An Alternate Perspective on the Use of Aerial ULV Spray to Attempt to Control Transmission of West Nile Virus in Sacramento County, 2005

James Northup, Jack Milton and Paul Schramski
Pesticide Watch Education Fund
July 10, 2008

Summary. Beginning in the summer of 2005 and continuing over the next two summers, the Sacramento-Yolo Mosquito Vector Control District (SYMVCD) has subjected the people of Sacramento and Yolo Counties to a grand experiment by spraying pesticides from the air in an attempt to control the spread of West Nile virus. While West Nile disease is one of concern, there is currently little evidence of safety or efficacy of aerial pesticide spraying in the published scientific literature. In May 2008, three years after the initial experiment, the California Department of Public Health (CDPH) and SYMVCD offered the Carney report of their 2005 spraying of Sacramento as evidence of efficacy, despite the lack of referee's reports from independent scientists.

A review of the 2008 Carney report of the 2005 spray reveals several areas of concern, including: 1) the data fails to support the hypotheses, 2) unjustified analytic methods were used to analyze a buffer zone at the margins of the spray area, 3) the spray protocol does not match WNv transmission dynamics, 4) inappropriate and/or inapplicable statistics were used, and 5) scientific studies that used appropriate methodologies have been misinterpreted and used to make inappropriate assumptions and draw incorrect conclusions. In particular, the buffer zones were not initially set up as control zones, and when these margins are treated correctly the perfect effect of the spray disappears.

Information about significant factors that also nullify any effect, such as the wind having interrupted the spray for a total of eight days, was omitted from the report. There are several alternative explanations of the results, but the authors do not consider any of the alternatives.

Several Public Record Act requests to CDPH failed to produce complete WNv infection data from the different zones, so we are unable to analyze the results independently. Regardless, the methodology in the Carney report is weak, rendering the missing raw data irrelevant. The authors' lack of transparency raises troubling questions about the accuracy and comprehensiveness of the data used to construct the study. In contrast to most of the Carney report, data available on the CDPH website demonstrate that the virus transmission was declining prior to the spray, as do the Sacramento 2005 and Yolo 2006 timelines.

The SYMVCD falsely claims that aerial pesticide spraying, and nothing else, works in slowing the transmission of WNv. Officials across the state and country have cited this flawed report as evidence of the need for spraying. Even though there is a lack of evidence that aerial spraying for mosquitoes has slowed the transmission of WNv, other methods of control have proven effective.

WNv is in many ways like another mosquito-borne disease that has been endemic in California for over 50 years. From what is known scientifically, WNv should follow the same pattern that Western Equine Encephalitis (WEE) has followed. This disease is more likely to produce a serious disease than is WNv, but it has reached what is called chronic endemicity, and the levels of transmission are so very small and cases so very few that it tends to be ignored by the public. Nonetheless, public officials have suggested that WNv is spreading, and once it gets firmly established in a region we can expect greatly increased rates of infection and numbers of serious cases of the disease. This view is background to the exaggerations that public officials and the media have engaged in on a regular basis since the introduction of WNv into the state.

Until public officials gain a better understanding of WNv, and until a more thorough assessment of the data has proven the safety and efficacy of aerial pesticide spraying, a moratorium should be placed on further spraying. In the interim, other safe and effective methods of mosquito control should be implemented and increased.

A Great Experiment Without Evidence of Safety or Efficacy. Beginning in the summer of 2005, the Sacramento-Yolo Mosquito and Vector Control District (SYMVCD) has engaged in the aerial release of insecticides over residential neighborhoods purportedly to control the transmission of West Nile virus. Our opposition to this program has always rested on one simple predicate: the release of toxic materials into the environment must first be thoroughly justified with rigorous scientific scrutiny and the careful evaluation of the risks and benefits. With respect to the ultra low volume (ULV) release of *piperonyl butoxide* and pyrethrum from aircraft over residential neighborhoods there is neither convincing scientific evidence of the safety to the public health and the environment nor is there evidence of the efficacy of this protocol at reducing or preventing the transmission of West Nile virus.

There had been no scientific test to evaluate the safety to human health and the environment of this protocol of aerial ULV release of pyrethrum and *piperonyl butoxide* or of any of the related pyrethroid insecticides. District and CDPH officials cited studies in their claim of safety. However, on review none of these studies measured the exposure risk from aerial release but instead measured the ULV ground spray release of these materials. The District exposed the residents of Sacramento County to an experiment with serious shortcomings – no data was gathered with respect to health and safety questions, nor were measurements taken of spray exposure. The actual safety of this protocol remains untested according to any rigorous scientific standard.

SYMVCD Falsely Claims That Spray, and Nothing Else, Works. Public health officials and the SYMVCD have consistently claimed that once infected mosquitoes transmit the virus to people, the only way "to break the cycle of transmission" (e.g. Howard, 2006) is to spray adult mosquitoes with insecticides. This claim is erroneous; there are a number of other potential approaches to reducing exposure to transmission of the virus, and this claim falsely assumes that the spray effectively reduces the transmission of the virus. There has been no support for either claim in the scientific literature. Recent studies by Harvard researchers concluded that the pyrethroid ULV spray protocol did not have any impact on the segment of the mosquito population that could be infected with the virus, those that are blood-engorged or gravid (Reddy, 2006). These mosquitoes tend to be more sedentary and conceal themselves, avoiding contact

with the spray. Other research sponsored by the World Health Organization (WHO) has shown an equivalent lack of impact on yellow fever and dengue transmission, two other mosquito-borne diseases (Rawlins, 1998; Newton and Reiter, 1992).

To provide the greatest impact in protecting public health, both the literature and successful practice in other regions indicate that alternate measures to control mosquitoes must be emphasized to a much greater extent. Reducing the aquatic breeding grounds of vector mosquitoes should be our first priority. Public health officials in the Washington D.C. area took this approach. Without the use of any spray against adult mosquitoes, and following a very severe WNv outbreak in 2002, with breeding ground reduction in place, there was no WNv transmission to people in the following four seasons (McCaffrey, 2007).

Increased expenditures on mosquito bite prevention education and to provide material support for building maintenance, including window and door screens or bed nets for vulnerable populations, should be a priority. These measures, along with increased primary health care, have been proven by WHO research to be the most effective measure at reducing epidemic transmission of malaria and yellow fever, two other mosquito-borne diseases, without any additional vector control efforts (Rojas, 2001).

Spray Protocol Fails to Match Transmission Dynamics. A review of the ecology of WNv further illuminates the problems with the use of aerial pesticide spraying of adult mosquitoes to control the transmission of the virus. The virus requires both birds and mosquitoes to propagate, it is restricted to a few species of mosquitoes, and the hosting of the virus is restricted to a few species of birds. The mosquitoes can transmit this virus to a number of other vertebrates but they can only catch the virus from birds, and the birds can only catch the virus from the bites of an infected mosquito. For this reason, transmission of the virus to humans requires the overlap of populations of these specific bird and mosquito species. Such areas would be typified by heavy tree canopy and associated bodies of water. It is no surprise to see that the areas with the highest symptomatic infection rates to date are rural and forested regions with significant watershed. For example, Glenn County, with a frequency of 49 cases per 100,000 was the highest in California. Los Angeles County, which didn't engage in aerial spray and is markedly urban and arid by contrast, had a rate of 4 per 100,000 at its peak. Sacramento County, which sprayed heavily, had a peak rate of nearly 15 per 100,000.

To eliminate the virus in the areas where transmission occurs, all contact between infected birds and mosquitoes must be prevented for a period longer than the virus persists in either species. The mosquito may pick up the infection at its first blood meal some 2 1/2 days into its adult life (Moon, 1976). The infection must incubate until the mosquito becomes contagious at day 7 (Wonham, 2004). It can carry the infection the entirety of its life, which may be as long as 14 days during the summer months in this area, and for longer than 4 months in the fall generation, a physiologic status known as diapause (Gillett, 1971). To prevent the virus from continuing into the next generation of mosquitoes, there must be no infected birds or mosquitoes as of the fall months.

Far More Rigorous Protocols Would Be Required. To prevent new infections from occurring, all mosquitoes would have to be killed for a period of time covering the incubation

and longest lasting infection in the host birds. Some species of birds may circulate sufficient virus in the blood to infect the vector mosquitoes for as long as 7 days (Reisen, 2006). Even with a minimum incubation time we see a period far greater than the 3 days covered by the aerial spray. Keep in mind also that while one infected mosquito remaining in a region during the summer months can only transmit the infection perhaps twice in its lifetime, one infected bird can infect hundreds of mosquitoes. The three-day spray protocol engaged in by the SYMVCD doesn't come close to meeting the requirement for elimination of the virus, even under ideal conditions.

Additionally, one must consider that kill rates in caged mosquitoes range 35-40 percent per spray application, instead of the 100 percent kill rate per treatment that would be required in the natural mosquito population effectively to reduce the virus. Actual kill rates are typically much lower than such rates, yielding an even more pessimistic assessment of this protocol. Indeed, according to David Pimentel, Ph.D., an entomologist at Cornell University, close to 99.9% of sprayed chemicals settle in the surrounding environment where they can have detrimental effects on public health and ecosystems, leaving 0.1% to actually hit the target pest (Pimentel, 1995).

A Badly Flawed Study, Widely Cited. For more than two years now representatives of the CDPH and SYMVCD have been publicizing a claim of a proof of efficacy for aerial pesticide spraying (e.g. CDPH 2005; ASPH 2008; Greenwood 2006). Several Public Record Act (PRA) requests were required to obtain a pre-publication draft of the CDPH/Carney report, and the final report was later posted online in May 2008 (Carney 2008). Unfortunately, the materials furnished did not include maps of the daily distribution of cases of WNv or the full data set that would be needed for a thorough critical analysis. Local officials cite this as a peer-reviewed scientific study, but not a single referee's report by an independent scientist was supplied in answer to our PRA requests.

No Appropriate Scientific Measurements, Studies Misinterpreted. The most significant problem with this study is the failure to organize and report complete scientific data for the important parameters in question. The study lacks actual incidence numbers of infection in people and the location where they acquired the infection, even in summary form that protects the identity of the individuals. In addition, the references cited in the report as "methodologically consistent" (Ruiz, 2004; Mostashari, 2001) do not support the assumption that all of the transmission of the virus to people occurred at or near their places of residence. Proper scientific method would require careful assessments concerning the location of transmission. Instead, authors of the study simply make an unsupported assumption.

There are two fundamental problems with the assessment of infection rates in the Carney report. First, WNv is very cryptic in people. That is, most people show either no symptoms or only very mild disease, making reports from those who experience symptoms a highly inaccurate sample of the infection rate. Second, the case reporting system, which depends upon patients seeking health care from their physicians, only reveals one in every 25 to 30 actual infections (Loeb, 2005; Mostashari, 2001). Obviously, such an assessment of infection rates will be badly flawed. The only accurate way to measure infection rates is to draw blood from randomly drawn samples of the population, known as serological assay.

As to the transmission of virus to people, the authors of studies cited in the CDPH report used a methodology known as landscape epidemiology to assess the relative risk factors for acquiring infection, including common locations and activities. The CDPH did not make use of serological assay or landscape epidemiology.

The cited studies, done under the more accurate methodologies, concluded that the majority of the transmission of infections occurred in the hours after sunset to people engaged in outdoor activities without adequate protection from insects. Instead of using appropriate scientific methodology, the CDPH has cited these conclusions and extrapolated that all infections must have occurred during hours when most people are at home and therefore all infections occurred at the place of residence. This extrapolation is not supported in the studies cited by the CDPH in the Carney report.

CDPH should have measured the rate of infection by randomly drawing serum samples from the exposed population and performed follow-up interviews with all seropositive samples to determine where and how they may have caught the disease. Instead of drawing well-supported conclusions about location of transmission, the CDPH has simply declared an unsupported assumption.

Data Fail to Support Hypotheses. The analysis presented by the CDPH requires all of the following assumptions be true:

1) All transmission occurred at the place of residence
2) The virus only re-enters any region in infected mosquitoes, not birds
3) Infected mosquitoes fly only 0.88 km in their lifetimes

Yet drawing from the literature, the only reasonable conclusions that can be drawn are that the majority of transmission happens outside the home (Ruiz, 2004), birds carry the virus from locality to locality (Reisen, 2004; Wonham, 2004), and mosquitoes may fly farther than 0.88 km in one night and 20 km in a lifetime (Dow, 1965; Gillett, 1971).

Given a profound spray effect as concluded by CDPH, then a clear pattern of the distribution of infection should be visible after the spray. This pattern would exhibit the least infection at the geometric center of the treated area. If the distribution of infections prior to the spray were uniform there would be a gradient of increased infection at the margin in the treatment area, with increasing infection into the center of the untreated area. However, what is actually observed is a greater rate of infection at the margin in the treatment areas than in the untreated areas.

Unjustified Analytic Method for Margins. In addition to the flawed assumptions, CDPH engages in an entirely unjustified analytic method with respect to the parameters of the spray area. The report states that in order to account for the infiltration of infected mosquitoes and the diminishing efficacy of spray due to drift, they have removed from consideration as treated an area of 0.88 km deep around the margins of the spray treatment zones. Instead of being analyzed as treated areas they were designated buffer zones. These areas were actually

sprayed from the aircraft but have been considered in the CDPH study as if they were not treated. It is inexplicable that the CDPH would concern itself with accounting for mosquito movements and migration and not the movements of people and birds. It is, in fact, well established in the literature that birds are carrying this disease from place to place, not mosquitoes (Reisen, 2004; Wonham, 2004).

A more legitimate adjustment would be to create buffer zones in the opposite direction, one-half mile into untreated regions to account for the drift of the spray. Then one might see a diminished treatment phenomenon at the margins of the treated area. But it is entirely unsupported in research methodology to take a region that was the recipient of the treatment and include it in the statistical analysis as an untreated sample.

Unreleased and Incomplete Data. The data tables released to us in the "pre-publication draft" and following several PRA requests are incomplete, so that we cannot review the accuracy of claims about t-test of means, since we do not have sufficient information about the temporal distribution of the listed cases. Neither can we fully review the post-spray Chi Square results since we do not have information about the spatial distributions of cases. Still, we can review the pre-spray Chi Square results.

Buffer Zones are not Control Zones. The conclusions in the report imply that the designated spray and no-spray areas had no difference in the number of human cases of WNv before the spray, and had significant differences after. But, in fact, there were marked differences in the pre-treatment rates, with the "buffer zones" having nearly a 2.5 times greater rate prior to spray as the southern treatment zone. If we lump the "buffer zones" back in with the treatment zones, the appearance of perfect efficacy disappears from the study. Since we do not have documentation of the daily distribution of cases by region we cannot be precise in the analysis. However, we can say with certainty that the zero count in either one or both the treatment areas greater than 2 weeks post spray should be shifted to non-zero. As a lumped group the "buffer zones" had a 3.6 per 100,000 rate of infection greater than 2 weeks post spray, while the untreated areas were 3.3 per 100,000.

The "buffer zones" don't present a valid control since the rate of infection was appreciably higher in the "buffer zones" prior to spray i.e., 14.5 vs 5.8 per 100,000, and effects of the spray can have no bearing on that particular distribution.

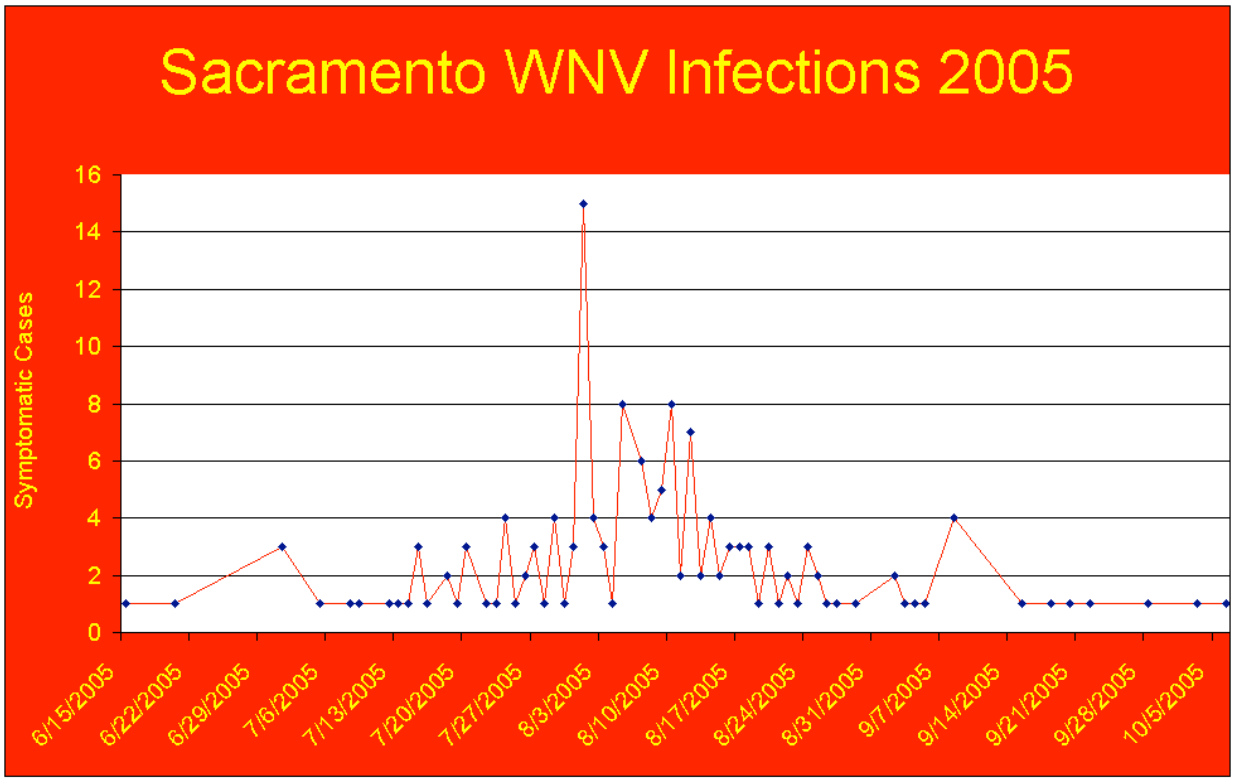
Alternative Explanations of the Results. If the "buffer zones" do not form a legitimate control zone, the distribution of the data must be further questioned. One hypothesis is that areas that receive an early colonization of the virus end up with higher rates of infection that take longer to recede.

Another is that the margins of large waterways are areas of greater transmission. Either of these hypotheses falls within the natural cycles of virus propagation and would have nothing to do with any treatment phenomena.

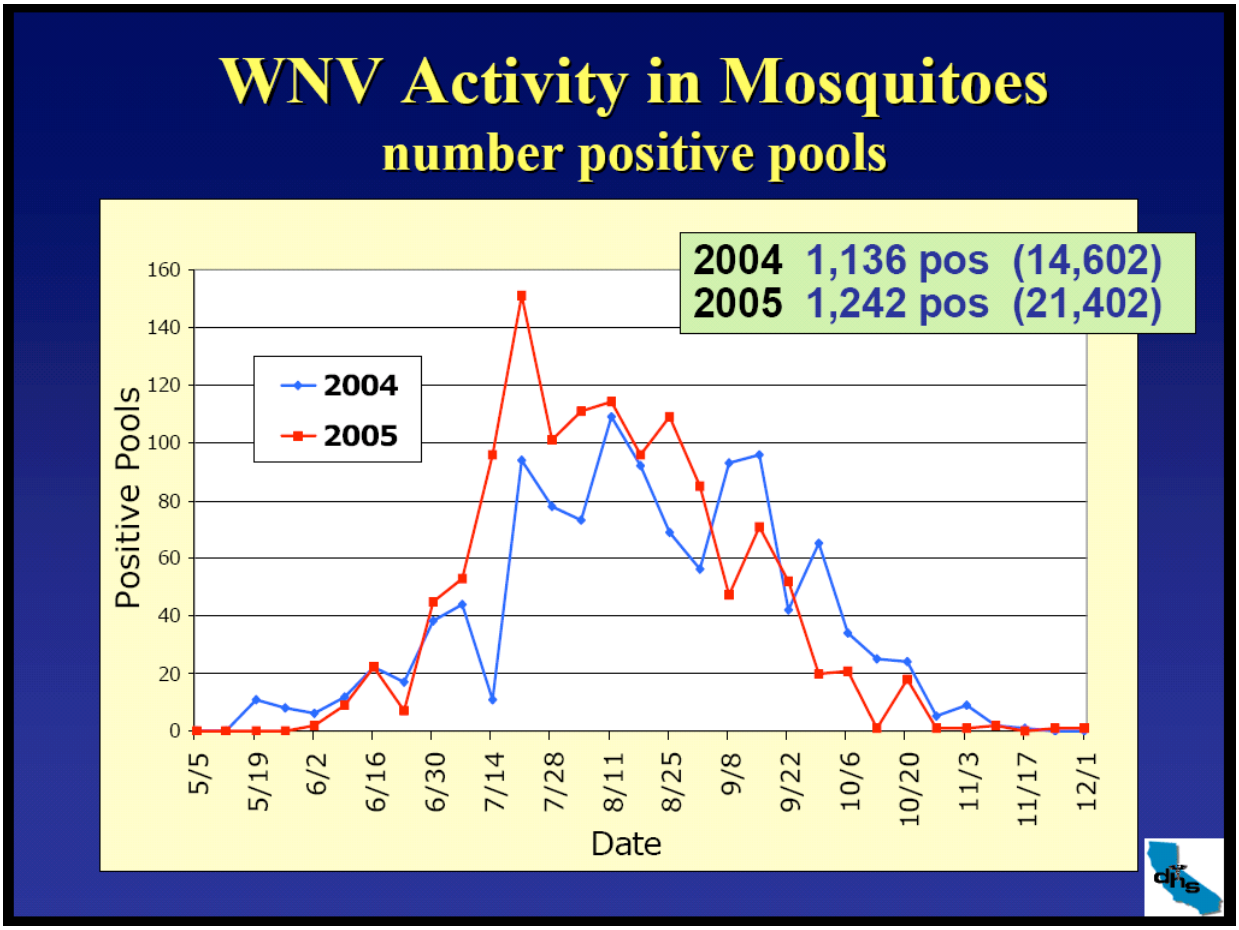
A third hypothesis has to do with weather conditions during the time when data was being collected. Though the CDPH report makes no mention of it at all, during the time period

of this aerial spray protocol there were 8 successive days of high winds that kept both aircraft and mosquitoes from flight activities. High winds create a "treatment effect" on the transmission of mosquito-borne disease since they inhibit flight and biting behavior in the vectors. This interferes with the reliability of mosquito traps, so that counts of mosquito populations taken during windy periods cannot be compared with those taken during periods of more favorable weather. Moreover, mosquito growth in the days between treatments would largely negate any effects of the initial treatment.

Virus Transmission Declining Before Spray. The question of the natural timeline of the epizootic (multiple simultaneous infections in a non-human host) and its relation to the appearance of symptomatic disease in people can be reviewed to some degree with the data in this CDPH report, supplemented by ecological data provided in other CDPH publications. The timeline of symptomatic disease showed the highest new case counts for Sacramento County on August 1st with 15 new infections reported having an onset of symptoms on that date.



The highest rate of virus observed in the birds and mosquitoes that same summer was in the sample of July 21st. (Reisen, 2006)

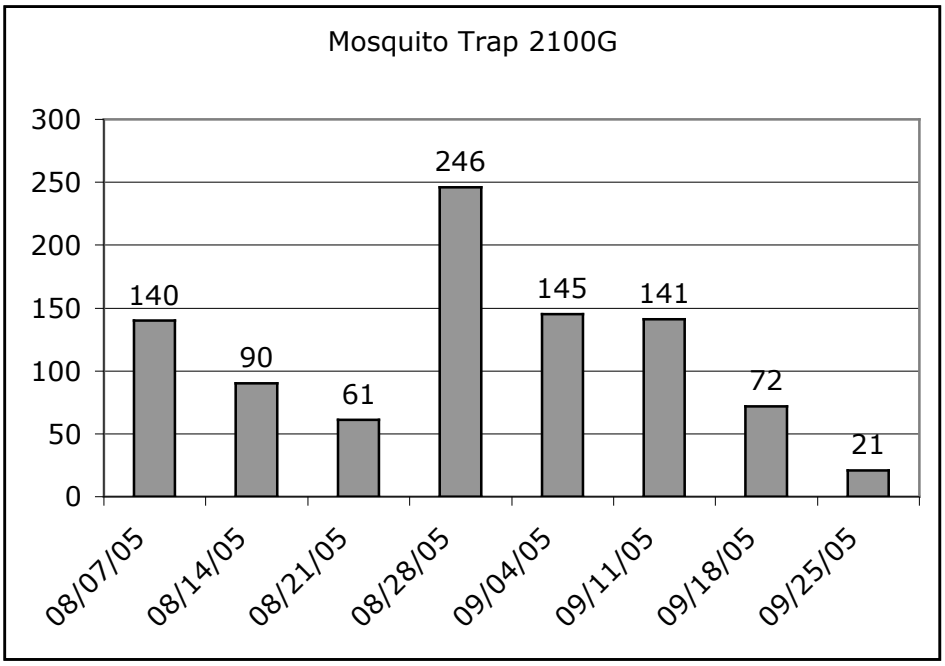


This indicates that the rate of transmission of the virus was declining more than 2 weeks before the aerial spray treatment. That is, the spray was done after the "peak". It is well acknowledged that anything done after the peak will appear to be effective, as the rate of infection is going down anyway.

Impossible for Spray to Cause Decline in Transmission. There's no possibility that the treatment that began on August 8th could account for declining transmission, which began between the July 21st measurements and the July 28th measurements indicated in the CDPH monitoring of the virus in birds and mosquitoes for California in 2005. And with the data showing an increase in mosquito population, not just through July but also through the end of August, it seems very unlikely that the spray could have been the causal agent for the declining infection rate, since it would require a precipitous decline in mosquito populations for the spray to have produced the decreasing transmission, and no such decline was observed.

The Carney report attempts to correlate the spray events in August to a claim of declining mosquito populations. It cites unpublished and unreleased mosquito count data of 46 trap days for one week before and one week after the spray. It notes that these data indicate substantial declines in Culex tarsalis and Culex pipiens populations, but the conclusion of substantially declining mosquito populations after the spray is refuted by the mosquito census data published by the SYMVCD on the Internet but not cited in the report. The SYMVCD data includes more

than 450 trap days in each weekly sample. Trap 2100G, situated in the North Spray Zone, was typical of the samples.

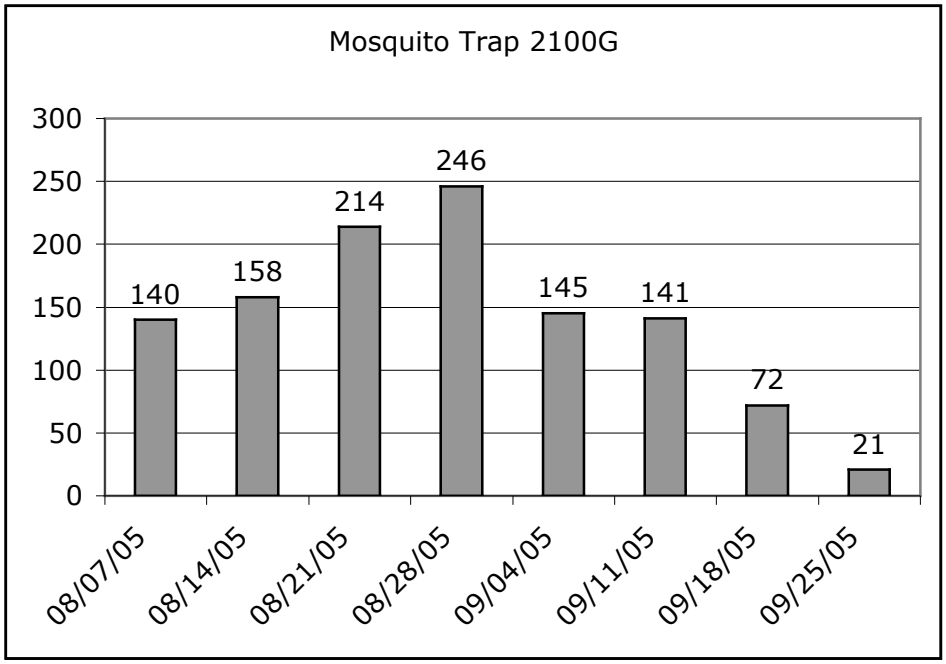


A great deal can be inferred from the numbers in this gravid trap sample. It shows a population increasing to its peak in late August at a rate of 0.027 per day or nearly 3% daily growth. This is a population doubling in a month's time, a rapidly increasing population. Since these counts were posted weekly, an initial view might have been to surmise a remarkably effective spray. The mosquito counts drop by one-third each of the first two weeks post spray. Then they rebound by more than four fold in the third week. This rebounding population belies the idea of any dramatic reduction of the vectors by the spray.

The growth rates in the weekly mosquito counts suggest that the appearance of dramatic decline in the two weeks after the aerial spray in the North Zone are artifacts of the wind, which was distributed 3 days and 5 days in those two weeks. Mosquitoes cannot increase their population size at a growth rate of 22 percent per day, which would be necessary for a four fold increase in one week, creating a level of certainty that the wind is more likely responsible for the temporary decrease in infection rates than a temporary eradication of the mosquito population.

Actual Population Dynamics. All the other sample weeks in the light trap data for August were free of high winds. To compare samples that have 7 good days of mosquito counts with those that have only 4 or 2, such as the samples of weeks 2 and 3 in August, we can either divide all weekly counts by the number of good trap nights and compare average nightly counts or we can standardize the weekly count by multiplying each by the reciprocal of the proportion of good trap nights in each sample week. Since the proportion of good trap nights in week 3 in August was 2 out of 7, 2/7, we would multiply these counts by 7/2.

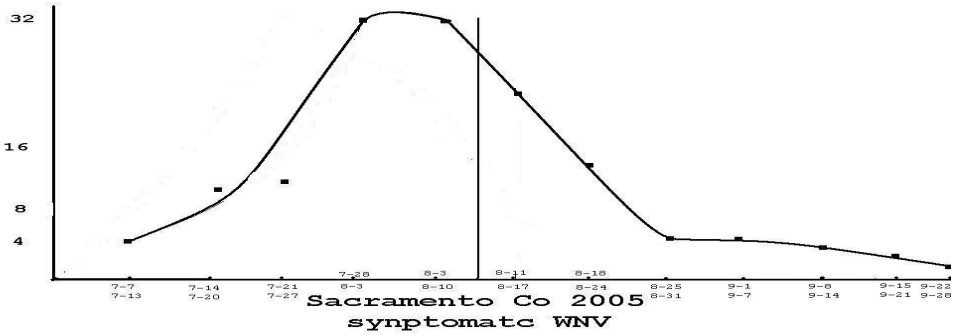
If the trap counts for the weeks of Aug 14 and Aug 21 are factored for the wind by multiplying by the reciprocal of the proportion of wind-free days in the week, the mosquito counts look like this:

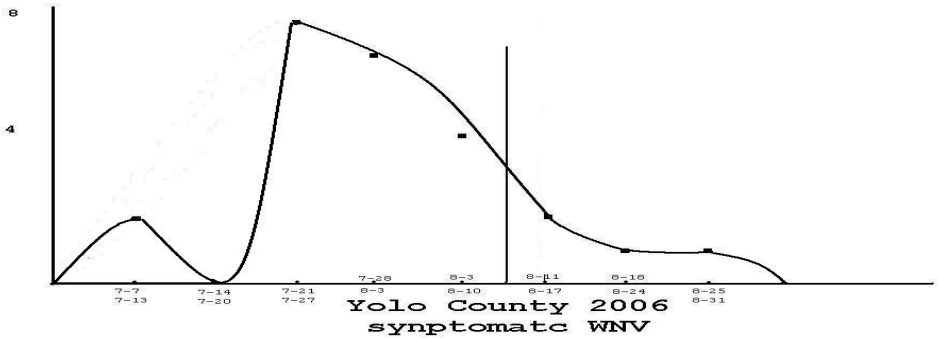


This shows an uninterrupted growth curve through August, with the daily increase falling right in the expected range. This suggests that this graph is more representative of the actual population dynamics, and the decline in numbers seen in the trap data for the weeks of the 14th and 21st represents the loss of sampling efficiency due to high wind. Any impact of aerial spray is so thoroughly hidden from the sample by the wind that it is hard to assess directly. However, due to the size the population reaches even after the spray, we can infer that the spray had no substantial effect. There is a 70 percent population increase in the three weeks from pre-spray Aug 7 to post-spray Aug 28. The declining population thereafter corresponds more closely to the cessation of irrigation in agriculture than to any other possible cause.

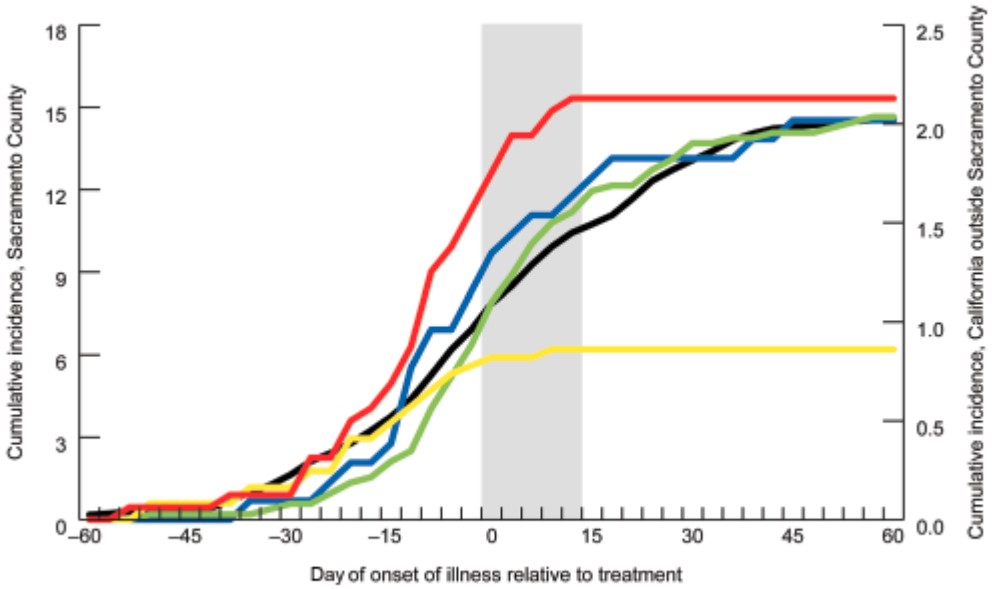
Simply put, for the mosquitoes to show the increase from beginning August to ending August of 2.7% per day there could not have been any great reduction of their numbers due to the aerial spray. And if the aerial spray didn't reduce the vector population it couldn't have been responsible for suspending epizootic transmission.

Sacramento 2005 and Yolo 2006 Timelines Confirm Pre-spray Decline. When the timelines of symptomatic cases in both Sacramento County in 2005 (CDPH 2006) and Yolo County in 2006 (CDPH 2007) are plotted against the spray date, it becomes clear that symptomatic cases were declining prior to spray (The vertical line is the point where the first possible effect of spray could appear).





Data in CDPH Report Demonstrate Pre-spray Decline. Even the curves presented in the Carney report show the rate of the epidemic declining prior to the spray dates.



The red line, northern treatment area, has its point of inflection some 9 days prior to spray; the yellow line, southern treatment, 6 days prior. The combined "buffer zones", blue line, appear to have begun declining at 9 days prior. Both the untreated areas and the rest of California, green and black, appear to have begun declining rates between 0 and 3 days after the spray date.

Spray Clearly Not Responsible for Epizootic Decline. A review of the 2005 CDPH case data (CDPH 2006) shows a peak in new case count for symptomatic disease in all of California on Aug. 1, with 37 cases showing onset of symptoms on that date. This was also the peak for Sacramento County with 15 of those 37 cases in Sacramento County. These reported cases are all 7 days prior to the initial spray date in Sacramento County and 10 days prior to the first possible impact of the spray. Since the virus requires a minimum of three days and as long as 14 days to express symptoms, the factors reducing the epizootic were already at play for at least 10 days ahead of the spray and at most 21 days ahead of it. Clearly, something other than spray was bringing the epizootic down throughout all of California as much as 3 weeks prior to the spray in Sacramento.

What is in the Future for WNv? It is not uncommon to hear expressions of concern about possible greatly increased rates of infection and numbers of serious cases of WNv disease in future years, when WNv "gets firmly established" in a region. Such concerns are often voiced prior to recommendations to spray adulticides to try to prevent this from happening. However, in addition to unproven assumptions about the efficacy of adulticiding, this concern reflects a fundamental misunderstanding of what is known scientifically about how the virus will develop in the coming years. When the virus is first introduced into a region there is no resistance in the bird population, since they have never before been exposed. Though many birds die in the initial exposure, the great majority of birds recover and are then permanently immune. When most of the birds in any given region have become immune, the potential pool of hosts for the virus is reduced and the total transmission declines commensurately. In a few years this will level off at a very slight amount, known as chronic endemicity, with many fewer infections in a given year than we have even now.

A good example of what this will be like is implicit in the related Western Equine Encephalitis virus, WEE, which has been chronically present in California for many years. This virus is more likely to produce a severe disease in people than WNv, but the levels of transmission are always so slight that it tends to be ignored by the public. Currently the WEE case average per year in California is 8 or less, but periodically there will be "outbreaks". These have happened at about 30 year intervals and may end up resulting in 70-100 cases statewide in peak outbreaks, such as in 1952 and 1987.

Health officials seem to be promulgating the view that the WNv will remain at a state of constant first introduction, ignoring the factor of the proportion of susceptible vs. immune birds, which actually dictates the potential transmission. This gives a skewed view to the potential risks, which are actually diminishing substantially because of chronic endemicity.

A more sensible approach to the WNv problem would entail an adjustment of control strategies to correspond with the diminishing risks as it declines over a number of seasons to

chronic endemicity. There is good reason to hypothesize that the continuous spray protocol will end up being counter productive to the public health, since unless the spray actually eliminates the virus in a given region it can only delay the attainment of chronic endemicity. This actually increases the time period where there is an elevated risk of human exposure rather than preventing exposure. Control measures should be adjusted to emphasize the safer and more effective larval control techniques that will have to be our permanent strategies for managing this disease.

Literature Cited

ASPH, "Aerial Spraying Effectively Reduces Incidence of West Nile Virus in Humans." Association of Schools of Public Health, ASPH Friday Letter, June 27, 2008.

Bowman, C et al "A mathematical model for assessing control strategies against West Nile virus." Bulletin of Mathematical Biology 2005 vol. 8:1107-1133

Carney, RM**.** et al, "Efficacy of Aerial Spraying of Mosquito Adulticide in Reducing Incidence of West Nile Virus, California, 2005". Emerging Infectious Diseases, May 2008 Vol. 14, No. 5:747-754

CDPH (2005) "2005 Summary of West Nile Virus Activity in Sacramento County" (web publication at CDPH)

CDPH (2006) "2005 Human WNV Case Linelist" (web publication at CDPH)

CDPH (2007) "2006 Human WNV Case Linelist" (web publication at CDPH)

Dow RP, Reeves WC, Bellamy RE. "Dispersal of female *Culex tarsalis* into a larvicided area". Am J Trop Med Hyg. 1965;14:656–70.

Gaulin C, Couillard M, Pilon PA, Tremblay M, Lambert L, Fradet MD, et al. "Assessment of surveillance of human West Nile virus infection in Quebec, 2003". Can Commun Dis Rep. 2004; 30:97–104.

Gillett, J. D. Mosquitoes (1971) Weidenfeld and Nicolson. London, 274 pp.

Greenwood, Michael, "Aerial Spraying Effectively Reduces Incidence of West Nile Virus in Humans," Yale School of Public Health, 2006.

Howard, Timothy (2006) Letter to Parents for a Safer Environment, incorrectly dated as 2005.

McCaffrey (2007) "Once Ominous, West Nile Wanes As Area Threat," Washington Post, July 30, 2007.

Moon, TE "A Statistical Model of the Dynamics of a Mosquito Vector (Culex tarsalis) Population". Biometrics 1976 vol. 32:355-368

Mostashari F, Bunning ML, Kitsutani PT, Singer DA, Nash D, Cooper MJ, et al. "Epidemic West Nile encephalitis, New York, 1999: results of a household-based seroepidemiological survey". Lancet. 2001;358:261–4.

Newton, EAC and Reiter, P "A Model of the Transmission of Dengue Fever with an Evaluation of the Impact of Ultra-Low Volume (ULV) Insecticide Applications on Dengue Epidemics". American Journal of Tropical Medicine and Hygiene, 47(6), 1992, pp. 709-720.

Northup, JO "A Response to the "Risk/Benefit" Analysis for Aerial Pesticide Release to Abate the Vectors of West Nile Virus" 2007, (web publication at Stop West Nile Spraying Now).

Pimentel, D. 1995. "Amounts of Pesticides Reaching Target Pests: Environmental Impacts and Ethics." Journal of Agricultural and Environmental Ethics 8(1):17-29.

Rawlins SC "Spatial distribution of insecticide resistance in Caribbean populations of *Aedes aegypti* and its significance". 1998 Rev Panam Salud Publica 4 (4) 243-251.

Reddy MR., Spielman A, Lepore TJ, Henley D,. Kiszewski AE, Reiter P. "Efficacy of Resmethrin Aerosols Applied from the Road for Suppressing Culex Vectors of West Nile Virus". Vector-Borne and Zoonotic Diseases. June 1, 2006, 6(2): 117-127

Reisen WK, Milby MM, Meyer RP. "Population dynamics of adult *Culex* mosquitoes (Diptera: Culicidae) along the Kern River, Kern County, California, in 1990". J Med Entomol. 1992;29:531–43.

Reisen, W.K. et al (2004) "West Nile Virus in California." (web publication at CDC page http://www.cdc.gov/ncidod/EID/vol10no8/04-0077.htm)

Reisen, W.K. et al (2006) "Role of Corvids in the epidemiology of West Nile virus." (web publication at CDC)

Rojas W, Botero S, Garcia HI. "An integrated malaria control program with community participation on the Pacific Coast of Colombia". Cad. Saúde Pública, Rio de Janeiro, 2001, 17(Suplemento):103-113

Ruiz MO, Tedesco C, McTighe TJ, Austin C, Kitron U. "Environmental and social determinants of human risk during a West Nile virus outbreak in the greater Chicago area, 2002". Int J Health Geogr. 2004;3:8. On-line at http://www.ij-healthgeographics.com/content/3/1/8

Theophilides CN, Ahearn SC, Grady S, Merlino M. "Identifying West Nile virus risk areas: the dynamic continuous-area space-time system". Am J Epidemiol. 2003;157:8:43-54.

Wonham MJ, de-Camino-Beck T, Lewis MA "An epidemiological model for West Nile virus invasion analysis and control applications". Proc. Royal Soc. London, B 2004 vol 271 501-507