aug. 13) in Voluntown, which is good news,” said Dr. Theodore Andreadis, chief medical entomologist for the Connecticut Agricultural Experimental Station.

“And, the mosquito is principally a bird biter. It’s not as significant as a human biter.” The only other municipality in New London or Windham counties in which the virus has been found this y ear is Groton. Overall, mosquitoes in more than 40 towns in the state have tested positive.

To put that single Voluntown mosquito into context, this y ear, the state has tested more than 139,000 mosquitoes, and 140 have tested positive for West Nile, five for Jamestown Canyon (another illness) and one for Eastern Equine Encephalitis.

West Nile has been around for years in Connecticut, and three people have died from it, the last in 2006. Eastern Connecticut has been able to limit human cases. Since 2003, there have been only two human cases in New London County — in Colchester and North Stonington, both in 2003 — and none in Windham County .

As a result, Eastern Connecticut residents haven’t been overly concerned about the virus. A few Brooklyn Fair attendees on Friday said they weren’t taking any precautions beyond their usual mosquito-avoidance habits.

“I’ll use spray and go inside if there’s a lot of mosquitoes around,” Canterbury resident Jane Norell said. “But it’s not because of the virus. Maybe it’s a matter of having blinders on, but I just slap them and move on.”

Pomfret resident Maria Sangiolo said she’s also largely unconcerned with the virus.

“We have a bat house and chickens, which would take care of the mosquitoes, anyway ,” she said.“If I see them, I just go inside.”

According to the Centers for Disease Control, only about one in 150 people who are infected with West Nile will get seriously ill — any thing from muscle weakness and vision loss up to numbness, paraly sis or death. About 20 percent of those infected suffer lesser symptoms, including fever, headaches and body aches and rashes.

The CDC say s people over the age of 50 are the most vulnerable to serious symptoms of West Nile.

“Treat every mosquito as if has the potential to infect you,” said Patrick McCormack, director of the Uncas Health District. “Be aware and know where the people of the highest risk are — young children and older folks.”

Surprisingly , the biggest places of risk for West Nile and other mosquito-borne illnesses aren’t in the country . They are in heavily populated areas and your backyard.

That’s because rural areas have fewer places where standing water can become a breeding ground. However, cities have catch basins — New Haven, for example, has about 20,000 of them — in which water can run off and stay for some time.

Also, an old tire with water inside or a bucket with a little standing water can become home for mosquitoes.

“In your backyard and house, make sure rain gutters are moved, your swimming pool is treated with chlorine and the tarp on your wood pile isn’t holding water,” said Paul Capotosto, the mosquito management supervisor for the Wildlife Division of the state DEEP. “Remove standing water once a week and you’ll remove the danger.”

Norwich City Manager Alan Bergren said the city is not planning any spray ing. He say s residents should follow Uncas recommendations about removing standing water.

The virus first showed up in the state in June, Capotosto said, and spread in July . Recent cool nights, in which temperatures have gotten down to the high 50s or low 60s, have slowed mosquitoes down. After mid-September, “mosquito activities start shutting down,” Capotosto said. “The first frost ends it.”
That frost is still six weeks or more away .

“We try to remind people that just because you don’t have a mosquito with West Nile Virus, it could be you don’t have a testing station,” McCormack said. “Just because a mosquito (in your area) hasn’t tested positive doesn’t mean you don’t have to take precautions.

Mosquitoes test positive for West Nile virus in 13 Conn. towns

Jul 26, 2012 3:12 AM CDT
By The Associated Press

HARTFORD, CT (AP) -
The state health department says mosquitoes testing positive for the West Nile virus have been found in 13 Connecticut towns.

The state's Mosquito Management Program says the mosquitoes were trapped for testing between June 27 and July 18.

The first positive test was reported in mosquitoes trapped in Stamford on June 27, and health officials said Wednesday more West Nile mosquitoes have since been found in Fairfield County and other areas.

The 13 towns include Bethel, Danbury,Greenwich, East Haven, New Haven, Newington, Newtown, Norwalk, Stamford, Stratford, West Haven, Wallingford and Wilton.

No human cases have been reported, but nine were identified last year. Last year West Nile-infected mosquitoes were found in 30 municipalities.

People are urged to use insect repellent and protective clothing, especially at dawn and dusk when mosquitoes are most active.

CT resident tests positive for west nile virus

WTNH-TV
Published : Thursday, 16 Aug 2012, 3:30 PM EDT

NEW HAVEN, Conn. (WTNH) -- A New Haven resident has tested positive for the West Nile virus.

This is the first case of human illness associated with West Nile virus reported in the state this year.

According to the Department of Public Health, the resident, who is in their 30's, had onset of the illness during the first week of August. They were hospitalized the following week with meningitis related to the West Nile virus. Symptoms included headache, stiff neck, nausea, and muscle weakness.

The person has since been discharged from the hospital and is recovering.

Officials said infected mosquitoes have been repeatedly trapped in New Haven and surrounding areas between July 12th and August 8th.

"August and early September is the time of the year when people are at greatest risk of illness associated with West Nile virus infections," said Dr. Randall Nelson, State Public Health Veterinarian, Department of Public Health. "DPH urges

everyone to take the warnings of the risk of mosquito-transmitted illness seriously and take precautions to prevent mosquito bites."

From June 27 to August 8 the Connecticut Agricultural Experiment Station (CAES) has identified West Nile virus positive mosquitoes in 40 towns: Bethel, Bridgeport, Cheshire, Chester, Danbury, Darien, East Haven, Fairfield, Farmington, Glastonbury, Greenwich, Groton, Hamden, Hartford, Killingworth, Meriden, Milford, Monroe, New Britain, New Canaan, New Haven, Newington, Newtown, North Branford, North Haven, Norwalk, Old Lyme, Shelton, South Windsor, Southington, Stamford, Stratford, Wallingford, Waterbury, West Hartford, West Haven, Westbrook, Westport, Wethersfield, and Wilton. Mosquitoes positive for eastern equine encephalitis were trapped in Chester on August 8.

For information on West Nile virus and what you can do to prevent getting bitten by mosquitoes, visit the Connecticut Mosquito Management Program website.

TEXAS

Dallas Revisits West Nile Virus Attack Plan

Birds not tested for virus; little larvicide purchased prior to epidemic
By Scott Friedman
NBC 5 (Dallas-Fort Worth)
Thursday, Sep 13, 2012

An NBC 5 investigation has found that Dallas County did not do some of the key things in the months leading up to the West Nile virus epidemic that experts recommend to identify and then slow the spread of the virus.

And, if the county wants a more aggressive program to fight West Nile virus, it may need to consider creating a new program or agency with additional funding to prevent future outbreaks.

One way to spot West Nile virus before it reaches humans is to follow a trail of birds.
That's what they do in Sacramento, where David Brown runs one of the country's best programs to combat West Nile virus, the West Nile Sacramento-Yolo Mosquito and Vector Control District.

Brown told the ***NBC 5 Investigates*** team that he believes closely monitoring birds can give researchers an earlier sense of what's going on because the disease is transmitted through birds, to mosquitoes and then to people.

To pinpoint the virus early Sacramento tests dead birds and then treats areas where birds tested positive.

One of the nation's top West Nile virus researchers agrees it can be an effective strategy.

"Very frequently (the birds) are some of the of the earliest indications of virus activity we see," said Dr. Bill Reisen, who runs a highly respected West Nile virus testing program at the University of California, Davis.

But in Dallas, in the months leading up to the West Nile virus epidemic, the city and county did not test dead birds. The Texas State Department of Health Services doesn't even accept them for testing anymore.

They used to send them to a lab in Wisconsin and said it was taking too much time to get results.

Reisen believes cities that don't test dead birds could be missing an opportunity to attack the virus earlier. But he said he understands it's another program that costs money.

Sacramento has a lot more money to fight mosquitoes, including a $10 million yearly budget for a dedicated mosquito control district. About 70 employees cover a two-county area.

Dallas County and the city of Dallas have just four employees each that are dedicated to mosquito control.

Sacramento runs hundreds of mosquito traps a week, testing them and quickly treating spots where the virus is found. Last week a ***NBC 5 Investigates*** report showed how the city of Dallas, per square mile, runs far fewer traps compared to cities such as Sacramento and Houston. And this year, Dallas didn't start trapping mosquitoes until May -- later than some experts recommend.

Chris Barker, a West Nile virus researcher at UC Davis, said setting more traps gives more specific information about the source of the problem.

“If money were unlimited, we'd love to have even more traps than we do in California,” said Barker.
Dallas County and city leaders tell us they're now re-examining the plan.
“We're going to look at every aspect of it,” said Dallas Mayor Mike Rawlings. “The amount of traps organizationally, the timing, it's not just one aspect, it's the whole kit and caboodle that needs to be looked at very closely.”

Sandy Flora contracted West Nile virus in Dallas in July and now suffers neurological damage.

“My daughter thought I was possibly having a stroke,” said Flora.

Now Flora wants the city and county to do more to keep others from being infected.

“I would tell them in the future they really need to start checking sooner,” said Flora. “And they just need to get out there and spray.”

In Sacramento, they search for water sources where mosquitoes breed. They find abandoned swimming pools and stock them with fish that eat mosquito larvae. They use aerial spraying before human cases arise and larvicide to stop mosquitoes from growing.

To get that level of service it costs the average taxpayer $15 to $20 a year. About the same price as a few bottles of mosquito repellant.

Having a dedicated budget stops the temptation to cut funding that can happen when West Nile virus cases drop -- or when health departments have competing priorities.

“They got the flu, they got STDs, they've got endless things to deal with and, often, mosquitoes, if there's not a big disease situation, they're not high on the radar screen,” said Reisen.

Records uncovered by ***NBC 5 Investigates*** show in the last few years Dallas County's health department rarely bought any larvicide to prevent mosquitoes from growing.

In 2012, the health department bought no larvicide until July 30 -- days after the Centers for Disease Control and Prevention told the city’s health department that Dallas was already at the highest-possible risk level for West Nile virus.

Records show no Dallas County Health Department larvicide purchases in 2010 or 2011 either.

County Health commissioner Zach Thompson would not talk to us on camera, but said the following in an email: "There was no need to purchase additional adulticide or larvicide in 2010 and 2011," mild years for West Nile virus.

Experts tell us staying aggressive in quiet times can help avoid outbreaks.

“I think you always want to have that program in place so you can intervene at the earliest possible moment; larviciding is one of the best possible ways to do that,” said Brown.

Dallas County Judge Clay Jenkins told ***NBC 5 Investigates*** the health department responded appropriately with the tools it had and they're studying how to make the program better.

“We're always looking to take the best ideas, as we move forward, to further the public good,” said Jenkins.

And, Jenkins said he's willing to consider creating a Dallas mosquito control district.

“I think that's certainly one of the options that will be discussed in the future,” said Jenkins. “We've got a public health committee that has input from all of the 31 cities here and they've got to be a part of the discussion.”

Asked if Dallas needs a mosquito control district, Rawlings responded: “Well, I'm not going to take anything off the table. I'm not into taxing people to take care of their mosquitoes. That doesn't sound like a good idea to me.”

For Flora, still suffering from West Nile virus, the extra cost would be worth it.

“It is a shame. It’s just a shame that things weren't done sooner -- not only for myself, but for all these other people,” said Flora.

West Nile's effects can sometimes last a lifetime

By Mitch Mitchell
Fort Worth Star-Telegram
August 25, 2012

Claire McCall says that a mosquito bite changed her life.

On Aug. 8, 2008, she entered a Dallas emergency room with a 103.4-degree fever. She would be diagnosed with West Nile virus.

"I went into a coma immediately," McCall said. "I was paralyzed. I couldn't swallow, couldn't talk and was being fed through a tube."

Four years later, McCall, 60, is on disability and still suffers with balance issues, has mild cognitive impairment, tremors and short-term memory loss. Fatigue, depression and chronic pain are a constant problem, she said.

Many people who catch West Nile feel like they have an extended form of the flu, with low-grade fever and fatigue that can last for weeks. But for others like McCall, the virus can attack the brain and nervous system, paralyze the body and leave life-changing impairments.

And in some cases, it can kill.

In Tarrant County, about a third of the reported West Nile cases have been neuro-invasive, the most severe form of the disease, public health officials report. Those who are 50 and over, diabetics, transplant recipients, others with weakened immune systems, chronic alcoholics and those with high blood pressure are most susceptible to the more severe effects. Some people may be genetically predisposed to contract the neuro-invasive infection.

But every case is different.

McCall, who is a nurse practitioner, said she considered herself very healthy before catching the virus. She had been taking a medication for psoriasis, a skin condition, that has since been taken off the market due to adverse reactions reported by other patients. Her treating physicians suspect that the medication may have weakened her immune system, McCall said.

Don Read, 70, a Dallas physician who was bitten in 2005, said he had a clean bill of health when he caught the virus. Read, who suffered through the worst symptoms the disease can cause, is serving on a committee that recommended aerial spraying for Dallas County.

Before he got sick, Read typically spent 88 hours a week as a surgeon with Texas Colon and Rectal Specialists. Now it's a strain for him to put in 35 hours a week, he said. He was paralyzed, had the brain swelling of encephalitis and also suffered from meningitis, the swelling of the membrane around the brain and spinal cord, all due to the West Nile virus.

"West Nile affected my stamina," Read said. "I have to wear braces on both legs. I can no longer do the abdominal surgeries because they require me to stand for long hours at the surgery table. And after standing awhile, I tend to fall down."

The disease affected his gait and required him to engage in physical therapy to learn how to walk and talk again, Read said. He has to take a nap every day to get through his part-time work schedule. Even people who suffer from West Nile fever, the milder form of the disease, can have long-term problems, Read said.

"It's like the flu times 10 or times 100," he said. "They may only get sick for a few weeks and then get better. But others will have decreased stamina for a week or two or even a year or for life because of West Nile fever."

Weakness, cognitive difficulties and behavioral changes are all pretty common residual effects for people diagnosed with West Nile encephalitis, said Richard Fulbright, a neuropsychologist who practices in North Texas.

"They go through an amazingly difficult period of unexpected physical difficulty," he said. "Then they go through long periods of rehabilitation."

The recovery process can grind down the will of some caregivers and children often have a difficult time reconnecting with a parent who has been diminished by the disease, Fulbright said.

"You can have a lot of behavioral changes," Fulbright said. "The patients can be irritable, it may look like they are being rude and inconsiderate, but it's the disease."

Kristy Murray, an associate professor of pediatrics at the National School of Tropical Medicine at the Baylor College of Medicine, questions whether West Nile disease ever goes away. The 200 West Nile virus sufferers she has followed for 10 years have experienced significant declines in kidney function years after their initial infection, Murray said. More than half of the people who suffer from neuro-invasive disease never return to their pre-infection health status, Murray said.

"It may be that it's a little like getting over the chicken pox," said William Schaffner, infectious disease specialist at Vanderbilt Medical School in Nashville. "We know that chicken pox can last in our bodies for decades and then resurrect itself as shingles later on. Every patient, it seems, does not eliminate the virus completely."

For Richard Worsham's mother, Dorris, the disease was fatal. Dorris Worsham, an 83-year-old North Richland Hills resident, died last week after being hospitalized and diagnosed with the virus, her son said.

An independent woman who still drove her own car, Dorris Worsham had taken precautions against the virus, using insect repellent and staying indoors in the morning and evenings. But still, she became infected and she died.

"The problem is clearly much worse than the number of confirmed cases indicates," her son said.

But while some call the cities' efforts to curtail the virus excessive, Richard Worsham said that cities could take an even tougher stance on this problem.

"It is disturbing to think that a more aggressive mosquito control policy may have avoided her death, and it's even more disturbing that there are people who fight taking action," he said.

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Missed Opportunities in Fight Against West Nile Virus

Dallas lacks resources, plans to fight virus as aggressively as other cities

By Scott Friedman

NBC 5 (Dallas-Fort Worth)

Monday, Sep 17, 2012

The plan for fighting West Nile virus in Sacramento, Calif., could offer lessons for Dallas and Dallas County.

In the months leading up to this year's West Nile virus epidemic, Dallas and Dallas County did not have the people, money or resources to fight the virus like cities with the most aggressive plans do, an NBC 5 investigation has found.

As of Sept. 5, Dallas County has 335 human cases of West Nile virus and 13 fatalities. There are more than 800 human cases and 23 deaths in all of North Texas.

Dallas County records obtained by the ***NBC 5 Investigates*** team show that the county health department knew on June 10 that 16 percent of trapped mosquito pools were already testing positive for West Nile virus.

Two weeks later, that number jumped to 28 percent. And by July 1, the numbers exploded -- 44 percent of the mosquito pools collected tested positive for West Nile virus.

Dr. Bill Reisen, who runs a West Nile virus-testing program at the University of California, Davis, one of the country's most respected programs, said he would have been alarmed to see the numbers Dallas County was looking at during the end of June and early July -- weeks before hundreds of people were sickened and weeks before county started talking about aerial spraying for mosquitoes.

When the Dallas numbers are put into the risk assessment worksheet he uses to advise California cities, it shows that Dallas County was likely at epidemic levels of West Nile virus as early as the end of June or beginning of July.

But it was three weeks after that point -- the end of July -- before Dallas County Judge Clay Jenkins said Dallas County Health and Human Services first alerted him the county was facing a crisis and needed more larvicide to fight mosquitoes. By then, Dallas County already had 64 human cases, and the first two victims had died.

When asked if he was concerned that health officials didn't ask for more help sooner, Jenkins said: “Well, hindsight is 20-20. But my focus right now is what do we need to do today to protect our citizens.”

Within days of talking with the health department in late July, Jenkins declared a public health emergency and started mustering support for aerial spraying.

By mid-August, planes were flying.

Jenkins said he is happy with the health department's response but is waiting for a Centers for Disease Control and Prevention report that is expected to address whether more could have been done leading up to the crisis.
In California, Sacramento had a similar outbreak in 2005.

Today, if the number of infected mosquitoes climbs to the levels the city of Dallas saw in late June or early July, Sacramento considers aerial spraying before human cases happen and before it reaches a point where the entire city needs to be sprayed.

“I think we've learned that when you see these kinds of numbers, you really have to make that intervention early to avoid having to do as widespread an intervention as we had to do in 2005 and you're having to do in Dallas now,” said David Brown, Sacramento-Yolo Mosquito and Vector Control District general manager.

Since the outbreak in Dallas started, city and county officials have reached out to Sacramento for advice.

Its mosquito control program is widely considered one of the best. Sacramento traps and tests a lot more mosquitoes than Dallas, and it tests year round, trying to catch the first signs of the virus early in spring.

“If you want to catch the beginning of transmission in order to sort of keep the genie in the bottle, so to speak, then early is good,” Reisen said.

NBC 5 Investigates learned that Dallas didn't start testing mosquitoes this year until May, even though the city's own mosquito plan suggests testing as early as April.

And experts told ***NBC 5 Investigates*** that April might even be late in a year with such a warm spring.

Brown agreed that a city could miss an opportunity to get a handle on things early by waiting to start trapping until May. Dallas Mayor Mike Rawlings said he's waiting for CDC recommendations on how early the city should trap and how many traps to use. He said the city plans to take a look at every aspect of its West Nile virus plan in the wake of the current crisis. “Let me tell you, any time someone dies and is sent to the hospital -- whether it's a police situation, a traffic accident -- you say, 'How can we do better?' And that's the way it should be,” he said. “We should be thinking, 'How can we be better all the time?'"

NBC 5 Investigates discovered that the city of Dallas right now only runs 20 mosquito traps per week -- about one for every 19 square miles.

Houston, which has the most aggressive plan in Texas, runs one trap for every six square miles. And Sacramento sets up hundreds every week to pinpoint where the virus lives.

Reisen said that adding more traps helps a city be more precise in finding and treating the virus.

But money is the key difference.

Sacramento's mosquito control district has 70 employees. The city of Dallas and Dallas County each have just four people dedicated to mosquito control in the areas they cover.

West Nile virus survivor Sean Lemoine, who suffered permanent neurological damage in 2009, said there's no question that Dallas should step up the fight.

“And my hope is that they take this lesson and make sure it doesn't happen in the future,” he said.

County Health Commissioner Zach Brown told ***NBC 5 Investigates*** that Sacramento's plan might be a good example, but the question is whether people in Dallas would be willing to conduct aerial spraying when the virus only shows up in mosquitoes and not in humans.

Currently, Dallas’ plans call for aerial spraying only when there are multiple human cases. Dallas County Health and Human Services Director Zachary Thompson said after the current crisis subsides, he wants to talk about the plan going forward.

West Nile Virus Victim Questions Whether Health Department Delays Added to Epidemic

CDC tells NBC 5 it recommended Dallas County "strongly consider" aerial spraying in July
By Scott Friedman
NBC 5 (Dallas-Forth Worth)
Monday, Oct 22, 2012

A doctor recovering from West Nile virus wonders if he and other victims would have been spared if aerial spraying had been advocated sooner.

A North Texas doctor recovering from West Nile virus wonders if he and some other victims who got the neuroinvasive form of the disease would have been spared if the Dallas County health department had pushed for aerial spraying sooner.

More than two months after Mike Clark got West Nile virus, he's still in rehab, trying to recover from the damage it did to his body and his memory.

"I just progressively worsened -- couldn't eat, couldn't drink," he said. "I got nauseated and finally had lost 26 pounds. It was, it was definitely scary. I mean, I didn't know if I was going to get better or worse."

If Clark wasn't recovering from West Nile virus, he might be treating patients with West Nile virus. He's a doctor, but he hasn't been able to work since August.

"There are a lot of people who wouldn't be in the hospital, who wouldn't be in the situation I'm in, had things been done faster," Clark said.

As the NBC 5 Investigates team first reported Thursday, the Centers for Disease Control and Prevention says it recommended that the Dallas County health department "strongly consider" aerial spraying in late July. The CDC said it gave the Dallas County health department that advice in a conference call on July 25. Dr. Janet McAllister with the CDC led that call.

"In the situation that was described, aerial spraying is, is the most effective way to treat large areas, which is what Dallas County was needing and experiencing widespread cases," she said.

But if the CDC advised aerial spraying in that call, Dallas County Health and Human Services Director Zach Thompson apparently never passed that information along to County Judge Clay Jenkins, the top official who could authorize aerial spraying.

In a statement, Jenkins told NBC 5 Investigates: "My first communication from anyone regarding the possibility of aerial spraying for the 2012 WNV outbreak was August 6, 2012."

Aug. 6 is 12 days after the date the CDC says it told the health department to strongly consider aerial spraying.

Thompson disputes what CDC told NBC 5 Investigates.

"I've set the record straight that the recommendation you're talking about is a recommendation that the CDC looks at overall planning," he said. "First you do surveillance, you do enhanced spraying, and then you, you, go to aerial spraying."

Thompson won't tell NBC 5 Investigates what he believes the CDC told him on that conference call, but he insists he followed CDC and county plans.

"The information you're pointing out is incorrect," he said. "There is a plan, and we followed that plan, so your information and your story that you put in place is incorrect, so have a good day."

In the end, Jenkins said he based his decision to aerial spray largely on advice he got directly from the CDC.

"The CDC told me Friday when I made the decision to request the planes that the time that we wait can be counted in additional West Nile cases and human life," Jenkins said at an Aug. 16 press conference.

But is it possible that Jenkins might have gotten some of that advice sooner if the county health director told him what the CDC says it told Thompson in the July 25 conference call?

Clark said he thinks any information gained from the CDC should have been shared with the decision makers.

"I mean, I think you should take into account what people who are supposedly experts on this are saying, and CDC is pretty expert on stuff," he said.

NBC 5 Investigates has repeatedly asked health director Thompson to sit down to talk in detail about how his department responded to the West Nile virus epidemic, but he has declined our requests.

Couple: Living With West Nile Virus a Challenge

Dallas man with serious form of disease contracted virus 10 years ago
By Ray Villeda
Thursday, Aug 2, 2012

Ran Kilpatrick is still fighting with West Nile Virus, a disease he contracted virus 10 years ago.

A Dallas couple says West Nile virus has changed their lives forever.

Ran Kilpatrick, contracted the virus 10 years ago, has short-term memory loss and seizures.

"My heart gets to beating real fast, and I ... have to stop, take a few deep breaths. I get shaky and dizzy and just my symptoms go berserk," he said.

His wife, Rebecca Kilpatrick, said she always checks up on him because he has seizures on a nearly daily basis.

"I have to make sure I know where he's at, what he's doing, who he's with," she said.

Kilpatrick said he doesn't remember getting bitten or much time before that.

"I don't remember that summer at all, I mean, literally, at all," he said. "I don't remember the school year beforehand, or anything like that."

Dr. Chris Perkins, Dallas County health department medical director/health authority, said Ran Kilpatrick's symptoms sound like the most severe form of West Nile virus, the neuroinvasive form that affects the nerve system.

"They have severe headache, neck stiffness, forgetfulness, memory loss, confusion, disorientation," Perkins said. "They might start having paralysis or advance to having seizures."

He said there is no cure for West Nile virus, saying doctors can just treat the symptoms. The damage is irreversible, he said.

Less than 1 percent of those infected with West Nile virus will experience the serious form of the illness. Most people bitten by a West Nile virus-infected mosquito will not show any symptoms.

Dallas County has seven confirmed human cases of West Nile virus in Dallas, Richardson and Irving.

The city of Dallas will spray for mosquitoes in several areas from 10 p.m. Friday night until 3 a.m. Saturday. The city sprayed near Dallas Love Field on Monday night and early Tuesday morning.

From hospital bed, DeSoto resident advocates for West Nile spraying

by JANET ST. JAMES
WFAA-TV (Dallas-Fort Worth)
Posted on August 14, 2012 at 3:55 PM
Updated Tuesday, Aug 14 at 10:34 PM

DALLAS — Just turning on the light in the hospital room is enough to bring Katharyn DeVille to tears.
"It's this killer, killer headache," she explained. "It just makes me feel like somebody has an axe in my head and it hurts really bad. So this light is pretty excruciating."

That's what West Nile meningitis feels like to her.

Two weeks ago, DeVille said flu-like symptoms exploded. "When I came in here Thursday for the spinal tap, I was like delirious and couldn't function," she said.

That's when she was admitted to Methodist Dallas Medical Center. It took three days to get a diagnosis of West Nile virus. Her brain scans confirmed West Nile meningitis and encephalitis.

"Which means that the virus has infected not only the lining of the brain, but part of the brain tissue itself," explained Dr. Edward Dominguez, a Methodist infectious disease specialist and DeVille's doctor. "Which would probably explain why she is more ill than some patients who only have fever or a rash and fever and get well quickly."

Dr. Dominguez said no one knows why one person is severely affected while another is not.

Katharyn DeVille is a healthy 42-year-old and a former Channel 8 producer who works in the interactive department of Belo. She never dreamed after 20 years of covering the news, she would become part of the news.

DeVille believes she was bitten in her DeSoto backyard. So when DeSoto Mayor Carl Sherman came to visit her in the hospital, DeVille encouraged him to consider every option, and to weigh the risks versus benefits of spraying for mosquitoes when it comes to protecting people.

"Somebody who sprays up on my street, let me tell you, it is not going to get down the hill, down through my backyard, to the creek that's behind my house, which is where those mosquitoes are breeding," she said. "If my daughter gets sick, I'm going to be mad... I'm going to be mad. If more of my friends get sick, more of my neighbors, because they sat on their hands, then I'll be very upset."

DeSoto is south of the Interstate 30 corridor. Mayors from the southern part of Dallas County were not invited to Monday's meeting to consider aerial West Nile spraying.

But DeSoto Mayor Sherman said the option was opened to his city on Tuesday.