HAWAIʻI CENTER FOR FOOD SAFETY

Review of Scientific Literature Relevant to Pesticide Buffer Zones

Executive Summary

Hawaiʻi is home to extensive cultivation of genetically engineered (GE) crops, primarily corn, which involves intensive use of pesticides. While overall pesticide use in Hawaiʻi is not known, sales data from the Hawaiʻi Department of Agriculture show that 123,000 pounds and 57,000 gallons of especially toxic "restricted use pesticides" (RUPs) were applied statewide in agriculture in 2013 (Enright 2014), the majority by five major pesticide/biotechnology firms. On Kauaʻi, one company (DuPont-Pioneer) sprays pesticides on 2 of every 3 days of the year; and makes on average 8.3-16 applications per application day (Jervis and Smith 2013). This intensive spraying far exceeds anything found on the mainland.

Hawaiʻi residents have legitimate reasons to be concerned about pesticide exposure. Many live quite near test fields, and pesticides frequently drift, particularly in windy conditions common in Hawaiʻi. In at least six episodes since 2006, dozens of teachers and schoolchildren have reported being sickened by chemicals, in all or most cases pesticides drifting from neighboring fields. Symptoms ranged from headaches, dizziness and respiratory distress to nausea and vomiting, some requiring treatment at hospitals (Leone 2008, Hillyer 2008, Kalani and Fujimori 2014). Because Hawaiʻi (unlike 11 other states) does not have a program to track pesticide poisoning incidents, these media reports likely represent a small fraction of actual cases.

Physicians concerned about pesticide drift in west Kauaʻi report that their patients are frequently afflicted with respiratory problems (including asthma), neurological impairment, and recurring nose bleeds and dermatitis, among other conditions (Kauaʻi Physicians 2013). More seriously, they suspect that pesticides may be responsible for the extremely high rates of rare cardiac birth defects and cancer in Westside residents (Raelson 2013).

There is a large and growing scientific literature on the health harms of pesticides. Farmers and children are at greatest risk. People are exposed to certain pesticides in their food and water; farmworkers take in pesticides via dermal contact and inhalation of spray. Pesticide drift represents an important additional exposure pathway (Goldman et al 2009). Medical science has established strong links between pesticide exposure and cancers (Schinasi and Leon 2014, Blair and Zahm 1995, Koutros et al. 2009, Mills et al 2005), Parkinson's disease (Priyadarshi et al. 2000, Brown et al. 2006, van den Mark et al. 2012), autism (Shelton et al. 2014, Roberts et al. 2007), attention-deficit hyperactivity disorder (Bouchard et al. 2010) and depression (Beard et al. 2014, Weisskopf et al. 2013, Bienkowski 2014), among other conditions.

It is well-established that the young are more susceptible to the harmful effects of pesticides than adults (National Research Council 1993, Roberts and Karr 2012), because even low-level exposure during fetal (Rull et al., 2009), neonatal (Chevrier et al., 2011) and infant life can disrupt critical developmental processes (Shelton et al., 2014). In an exhaustive review of the medical literature entitled Pesticide Exposure in Children (Roberts and Karr 2012), the American Academy of Pediatrics singles out childhood cancers (Infante-Rivard et al. 1999), neurological impacts such as reduced IQ (Rauh et al. 2011, Bouchard et al. 2011, Engel et al 2011), birth defects and other adverse birth outcomes (Garry et al. 1996, 2002), and asthma (Salam et al 2004) as the most important and best-supported impacts of pesticides on our children's health.

A study of pesticide exposure at schools shows that many children are exposed via pesticide drift from neighboring farmland (Alarcon et al 2005). Exposure to pesticide drift causes numerous short-term impacts that include headaches, dizziness, difficulty breathing, nausea, vomiting, weakness, chest pain, fatigue, rashes, and eye ailments (Owen and Feldman 2004, CA PISP 1992-2011). While it is often assumed that people suffer no permanent harm from a single (acute) pesticide exposure, research is proving that this is often not the case. For instance, scientists have found increased rates of depression (Stallones and Beseler 2002) and impaired cognitive functioning (Rosenstock et al 1991) in people exposed acutely to certain toxic pesticides. Longer-term exposure to drift is also hazardous. Epidemiological studies show greater risk of diseases such as autism (Roberts et al. 2007), Parkinson's disease (Costello et al. 2009) and childhood leukemia (Rull et al. 2009) in people living near agricultural fields sprayed with pesticides, suggesting that exposure via drift is responsible. Indeed, monitoring in California and Washington has found that airborne pesticide levels sometimes exceed acceptable health standards (Goldman et al. 2009).

In recognition of its failure to adequately prevent pesticide drift and its harms, EPA proposed stronger regulations in 2001 (EPA 2001), but these regulations were never finalized and are not in effect, leading public interest and farmworker groups to formally petition EPA to establish regulations to protect children from pesticide drift (Goldman et al 2009). The American Academy of Pediatrics and other medical scientists recognize pesticide drift as a health threat to children and recommend no-spray zones for schools (AAP 2012; Alarcon et al 2005). A growing number of states and counties have established no-spray buffer zones around schools, hospitals, nursing homes, public parks and playgrounds to protect their citizens and especially children from pesticide drift (Owens and Feldman 2004, CPR 2010, Hurley et al 2014).

In this review, Center for Food Safety summarizes the available information on the use and adverse impacts of pesticides in Hawai'i, and discusses some of the scientific literature on the impacts these pesticides can have on public health, particularly children. It is our hope that legislators will inform themselves about this serious health threat and establish buffer zones around schools to ensure the health and well-being of Hawai'i's keiki.

Pesticide Use and GE Crops

Hawai'i has had more field tests of genetically engineered crops than any other state (ISBa, 2014). In 2013 alone, 178 permits were issued for GE crop field tests on 1,124 sites statewide (ISBb, 2014). Because Hawai'i is much smaller than Midwestern states that have experienced nearly as many releases (e.g. Illinois), it has a much higher density of field tests. This means that many of the state's citizens live in dangerous proximity to pesticide-intensive GE crop fields.

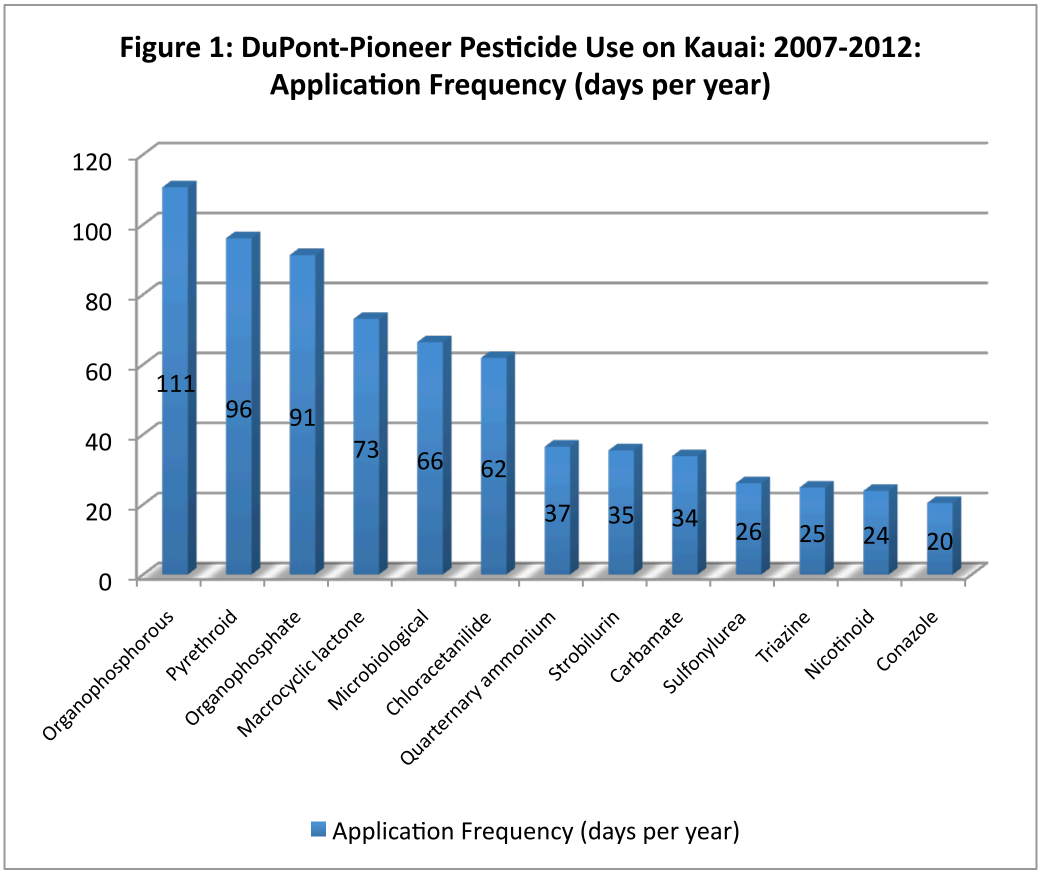
Corn is by far the dominant GE crop grown in the state, and it is produced for seed, not for food. Corn production involves large applications of pesticides and fertilizers, and growing GE corn for seed is still more chemical-intensive (Thomison undated). Many GE crops grown on Hawai'i are engineered to withstand heavy application of weed-killing pesticides (i.e. herbicides), increasing pesticide use and exposure still more. A recent study using data from U.S. Department to Agriculture pesticide use found that only the first 5 years of agriculture production of Herbicide Resistant (HR) crops showed a reduction in herbicide application. After 5 years GE crops had an increase of pesticide application. The sixth year of HR crop cultivation alone recorded an increase of pesticides that was double the cumulative amount which had been reported during the initial 5 year period (Benbrook, 2012). While in line with this study according to the USDA's National Agricultural Statistics Service, soybean producers have been steadily increasing their acreage GE crops since 1996. Herbicide use for soybean crops has also increased from 61 million pounds in 1996 to 133 million pounds in 2012 (USDA, 2014). Hawai'i's climate allows for multiple plantings of GE corn and other crops each year, facilitating year-round pesticide use.

Pesticide is a generic term for any chemical used to kill an unwanted pest, and it includes herbicides and fungicides as well as insecticides. Records obtained from DuPont-Pioneer in the context of a lawsuit on behalf of Kaua'i residents show that this single company sprayed pesticides on its GE corn fields in Kaua'i on two-thirds (65%) of the days from February 2007 to the end of 2012 (Jervis and Smith 2013); and that an annual average of 8.3 to 16 applications were made per application day in various years of this period. Even if one accounts for the fact that these applications were made to only portions of DuPont-Pioneer's overall test fields, this represents extremely intensive pesticide use. The third-most frequently applied group of pesticides was also among the most toxic: organophosphate insecticides such as chlorpyrifos (discussed below), which were sprayed on average 91 days each year (see Figure 1 and Table 1). The records also show that DuPont-Pioneer applied over 90 pesticide formulations containing 63 different active ingredients (Jervis and Smith 2013). In contrast, field corn on the mainland is treated much less intensively: while nearly all (98%) of field corn is treated with herbicides, only 8% and 12% of corn acres are sprayed with fungicides and insecticides, respectively (USDA NASS 2011).

EPA designates pesticides as either general use or restricted use. The restricted use category is reserved for pesticides whose "toxicity exceeds one or more … specific hazard criteria."[1] Due to the greater hazards they pose, restricted use pesticides (RUPs) can only be applied with protective equipment by those specially licensed to apply them, though it should be noted that general use pesticides can also be harmful. Sales data released by the

[1] See 40 CFR Part 152.170: Criteria for restriction to use by certified applicators.

Hawai'i Department of Agriculture show that "private applicators" applied 122,885 pounds and 57,067 gallons of restricted use pesticides (RUPs) in Hawai'i in 2013 (Table 2). Usage was particularly high in Maui and Oahu, though considerable quantities of RUPs were also applied on Kaua'i and the Big Island.[2]



Source: Jervis and Smith (2013). Chart based on data released by DuPont-Pioneer regarding its pesticide use practices on its GE crop test fields near the town of Waimea, Kaua'i. See Table 1 below for key.

Table 1: Key to DuPont-Pioneer Pesticides Used on Kau

Pesticide Class	Pesticide Type	Examples
Organophosphorous	Herbicide	Glyphosate, glufosinate
Pyrethroid	Insecticide	Permethrin, zeta-cypermethrin
Organophosphate	Insecticide	Chlorpyrifos
Macrocyclic lactone	Insecticide	Avermectin
Microbiological	Insecticide	Bacillus thuringiensis
Chloracetanilide	Herbicide	S-metolachlor, alachlor
Quaternary ammonium	Herbicide	Paraquat, diquat
Strobilurin	Fungicide	Azoxystrobin

[2] Sales figures for the various islands should correspond to usage on those islands, but some inter-island shipment of RUPs is possible (Personal communication, Thomas Matsuda, Pesticides Branch Manager, Hawai'i Dept. of Agriculture, 11/14/14).

Carbamate	Insecticide	Methomyl
Sulfonylurea	Herbicide	Chlorimuron
Triazine	Herbicide	Atrazine
Nicotinoid	Insecticide	Imidacloprid
Conazole	Fungicide	Propiconazole

TABLE 2: SALES OF RESTRICTED USE PESTICIDES (PRIVATE APPLICATORS) - 2013		
COUNTY	POUNDS PRODUCT	GALLONS PRODUCT
Island of Hawai'i	5,931.75	2,921.04
Island of Kaua'i	5,153.97	5,153.50
Island of Maui	101,736.85	5,871.14
Island of Molokai	0	529.00
Island of Oahu	10,062.12	42,592.56
TOTALS	122,884.69	57,067.24

Source: Enright (2014). According to Hawai'i Dept. of Agriculture Chairperson Scott Enright, the figures for "private applicators" presented here include at least some of the RUP use by "large farming corporations" and so the major pesticide-biotechnology firms on the islands. Enright (2014) also reports RUP figures for "commercial applicators," some of which are also used in agriculture (Personal communication, Thomas Matsuda, Pesticides Branch Manager, Hawai'i Dept. of Agriculture, 11/14/14). Thus, these RUP figures underestimate agricultural RUP use on the islands.

More nuanced sales data for Kaua'i show that 22 RUPs representing 18 active ingredients are applied in agriculture.[3] From 2010 to 2012, an average of 20,206 lbs. of agricultural RUPs were applied annually on Kaua'i. Eighty-one percent (81%) of RUPs by weight were applied to corn, 19% to coffee, with negligible amounts used on ornamentals, soybeans, sugarcane, tomatoes and turf. The major users of agricultural RUPs were Dow (and its subsidiary Agrigenetics), Syngenta, DuPont-Pioneer, Terminix International[4] and Kaua'i Coffee Company. See Figure 2 for breakdown by active ingredient.

ADVERSE IMPACTS OF PESTICIDES REPORTED IN HAWAI'I

Lessons from History

Many Hawai'i residents can remember incidents in our recent history when the public's health was threatened by pesticide exposure. For example, in 1980 drinking water wells in the Oahu town of Kunia were closed due to contamination with hazardous levels of several pesticides, including ethylene dibromide (EDB) and dibromochloropropane (DBCP), the latter a Dow Chemical nematicide infamous for causing sterility or impaired fertility in tens of thousands of farmworkers worldwide (Gonzalez and Loewenberg 2003). In 1982, milk on Oahu was found to be contaminated with hazardous levels of the pesticide heptachlor,[5] and elevated levels were also found in human breast milk (Smith 1982). All three pesticides were long used in pineapple production, and are today banned for food uses as probable human carcinogens[6] and for other health risks. Contamination of Kunia wells

---

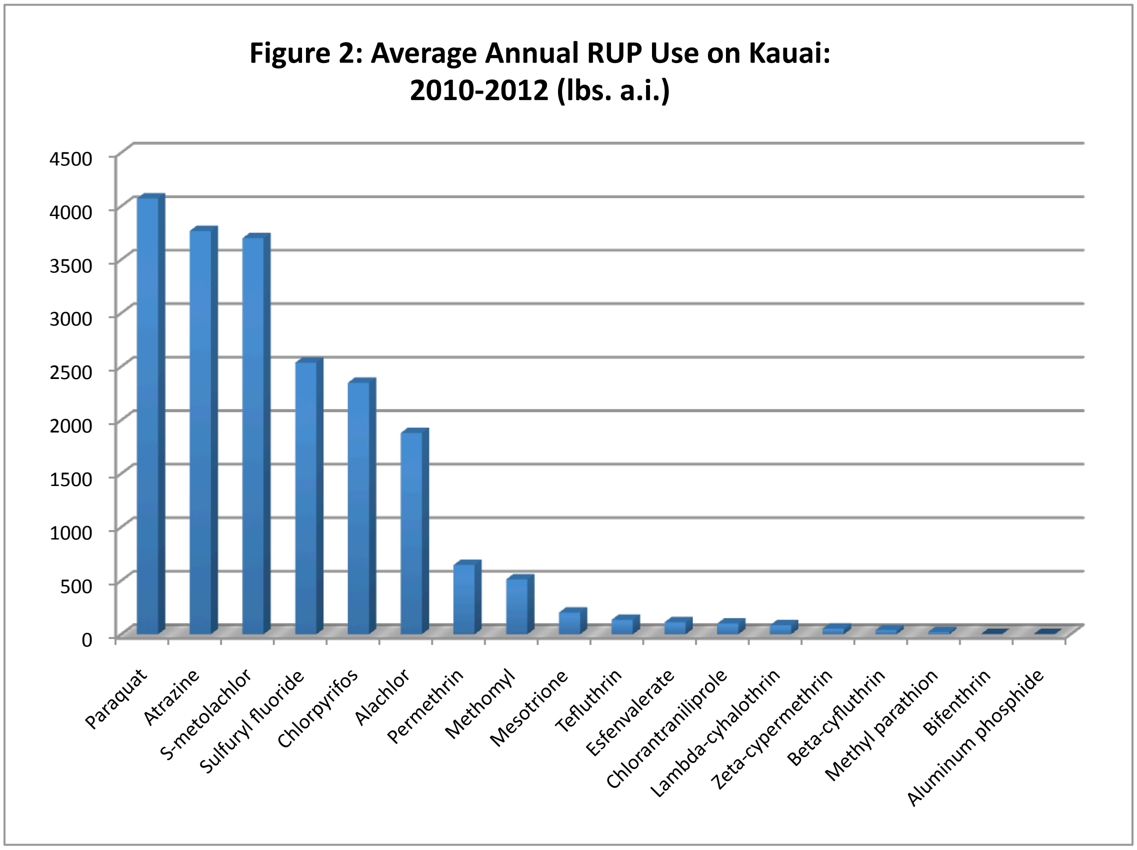
[3] The following figures are based on "Restricted Use Pesticides Sold on Kaua'i: 2010-2012," a spreadsheet obtained from Kaua'i County Council member Gary Hooser.

[4] Terminix is known for residential pest control (e.g. termites), but the Kaua'i records clearly show that Terminix purchased substantial amounts of RUPs for pest control in seed corn.

[5] Heptachlor was used in the pineapple industry. Heptachlor-contaminated pineapple leaves were fed to dairy cows.

[6] For DBCP, see http://www.epa.gov/ttnatw01/hlthef/dibromo-.html; for EDB, see http://www.epa.gov/ttnatw01/hlthef/ethyl-di.html; for heptachlor, see http://www.epa.gov/ttnatw01/hlthef/heptachl.html, last visited 11/12/14.

was so serious that EPA designated the area a Superfund site.[7] Researchers have found that exposure to DBCP and heptachlor is one likely factor driving increased breast cancer rates in Hawai'i (Allen et al. 1997).



Source: Based on "Restricted Use Pesticides Sold on Kaua'i: 2010-2012," a spreadsheet obtained from Kaua'i County Council member Gary Hooser. RUPs reported in gallons converted to pounds active ingredient based on EPA-approved labels for the respective pesticides. Because some pesticides are much more potent than others (applied at much lower rates), lesser use does not necessarily mean less concern for human health impacts.

These episodes teach important lessons. First, pesticides initially approved and used for many years as "safe" are found to be hazardous based on further scientific study. Second, powerful agricultural interests often succeed in keeping hazardous pesticides on the market even after their toxicity is well understood. For instance, even though EPA banned heptachlor in 1988,[8] it was used as late as 1993 in Del Monte pineapple fields (Allen et al. 1997); and while EPA banned DBCP in 1979, an exemption was granted for continued use on pineapples in Hawai'i until at least 1985.[9] Most disturbing, perhaps, is how Hawai'i officials have covered up pesticide contamination and denied clear health risks to citizens to protect agricultural interests (Smith 1982).

---

7 http://yosemite.epa.gov/r9/sfund/r9sfdocw.nsf/vwsoalphabetic/Del+Monte+Corp+(Oahu+Plantation)?OpenDocument, last visited 11/12/14.

8 http://www.epa.gov/ttnatw01/hlthef/heptachl.html, last visited 11/12/14.

9 http://www.epa.gov/ttnatw01/hlthef/dibromo-.html, last visited 12/12/14.

These lessons are still relevant today because hazardous pesticides continue to be applied today on the GE corn fields that have largely replaced the pineapple and sugarcane plantations. And these GE seed corn operations are run by some of the very firms that produced the plantation-era pesticides and long assured us of their supposed safety. While the crops and chemical names have changed, Hawai'i residents continue to be threatened by pesticides – not only in the water, but in the very air we breathe.

Pesticide drift in Hawai'i

Any pesticide can drift beyond the field where is it applied. Spray drift occurs during application, and is favored by windy conditions. Volatile pesticides like 2,4-D and dicamba are subject to vapor drift, which occurs when previously applied pesticides evaporate from plant and soil surfaces days to months after application (USGS 2003), and is more likely in hot, still conditions. Finally, pesticides can be carried in the wind as pesticide-laden dust. State pesticide officials receive over 2,000 complaints of pesticide drift each year, many involving crop damage (AAPCO 1999, 2005), but all agree this vastly underestimates the true scope of the problem.

There have been at least six episodes of pesticide-induced illness at schools since just 2006. Teachers and schoolchildren in Waimea have reported becoming sick on three separate occasions following chemical applications to a nearby seed corn plot (Leone 2008). In a 2008 episode, 60 children and at least two teachers experienced headache, dizziness, nausea and/or vomiting; 10 or more children were treated at an emergency room; several were put on a nebulizer to relieve respiratory distress; and one was given an anti-vomiting medication intravenously. A teacher who was also affected firmly rejected the explanation given by Hawai'i officials and Syngenta that "stinkweed" was the culprit, saying that she was familiar with stinkweed's odor and that this was not the cause (Leone 2008, Hillyer 2008).

At least three similar episodes have been reported on Oahu. In 2007, 15 students were sickened by pesticide drift at Kahuku Intermediate and High Schools, forcing closure of the school for three days, while other students reported ill effects from the use of the insecticide malathion at St. Joseph School in Waipahu in 2008 (Hillyer 2008, Leone 2008). In 2014, 31 students and staff at Kahaluu Elementary School experienced nausea, burning eyes, shortness of breath, dizziness, sore throat and coughing, and 26 were evacuated to and treated at nearby hospitals, due to a strong chemical odor that the Fire Department linked to reports of pesticide spraying in the area (Kalani and Fujimori 2014). These symptoms are all commonly reported effects of exposure to pesticides (AAP 2012: Table 2).

These media reports likely represent a small fraction of actual pesticide poisoning cases, for several reasons. First, as acknowledged by our EPA, "many [pesticide drift] incidents are unreported" (EPA 2001). Second, even when victims of pesticide poisoning do seek medical attention, California officials have found that "[p]hysicians often do not report potential pesticide illnesses" (CA PISP Fact Sheet), often because many physicians are ignorant of the effects of pesticide poisoning (AAP 2012). Finally, Hawai'i does not have a "pesticide poisoning surveillance program" of the sort established in 11 other states – California, Florida, Iowa, Louisiana, Michigan, Nebraska, New York, North Carolina, Oregon, Texas and Washington.[10] Such a program, if established in Hawai'i, would likely capture many more pesticide-induced illnesses.

Evidence from other states also suggests that pesticide drift is a frequent occurrence. A study of pesticide exposure at schools in eight states from 1998 to 2002 identified 2,593 individuals who had experienced acute pesticide-related illnesses. Of the 406 cases for which more detailed information was available, nearly one third (31%) involved pesticide drift from farmland while the others involved pesticide use at the school (Alarcon et al 2005). In a single year, seven pesticide drift cases involving school buses were reported in California's San Joaquin Valley (Khokha 2010). According to Teresa de Anda of Californians for Pesticide Reform, who has visited many rural communities in her state, drift has become "so commonplace that people don't report it" (as quoted in Khokha 2010).

Physicians concerned about pesticide drift in west Kaua'i encounter "almost daily reports of respiratory symptoms in patients that have no history of these respiratory illnesses…," and that many do not recover despite healthy lifestyle changes or pharmacological interventions. They also report recurring nose bleeds in children, recurring dermatitis, patients who experience a "metallic taste" in their mouths, as well as high levels of infertility and gout (Kaua'i Physicians 2013). Waimea residents are frequently afflicted with "fugitive dust" blowing into their town, which is downwind of a 1,000-acre DuPont-Pioneer seed corn operation (Jervis and Smith 2013). The dust comes so frequently and in such quantity that continual laborious cleaning is required, disrupting residents' lives. Windblown dust is also frequently a problem on Molokai. Extremely fine dust can penetrate into the lungs and cause bronchitis (CCOHS 2012). As the U.S. Geological Survey has observed, "pesticides can become airborne attached to wind-blown dust" (USGS 2003). Thus, pesticide-laden dust must be considered together with spray drift as well as water and food contamination when considering the health threats of pesticides near GE test fields.

Pesticides may also be a factor in still more serious health threats. Dr. James Raelson and his colleague Dr. Chatkupt, practicing pediatricians in Kaua'i, have noted an unusually high incidence of rare birth defects involving malformations of the heart in Kaua'i over the past seven years, at roughly ten times the national rate (Raelson 2014). They note that Hawai'i has not had surveillance for birth defects since 2005, and have called for unbiased epidemiology studies by the U.S. Centers for Disease Control and Hawai'i's Department of Health to better understand the causes. Kaua'i physicians and residents have also noted a "cancer cluster" in Waimea - 37 cases in a neighborhood of just 800 – which is said to be 10 times the statewide cancer rate. Although a one-page report by the Hawai'i Department of Health disputes the existence of a cancer cluster on Kaua'i, the author conceded that her analysis was inconclusive, and reportedly said: "If I lived there, it would concern me" (Skolnick 2013).

To our knowledge, there have been no official medical investigations of the episodes reported above. To have a chance of success, investigations of this sort must be conducted promptly after the episode occurs, be led by epidemiologists and other medical personnel, and involve detailed examination of victims and thorough assessments of possible causes. Chemists with the University of Hawai'i conducted an "air sampling" study at the Waimea school where the 2008 episode recounted above occurred (Li et al 2013). However, this investigation was begun more than two years after the fact; was conducted by chemists

[10] See http://www.cdc.gov/niosh/topics/pesticides/Statebase.html, last visited 11/12/14.

rather than physicians; involved no questioning or examination of victims; and perversely focused on stinkweed as the possible cause despite eyewitness testimony to the contrary. Some of the 24 pesticides measured are no longer in use; and conversely, no testing was conducted for the great majority of the 63 pesticide active ingredients known to be used on Kaua'i by DuPont-Pioneer (Jervis and Smith 2013). Thus, it is unsurprising that the results were inconclusive. However, the testing did detect the insecticide chlorpyrifos in the ambient air inside and outside the school, and exposure to organophosphate insecticides like chlorpyrifos is known to cause nausea, dizziness, respiratory distress and the other reported symptoms (AAP 2012: Table 2). Other pesticides detected in the ambient air inside and outside the school included bifenthrin, benzene hexachloride (BHC), and dichlorodiphenyltrichloroethanes (DDT). While the concentrations of these pesticides detected more than two years after the fact were thought to be below health concern limits, their presence demonstrates a troubling history of pesticide drift, and together with eyewitness evidence supports pesticides as the likely cause of the poisoning episodes on Kaua'i.

Even when victims appear to fully recover from short-term or "acute" pesticide exposures of this sort, they can have serious long-term health consequences, as discussed further below.

Health Impacts of Pesticide Exposure

Pesticides have a long history of having negative, often unforeseen, impacts on human health. People are exposed to certain pesticides in their food and water; farmworkers take in pesticides via dermal contact and inhalation of spray. Pesticide drift represents an important additional exposure pathway (Goldman et al 2009). In general, farmers and children are at greatest risk: farmers are more highly exposed than the general population; and children are more susceptible to the harmful effects of pesticides than adults.

Farmers and pesticide applicators

Age-adjusted cancer rates in the U.S. population nearly doubled (85% rise) from 1950 to 2001, corresponding to the period of rapid growth in use of pesticides and other industrial chemicals (Clapp et al. 2006). Significant associations between agricultural chemical use and cancer deaths have been found in 1,497 rural U.S. counties (Steingraber 2010, p. 64). National Cancer Institute scientists have found that farmers in the U.S. and elsewhere suffer from higher rates of certain cancers – including leukemia, non-Hodgkin's lymphoma, multiple myeloma, and brain cancer – than the general population, even though they have fewer cancers and are healthier overall (Blair and Zahm 1995).

These findings drove considerable research into potential causes, particularly pesticide exposure. Non-Hodgkin's lymphoma (NHL) is a terrible cancer of the immune system that kills 30% of those who contract it. A large number of studies have associated NHL with exposure to chlorophenoxy herbicides like 2,4-D (Zahm et al. 1990, Cantor et al. 1992, Blair and Zahm 1995, Mills et al. 2005); dicamba herbicide (Cantor et al. 1992, McDuffie et al., 2001); glyphosate (Hardell et al. 2002, De Roos et al. 2003, Schinasi and Leon 2014);

and to organophosphate insecticides (reviewed in Schinasi and Leon 2014). The findings involving 2,4-D and dicamba are especially concerning because GE crops resistant to these herbicides have been developed by Dow (2,4-D) and Monsanto (dicamba); they involve heavier and more frequent applications than are otherwise used (Mortensen et al. 2012); and these crops have been field-tested in Hawai'i (ISBc, 2014). In addition, both 2,4-D and dicamba are among the most drift-prone herbicides in use today, as evidenced by the large number of drift-related crop damage complaints attributable to them (AAPCO 1999, 2005). And if drift is damaging crops, people are likely being exposed as well.

Exposure to imidazolinone herbicides has been strongly associated with bladder and colon cancer in the Agricultural Health Study (Koutros et al. 2009), and BASF has field-tested imidazolinone-resistant corn and soybeans on Hawai'i (ISBc, 2014). Exposure to chlorpyrifos, a restricted use organophosphate insecticide heavily used in Hawai'i (see Figures 1 and 2, Table 1), has been linked to lung cancer (Lee et al. 2004), colorectal cancer (Lee et al. 2007), and non-Hodgkin's lymphoma (Schinasi and Leon 2014).

Many pesticides affect the nervous system. Thus, it is not surprising that a large body of research finds that pesticide exposure can trigger neurological disorders. Several major meta-analyses[11] have demonstrated a strong association between pesticides and Parkinson's disease. For instance, Priyadarshi et al. (2000) assessed 19 studies published between 1989 and 1999, and found that the majority reported that pesticide exposure elevated the risk of Parkinson's disease (PD). Brown et al. (2006) made similar findings, which were "strongest for exposure to herbicides and insecticides, and for long durations of exposure." A review by van den Mark et al. (2012) came to the same conclusions. Particular (classes of) pesticide implicated in PD include paraquat and rotenone (Tanner et al. 2011), chlorophenoxy herbicides (Brighina et al. 2008, Elbaz et al. 2009) and 2,4-D (Tanner et al. 2009).

Paraquat is one of the most heavily used RUPs on Kaua'i (Figure 1). Besides the association with PD, it is also one of the most acutely toxic herbicides in use, and is banned in 32 countries, including the European Union and Switzerland, home of Syngenta, its major producer (Watts 2010). It is responsible for thousands of deaths, both accidental poisonings and suicides (Watts 2010). While ingestion of as little as a teaspoon of concentrate is fatal, paraquat is 1,000-fold more toxic when inhaled (Ames et al. 1993) due to its extreme toxicity to lung tissue (McDonald et al., 1992). Paraquat also frequently causes topical injuries and may be associated with skin cancer (Wesseling et al. 2001). Kentucky agricultural extension agent Gordon Johnson reports that paraquat can drift for miles (Johnson 2008). Paraquat drift sickened dozens of people in a small California agricultural community, inducing respiratory distress, nausea and diarrhea, among other symptoms (Ames et al. 1993).

Several studies have also found a positive relationship between pesticide exposure and depression (reviewed in Bienkowski 2014, see also Beard et al. 2014). Higher rates of clinically diagnosed depression were found in both farmers with high cumulative exposure and those who reported pesticide poisoning (Beseler et al. 2008, Beseler and Stallones, 2008). A study in France found nearly double the rate depression in agricultural workers exposed to herbicides, with greater risk from longer-term exposure (Weisskopf et al. 2013). Bjorling-Poulsen et al. (2008) provide further information on the neurotoxicity of pesticides.

[11] A meta-analysis is a "study of studies." By assessing the findings of multiple studies for a particular disease outcome, more definitive conclusions can be reached than is possible with individual studies.

The troubling implication of many of these studies is that acute poisoning episodes can have chronic, long-term consequences for mental health.

Pesticides can also disrupt our hormonal or endocrine systems. Extremely low levels of atrazine, an herbicide categorized as a restricted use pesticide and heavily used on Hawai'i (Figure 2), have been shown to cause feminization of male frogs – a process described as chemical castration (Hayes et al 2011). Atrazine is a persistent compound (i.e. it breaks down slowly), and is one of the most frequently detected pesticides contaminating our water supply. In fact, an estimated 33 million Americans are exposed to atrazine in tap water (Duhigg 2009). Exposure to atrazine has been linked to reduced male fertility (Swan et al 2003) and the immune system cancer non-Hodgkin's lymphoma (De Roos et al. 2003). The European Union banned atrazine in 2003 on the basis of its persistence (Sass and Colangelo 2006). Atrazine's maker, Syngenta, has conducted a dirty tricks campaign in an attempt to discredit atrazine critics, and heavily lobbied the EPA to keep the herbicide registered in the U.S. (Howard 2013, Duhigg 2009). Atrazine's resistance to breakdown suggests that residues might also be present in the soil, and be carried with wind-blown dust. Atrazine is discussed further below.

Our Keiki at Risk

It is well-established that the young are more susceptible to the harmful effects of pesticides than adults (National Research Council 1993, Roberts and Karr 2012), for several reasons. First, infants and children are more highly exposed to pesticides, because they consume more food and water on a body-weight basis, and have a higher breathing rate, than adults. Secondly, children have greater hand-to-mouth activity, increasing opportunities for exposure to pesticide residues in dirt and dust. Finally, the immature, developing physiological systems of children are more susceptible to disease-causing disruption. This applies particularly to neurological impacts and cancer (NRDC 1997). Exposure of pregnant mothers to pesticides is particularly hazardous, since pesticides can be potent disruptors of fetal development.

The American Academy of Pediatrics (AAP) recently published a major report entitled "Pesticide Exposure in Children" that comprehensively reviewed 195 medical studies on the subject (see Roberts and Karr 2012). Among other impacts, their chief concerns were as follows:

1) ***Childhood cancers***, especially leukemia and brain tumors;
2) ***Neurobehavioral and cognitive deficits***, such as reduced IQ and attention deficit/hyperactivity disorder;
3) ***Adverse birth outcomes***, including preterm birth, low birth weight, and congenital anomalies; and
4) ***Asthma.***

We briefly discuss each of these impacts below, with reference to the AAP's comprehensive review.

<u>Childhood cancers:</u>

Five of six recent case-control studies found a statistically significant relationship between pesticide exposure and leukemia (see Roberts and Karr 2012, p. e1773-e1774). Two of the studies had the most detailed exposure assessment conducted to date, and found increasing risk with rising exposure, a strong indication that the observed associations are real. Maternal exposure to pesticides between the periods of preconception through pregnancy was the primary risk factor. Maternal use of either herbicides or insecticides was associated with nearly double the risk of childhood leukemia (Infante-Rivard et al. 1999). A meta-analysis provided additional support, also showing double the risk of leukemia in mothers exposed to pesticides while pregnant or while their children were young (Wigle et al. 2009). Monge et al. (2007) also found increased risk of leukemia in children borne to parents exposed occupationally to pesticides in Costa Rica.

Nine of the ten studies examining pesticides and brain cancer that have been conducted since 1998 demonstrated an increased risk estimate of brain tumors with maternal and/or paternal exposure to pesticides, though not all achieved statistical significance. Among the better quality studies, one that involved 321 cases demonstrated that maternal exposure to insecticides before or during pregnancy was associated with a 90% greater risk of astrocytoma (a type of brain cancer) in the child, as well as a trend to higher risk in exposed fathers (van Wijngaarden et al. 2003).

Neurobehavioral and cognitive deficits:

Exposure to many pesticides causes acute neurological symptoms, such as headaches and dizziness. However, a spate of recent studies is building an irrefutable case that long-term, low-level exposure to organophosphate insecticides (OPs) in early life (particularly *in utero*) has profoundly negative impacts on children's neurological development. The National Institutes of Health and the EPA are sponsoring three large-scale studies into this subject, two in urban settings and one in a rural community (see Roberts and Karr 2012, e1775-e1776). Women were enrolled during pregnancy, and their exposure to OPs carefully measured. Their children were tested for neurological development in the following years. What do the studies show? At two to four years of age, higher prenatal OP exposure was associated with "significantly poorer mental development," "pervasive developmental disorder," and in one group "increased scores for attention-deficit/hyperactivity disorder" (Eskenazi et al. 2007, Rauh et al. 2006). At seven years of age, kids more highly exposed to OPs in the womb had lower IQ scores in all three groups (Rauh et al. 2011, Bouchard et al. 2011, Engel et al 2011). Bouchard et al (2010) similarly found increased rates of attention-deficit/hyperactivity disorder in eight to 15-years olds whose urine had higher levels of OP breakdown products, a sign of greater exposure.

These findings are even more concerning when one considers the intensive use of chlorpyrifos in Hawai'i's seed corn operations, coupled with its propensity to drift. Records released by DuPont-Pioneer show the company sprays OPs on Kaua'i quite frequently, once every four days (91 days/year) (Figure 1). The OP insecticide chlorpyrifos is also one of the most heavily used RUPs on Kaua'i, with on average 2,350 lbs. applied each year (Figure 2). Air monitoring in California and Washington has found levels of chlorpyrifos exceeding health limits on several occasions (Goldman et al. 2009, pp. 9-10). An examination of California's Pesticide Illness Surveillance Program shows that chlorpyrifos was among the most frequently cited culprits in drift-related pesticide illnesses over the past two decades (CA PISP 1992-2011). The US Geological Survey has found "toxic rainfall" containing excessive levels of chlorpyrifos (for aquatic life) in California (USGS 2003). As noted above, air sampling at Waimea school consistently detected chlorpyrifos. Based on these multiple lines of evidence, there is every reason to expect that chlorpyrifos drift is adversely affecting the health of residents living near GE seed corn fields.

Adverse birth outcomes

The American Academy of Pediatrics is also concerned about the possible role of pesticides in triggering adverse birth outcomes (see Roberts and Karr 2012, e1776-e1778). Two studies in Minnesota have revealed a higher rate of birth defects in children fathered by male pesticide applicators in areas of the state where chlorophenoxy herbicides (e.g. 2,4-D) and fungicides are most heavily applied. These studies also found a seasonal effect, with children conceived in the spring, when herbicide use is heaviest, exhibiting the highest birth defect rates (Garry et al. 1996, Garry et al. 2002). Six additional studies described by Roberts and Karr (2012) found higher risk ratios for birth defects in mothers exposed to pesticides, with three of them showing statistically significant effects. A study of expectant mothers carried out in New York demonstrated an association between exposure to chlorpyrifos and reduced birth weight and length (Perera et al. 2003). Wolff et al. (2007) also found reduced birth weight in infants born to mothers exposed to OPs during pregnancy, but only in those children with a mutation that reduces their ability to detoxify OPs. Another study found that *in utero* exposure to OPs was associated with reduced gestation time (Eskenazi et al. 2004). Prenatal atrazine exposure has been associated with suppression of fetal growth (Chevrier et al 2011) and exposure to chlorophenoxy herbicides and certain other classes of herbicide, such as triazines (e.g. atrazine), with increased risk of spontaneous abortion (Arbuckle et al. 1999, 2001).

Asthma

The AAP also considers asthma to be a major adverse health outcome of pesticide exposure (see Roberts and Karr 2012, e1779). Asthma is the most common, chronic noninfectious disease of childhood, and is estimated to affect 300 million people worldwide, causing a quarter of a million deaths each year (Strina et al. 2014). Asthma is characterized by intermittent breathing difficulty, including chest tightness, wheezing, cough and shortness of breath. There have been few studies of pesticides and asthma in children, but those conducted raise serious concerns. For instance, exposure to either herbicides or insecticides in the first year of life was strongly linked to a diagnosis of asthma before the age of five in a study carried out in southern California – an over four-fold higher risk from herbicides and more than two-fold greater risk from insecticide exposure (Salam et al 2004). Studies of adults provide similar evidence. Farmers are at high risk of asthma and other respiratory diseases (Hoppin 2002), and exposure to organophosphate and carbamate insecticides has been linked to asthma in Canadian farmers (Senthilselvan et al. 1992). Two studies in the U.S. have associated exposure to a number of pesticides with wheezing, one of the major symptoms of asthma. Hoppin et al (2002) found a higher incidence of wheezing in farmers exposed to the herbicides atrazine, alachlor and paraquat, as well as the OP insecticides chlorpyrifos, parathion and malathion. All of these pesticides are used heavily and frequently in Hawai'i (Figures 1 and 2, Table 1). These findings take on added weight when one considers the testimony of Kaua'i physicians that Westside residents are very frequently afflicted with symptoms of respiratory distress.

Children may be exposed to and harmed by pesticides even when they are exposed only at second hand. For example, farmworkers exposed to pesticides may accumulate residues on their skin and clothing, and thereby inadvertently expose their families

(Thompson et al., 2003). Similarly, rural homes have much higher levels of pesticide residues in dust than non-rural residences (Simcox et al, 1995, 1999; Rull et al., 2009). These take-home pathways can contribute to children's exposure to pesticides in agricultural communities (Lu et al, 2000).

Health harms specifically linked to pesticide drift

The medical studies discussed above address the harms of pesticides from a variety of exposure pathways: food, water, dermal contact, inhalation and/or drift. Below, we discuss studies that specifically address health outcomes where drift is the presumed exposure pathway.

A growing body of research supports the proposition that living near pesticide-sprayed fields increases the risks of a number of serious diseases, and exposure via pesticide drift is the only logical explanation. Many of these studies have been conducted in California, which has an extremely fine-grained pesticide reporting system that provides precise information on which pesticides are sprayed near any given community, when, and in what amounts. Epidemiological studies based on this information have made some troubling findings. For instance, Costello et al. (2009) have found that exposure to paraquat and maneb within 500 meters of the home increased the risk of Parkinson's disease by 75%, with those under 60 years of age at higher risk. Roberts et al. (2007) conducted a similar analysis, and found that expectant mothers residing within 500 meters of fields sprayed with organochlorine insecticides (e.g. dicofol and endosulfan) during early pregnancy had a six-fold higher risk of bearing children with autism spectrum disorder than mothers not living near such fields; this ASD risk declined with increasing distance from field sites and increased with rising application amounts. Shelton et al. (2014) found a 60% increased risk of autism spectrum disorder (ASD) in children of mothers who lived near fields sprayed with organophosphate insecticides at some point during their pregnancies, with much higher risk when exposure occurred in the second trimester of their pregnancies. Similarly increased risk – for both ASD and developmental delay – was found for children of mothers near fields treated with pyrethroid insecticides just prior to conception or during their third trimester. Proximity to carbamate-treated fields was also linked to higher risk of developmental delay. These findings take on added significance when one considers that most of the insecticides at issue in this California study are used on Kaua'i and likely on other islands as well: one of the three organophosphates (chlorpyrifos); four of the five pyrethroid insecticides – permethrin, lambda-cyhalothrin, cypermethrin and esfenvalerate; and one of the two carbamates (methomyl) (see Figures 1 and 2, Table 1).

Why Buffer Zones?

Pesticide Drift Can Cause Lasting Harm

All of the symptoms reported above in Hawai'i's schoolchildren are among those typically caused by pesticide drift, which include headaches, dizziness, difficulty breathing, nausea, vomiting, weakness, chest pain, fatigue, rashes, and eye ailments (Owen and

Feldman 2004, CA PISP 1992-2011). It is often assumed that people suffer no permanent harm from a single (acute) pesticide exposure, but research is proving this to be untrue. For instance, many studies have found increased rates of lasting depression (e.g. Stallones and Beseler 2002, Beseler and Stallones 2008), impaired cognitive functioning (Rosenstock et al 1991), and reduced neuromuscular control (Kofman et al. 2006) in people exposed acutely to certain toxic pesticides. This means that children and others exposed just a single time to a pesticide, even though they may appear to fully recover, in certain cases go on to develop chronic, long-term illnesses that may persist throughout their lives.

EPA is Asleep at the Wheel

We would all like to believe that EPA protects us from pesticide harms. But sadly, this is often not the case. Above, we described numerous examples of EPA-approved pesticides that medical scientists have found to be hazardous, several of which are banned in other nations. Below, we recount some of the weaknesses in EPA's assessment process that lead to approval of hazardous products (for the following discussion, see Jacobs and Clapp 2008).

First, EPA requires testing only on the pesticide product's active ingredient (a.i.), even though it is well-known that so-called "inert ingredients"[12] in pesticide formulations that are actually used can be toxic in their own right, or increase the a.i.'s toxicity. Similarly, EPA assesses risks from exposure to only one a.i. at a time, even though in the real world we are exposed to multiple pesticides that can in some cases have additive or synergistic effects. Importantly, EPA relies almost entirely on animal experiments conducted by the financially interested pesticide company, and virtually ignores more relevant human epidemiological studies carried out by independent medical scientists. In addition, EPA approves hazardous pesticides based on the assumption that farmers and pesticide applicators will comply perfectly with exposure reduction measures (e.g. rubber gloves, boots, goggles, long-sleeve shirt), despite clear evidence that such measures are unrealistic and often not followed. Of course, schoolchildren and others exposed to drift have no way of implementing such protective measures.

As deficient as EPA regulation is generally, it provides still less protection in the case of pesticide drift. Though EPA has long required pesticide labels to include admonitions to applicators to avoid spray drift, as described above it is an extremely frequent occurrence. Despite rules that ostensibly prohibit application in windy conditions, the Association of American Pesticide Control Officials (AAPCO) "has experience that supports that there are numerous pesticide applications made when it is too windy" (AAPCO 2002). Neither does EPA take drift exposure into account when it registers or re-registers individual pesticides. Finally, EPA's very definition of drift is deficient, in that it leaves out vapor drift and pesticide-laden dust, considering only the form of drift that occurs during application.[13]

[12] In this context, "inert" means non-toxic to the target pest, and says nothing about the ingredient's toxicity to people or the environment.

[13] EPA's definition: "Spray or dust drift is the physical movement of pesticide droplets or particles through the air at the time of pesticide application or soon thereafter from the target site to any non-, or off-target site. Spray drift shall not include movement of pesticides to non- or off-target sites caused by erosion, migration, volatility, or windblown soil particles that occurs after application or application of fumigants unless specifically addressed on the product label with respect to drift

One clear example of EPA's deficient regulation is chlorpyrifos, the organophosphate (OP) insecticide used heavily in Hawai'i, and implicated in many of the serious health threats described above. EPA knows that chlorpyrifos is toxic, which explains why it began a phase-out of residential uses of the insecticide in the year 2000 (but not agricultural uses), specifically to protect children (Goldman et al 2009, pp. 12, 15-19). Yet EPA has left rural kids unprotected, even though ambient air levels of chlorpyrifos have been found to exceed health standards in agricultural areas, as discussed above. This sets up an unfortunate double standard. Urban and suburban kids are protected from the health harms of chlorpyrifos, but rural kids are not. In Hawai'i, GE crop field tests often occur so close to populated areas that people in both rural areas and towns are at risk of drift exposure to this and other toxic pesticides.

Momentum Building to Protect Kids from Pesticide Drift

In a failed attempt to better protect human health and the environment from drift, EPA proposed improved pesticide labeling in 2001 (EPA 2001), but this proposal was never finalized and is not in effect. In an important sign of the times, public interest and farmworker groups formally challenged EPA for its inaction, and petitioned the Agency to establish regulations to protect children from pesticide drift. This petition, entitled Pesticides in the Air – Kids at Risk, provides further information documenting the harms from this neglected health threat (Goldman et al 2009). We have also discussed in this paper a seminal review of the medical literature on the threat of pesticides to children's health by the premier organization representing our nation's pediatricians, the American Academy of Pediatrics (Roberts and Karr 2012). AAP has also released an official policy statement based on this review that makes specific recommendations to mitigate health threats from pesticides (AAP 2012). Among the local policy approaches listed are the establishment of no-spray buffer zones around schools, as well as posting warning signs of pesticide use and restricting specific types of pesticides in schools. Medical scientists from the federal and state governments, writing in the prestigious Journal of the American Medical Association, also support "adoption of pesticide spray buffer zones around schools" (Alarcon et al 2005).

As of 2004, at least seven states had established no-spray buffer zones around schools, hospitals, nursing homes, public parks and playgrounds (Owens and Feldman 2004). More recent information shows that nine states (Hurley et al. 2014) and 14 counties in California (CRP 2010) have established similar no-spray zones. States with notification requirements for pesticide applications near schools have increased in number from eight in 2004 to 11 today (Owens and Feldman 2004, Hurley et al 2014). These policy actions evince growing awareness of the serious health threats posed by pesticide drift.

Conclusion

Experience on Kaua'i and Oahu show that pesticide drift is a real threat causing real harms to the state's citizens, particularly our keiki, who are particularly vulnerable. The

control requirements." See http://www.epa.gov/PR_Notices/prdraft-spraydrift801.htm, last visited 7/14/13.

medical evidence of short- and serious long-term health impacts from exposure to pesticides, including pesticide drift, is overwhelming, confirmed by leading scientists and pediatricians. Equally clear is that neither federal nor state authorities have taken the actions needed to address this insidious health threat. Clearly, many reforms and initiatives are necessary. EPA must do a better job of assessing pesticides; states require more personnel and expertise to better regulate them; and physicians must be better trained to recognize pesticide-induced illnesses in their patients. In the meantime, we must reduce the exposure of our citizens to these hazardous compounds.

We respectfully urge you, our state's legislators, to join the growing movement of physicians and state and local officials to protect our keiki from pesticide drift by passing the Buffer Zones Bill. We would be happy to answer any questions you may have.

References

AAP 2012. Pesticide exposure in children. Policy Statement, American Journal of Pediatrics, Council on Environmental Health. Pediatrics 130(6): e1757-e1763

AAPCO 2002. Letter from Donnie Dippel, President of AAPCO, to Jay Ellenberger of EPA, March 25, 2002.

AAPCO 1999, 2005. "1999 and 2005 Pesticide Drift Enforcement Surveys," Association of American Pesticide Control Officials, 2005. http://www.aapco.org/documents/surveys/drift99.html and http://www.aapco.org/documents/surveys/DriftEnforce05Rpt.html.

Alarcon WA et al. 2005. Acute illnesses associated with pesticide exposure at schools. Journal of the American Medical Association 294(4): 455-465.

Allen, R. H., Gottlieb, M., Clute, E., Pongsiri, M. J., Sherman, J., and Obrams, G. I. 1997. Breast Cancer and Pesticides in Hawai'i: The Need for Further Study. *Environmental Health Perspectives*. 105(Supplement 3): 679-683.

Ames RG, Howd RA, Doherty L. 1993. Community Exposure to a paraquat drift. Archives of *Environmental Health Perspectives*, 48 (1) 47-52.

Arbuckle T. E., Lin Z., and Mery L. S. 2001. An Exploratory Analysis of the Effect of Pesticide Exposure on the Risk of Spontaneous Abortion in an Ontario Farm Population. *Environmental Health Perspectives,* 109(8): 851-857.

Arbuckle TE, Savitz DA, Mery LS, Curtis KM. 1999. Exposure to phenoxy herbicides and the risk of spontaneous abortion. Epidemiology. 10(6): 752–760.

Beard J. D., Umabach D. M., Hoppin J. A. Richards M., Alavanja M. C. R., Blair A., Sandler D. P., and Kamel, F. 2014. Pesticide Exposure and Depression among Male Private Pesticide Applicators in the Agricultural Health Study. *Environmental Health Perspectives*, 122(9): 984-991.

Beseler CL, Stallones L, Hoppin JA, Alavanja MC, Blair A, Keefe T, Kamel F. 2008. Depression and pesticide exposures among private pesticide applicators enrolled in the Agricultural Health Study. Environ Health Perspect 116(12): 1713-9.

Beseler CL, Stallones L. 2008. A cohort study of pesticide poisoning and depression in Colorado farm residents. Ann Epidemiology 18:768–774

Bienkowski B. 2014. Pesticide use by farmers linked to high rates of depression, suicides. Environmental Health News, October 6, 2014. http://www.environmentalhealthnews.org/ehs/news/2014/oct/pesticides-depression/.

Bjorling-Poulsen M, Andersen HR, Grandjean P. 2008. Potential developmental toxicity of pesticides used in Europe. Environmental Health. 7:50.

Blair A., Zahm S.H., Pearce N.E., Heineman E.F., Fraumeni J.F. Jr. 1992. Clues to Cancer Etiology from Studies of Farmers. *Scandinavian Journal of Work, Environment & Health,* 18: 209-215.

Blair A. and Zahm S.H. 1995. Agricultural exposures and cancer. *Environmental Health*

Perspective, 103(suppl 8): 205–8.

Bouchard MF, Chevrier J, Harley KG, et al. 2011. Prenatal exposure to organophosphate pesticides and IQ in 7-year-old children. Environ Health Perspect. 119(8): 1189–1195.

Bouchard MF, Bellinger DC, Wright RO, Weisskopf MG. 2010. Attention-deficit/hyperactivity disorder and urinary metabolites of organophosphate pesticides. Pediatrics. 125(6). Available at: www.pediatrics.org/cgi/content/full/125/6/e1270.

Boyd, D. R. 2006. The Food We Eat: An International Comparison of Pesticide Regulations. David Suzuki Foundation, 2006.

Brighina L, Frigerio R, Schneider NK, Lesnick TG, de Andrade M, Cunningham JM, Farrer MJ, Lincoln SJ, Checkoway H, Rocca WA, Maraganore DM. 2008. "Alpha-synuclein, pesticides, and Parkinson disease: A case–control study," Neurology 70(16 pt 2): 1461–1469.

Brown, TP et al. 2006. "Pesticides and Parkinson's Disease – Is There a Link?" Environmental Health Perspectives 114(2): 156-164.

CA PISP 1992-2011. Based on symptoms reported in records obtained from a search of agricultural drift episodes in California from 1992-2011. CA Pesticide Illness Surveillance Program Illness Query database, search conducted 7/12/14. http://www.cdpr.ca.gov/docs/whs/pisp.htm.

CA PISP Fact Sheet. Preventing pesticide illness. California's Pesticide Illness Surveillance Program. CA Dept. of Pesticide Regulation.

Cantor, K.P.; Blair, A.; Everett, G.; Gibson, R.; Burmeister, L.F.; Brown, L.M.; Schuman, L.; Dick, F.R. 1992. Pesticides and other agricultural risk factors for non-Hodgkin's lymphoma among men in Iowa and Minnesota. Cancer Res. 52: 2447–2455.

CCOHS 2012. What are the effects of dust on the lungs? Canadian Centre for Occupational Health and Safety, October 1, 2012.

Chevrier C., Limon G., Monfort C., Rouget F., Garlantezec R., Petit C., Durand G., and Cordier A. 2011. Urinary Biomarkers of Prenatal Atrazine Exposure and Adverse Birth Outcomes in the PELAGIE Birth Cohort. *Environmental Health Perspectives*, 119(7): 1034-1041.

Clapp, RW et al. 2006. "Environmental and occupational causes of cancer revisited," Journal of Public Health Policy 27(1): 61-76.

Costello, S. 2009. Parkinson's Disease and Residential Exposure to Maneb and Paraquat From Agricultural Applications in the Central Valley of California. *American Journal of Epidemiology*, 169(8): 919-926.

CPR 2010. Pesticide Protection Zones: Keeping Kids Safe at School. Californians for Pesticide Reform, Pesticide Watch, Center for Environmental Health, March 2010.

De Roos A. J., Zahm S. H., Cantor K. P., Weisenburger D. D., Holmes F. F., Burmeister L. F., and Blair A. 2003. Integrative Assessment of Multiple Pesticides as Risk Factors for Non-Hodgkin's Lymphoma among Men. Journal of Occupational Medicine, 60(11). Available at: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1740618/

Duhigg, C. 2008. Debating how much weed killer is safe in your water glass. The New York Times, August 23 2008. http://www.nytimes.com/2009/08/23/us/23water.html?_r=1&pagewanted=print

Elbaz A, Clavel J, Rathouz PJ, Moisan F, Galanaud JP, Delemotte B, Alperovitch A, Tzourio C. 2009. "Professional exposure to pesticides and Parkinson disease," Annals of Neurology 66(4): 494–504.

Engel SM, Wetmur J, Chen J, et al. 2011. Prenatal exposure to organophosphates, paraoxonase 1, and cognitive development in childhood. Environ Health Perspect. 119(8): 1182–1188.

Enright, S. 2014. Information Requested at House Committee on Agriculture on January 27, 2014 [Memorandum]. Honolulu, HI: Department of Agriculture.

EPA 2001. Pesticide Registration (PR) Notice 2001-X Draft: Spray and Dust Drift Label Statements for Pesticide Products. Office of Pesticide Programs, Environmental Protection Agency, 2001.

Eskenazi B, Marks AR, Bradman A, et al. 2007. Organophosphate pesticide exposure and neurodevelopment in young Mexican-American children. Environ Health Perspect. 115(5): 792–798.

Eskenazi B, Harley K, Bradman A, et al. 2004. Association of in utero organophosphate pesticide exposure and fetal growth and length of gestation in an agricultural population. Environ Health Perspect. 112(10): 1116–1124.

Goldman P, Brimmer JK and Ruiz V. 2009. "Pesticides in the Air – Kids at Risk: Petition to EPA to protect children from pesticide drift," Earth Justice and Farmworker Justice, October 2009.

Gonzalez D, Loewenberg S. 2003. Banana workers get day in court. New York Times, January 18, 2003.

Garry VF, Harkins ME, Erickson LL, Long-Simpson LK et al. 2002. Birth Defects, Season of Conception, and Sex of Children Born to Pesticide Applicators Living in the Red River Valley of Minnesota, USA. Environmental Health Perspectives 110 (Suppl. 3): 441-449.

Garry V.F., Schreinemachers D., Harkins M. E., and Griffith J. 1996. Pesticide appliers, biocides, and birth defects in rural Minnesota. *Environmental Health Perspective*, 104 (4), 394-399.

Hardell, L.; Eriksson, M.; Nordstrom, M. 2002. Exposure to pesticides as risk factor for non-Hodgkin's lymphoma and hairy cell leukemia: Pooled analysis of two Swedish case-control studies. Leuk. Lymphoma 43: 1043–1049.

Hayes T. 2011. Demasculinization and Feminization of Male Gonads by Atrazine: Consistent Effects Across Vertebrate Classes. *Journal of Steroid Biochemistry & Molecular Biology,* 127: 64-73.

Hillyer B. 2008. Lawmakers concerned about pesticide spraying. Hawai'i News Now, February 5, 2008. http://www.Hawai'inewsnow.com/story/7822628/lawmakers-concerned-about-pesticide-spraying.

Hoppin JA, Umbach DM, London SJ, Alavanja MCR, Sandler DP. 2002. Chemical predictors of wheeze among farmer pesticide applicators in the Agricultural Health Study. Am. Journal of Respiratory and Critical Care Medicine 165: 683-689.

Howard, C. 2013. "Syngenta's campaign to protect atrazine, discredit critics," Environmental Health News, June 17, 2013. http://www.environmentalhealthnews.org/ehs/news/2013/atrazine.

Hurley JA et al. 2014. Regulating pesticide use in United States Schools. American Entomologist 60(2): 105-114.

Infante-Rivard C, Labuda D, Krajinovic M, Sinnett D. 1999. Risk of childhood leukemia associated with exposure to pesticides and with gene polymorphisms. Epidemiology 10(5): 481-487.

ISBa 2014. Information Systems for Biotechnology. A National Resource in Agbiotech Information. 2014. Top Ten Most Frequent Locations for Crop Release (Permits and Notifications). Retrieved 10/21/14 from http://www.isb.vt.edu/release-summary-data.aspx

ISBb 2014. Information Systems for Biotechnology. A National Resource in Agbiotech Information. 2014. Locations and site charts by year: 2013 data [Chart]. Retrieved 10/17/14 from http://www.isb.vt.edu/locations-by-years.aspx

ISBc 2014. Information Systems for Biotechnology, A National Resource in Agbiotech Information. 2014. Conduct searches for the respective companies and the "phenotype category" herbicide-tolerance (HT). http://www.isb.vt.edu/search-release-data.aspx.

Jacobs M & Clapp S. 2008. "Agriculture and Cancer: A Need For Action," October 2008. http://www.sustainableproduction.org/downloads/AgricultureandCancer_001.pdf

Jervis, G. and Smith, K. 2013. Presentation by plaintiffs' attorneys in lawsuit by Waimea, Kaua'i residents against Pioneer, DuPont. July 13, 2013. http://vimeo.com/70580803.

Johnson G. 2008. Watch spray drift. Kent County Agriculture Cooperative Extension, University of Delaware, May 4, 2008. http://extension.udel.edu/kentagextension/tag/paraquat/.

Kalani N. and Fujimori L. 2014. Dozens of Kahaluu students sickened by fumes at campus. Honolulu Star-Advertiser, April 4, 2014. http://www.staradvertiser.com/s?action=login&f=y&id=253867411&id=253867411.

Kaua'i Physicians 2013. Letters from Kaua'i physicians to Kaua'i Mayor Carvalho, October 2013. http://www.stoppoisoningparadise.org/#!doctors-and-nurses-letters-to-mayor/cs1m.

Kofman O, Berger A, Massarwa A, Friedman A, Jaffar AA. 2006. Motor inhibition and learning impairments in school-aged children following exposure to organophosphate pesticides in infancy. Pediatr Res. 60(1): 88–92.

Koutros S., Lynch C. F., Ma X., Lee W. J., Hoppin J. A., Christensen C. H., Andreotti G., Freeman L. B., Rusiecki J. A., Hou L., Sandler D. P., and Alavanja M. C. R. 2009. Heterocyclic Aromatic Amine Pesticide Use and Human Cancer Risk: Results from the U.S. Agricultural Health Study. *International Journal of Cancer*, 124(5): 1206-1212.

Lee WJ, Sandler DP, Blair A, Samanic C, Cross AJ, Alavanja MC. 2007. Pesticide use and colorectal cancer risk in the Agricultural Health Study. Int J Cancer. 121(2): 339-46.

Lee WJ, Blair A, Hoppin JA, Lubin JH, Rusiecki JA, Sandler DP, Dosemeci M, Alavanja MC. 2004. Cancer incidence among pesticide applicators exposed to chlorpyrifos in the Agricultural Health Study. J Natl Cancer Inst. 96(23): 1781-9.

Leone D. 2008. Odor that got kids sick debated. Honolulu Advertiser, February 24, 2008. http://the.honoluluadvertiser.com/article/2008/Feb/24/ln/Hawai'i802240350.html.

Li, Q. X., Wang J., and Boesh R. 2013. Final Project Report for Kaua'i Air Sampling Study. University of Hawai'i Department of Molecular Bioscience and Bioengineering

Lu C, Fenske RA, Simcox NJ, Kalman D. 2000. Pesticide exposure of children in an agricultural community: evidence of household proximity to farmland and take home exposure pathways. Environ Res. 84: 290–302.

McDonald S. A., Wamsley M. A., Victor J. K., Hoffelt M., and Funt R. C. 1992. Applying Pesticides Correctly – A guide or private and commercial applicators. Ohio State University:

McDuffie HH, Pahwa P, McLaughlin JR, Spinelli JJ, Fincham S, Dosman JA, et al. 2001. Non-Hodgkin's lymphoma and specific pesticide exposures in men: cross-Canada study of pesticides and health. Cancer Epidemiology Biomarkers Prev 10:1155-1163.

Mills PK, Yang R, Riordan D. 2005. Lymphohematopoietic Cancers in the United Farm Workers of America (UFW), 1988-2001. Cancer Causes & Control 16(7): 823-830.

Monge et al, 2007. Parental occupational exposure to pesticides and the risk of childhood leukemia in Costa Rica. Scand J Work Environmental Health 33(4): 293-303.

Mortensen DA, Egan JF, Maxwell BD, Ryan MR, Smith RG. 2012. "Navigating a Critical Juncture for Sustainable Weed Management," Bioscience 62(1): 75-84.

National Research Council. 1993. Pesticides in the Diets of Infants and Children. National Academy Press: Washington D.C.

NRDC 1997. Our Children At Risk: The Five Worst Environmental Threats to Their Health, Chapter 5. By Lawrie Mott, David Fore, Jennifer Curtis, Gina Solomon. National Resources Defense Council. November 1997. http://www.nrdc.org/health/kids/ocar/chap5.asp

Owens, K and Feldman J. 2004. "Getting the drift on chemical trespass: Pesticide drift hits homes, schools, and other sensitive sites throughout communities," Pesticides and You, Vol. 24, No. 2: 16-21.

Perera FP, Rauh VA, Tsai WY, et al. 2003. Effects of transplacental exposure to environmental pollutants on birth outcomes in a multiethnic population. Environ Health Perspect. 111(2): 201–205.

Priyadarshi A, Khuder SA, Schaub EA, Shrivastava S. 2000. "A meta-analysis of Parkinson's disease and exposure to pesticides," Neurotoxicology 21(4): 435-40.

Raelson, J. 2013. "Subject: Birth Defects West Side Babies." Email testimony to Kaua'i County Council for Bill 2491, September 12, 2013.

Rauh V, Arunajadai S, Horton M, et al. 2011. Seven-year neurodevelopmental scores and prenatal exposure to chlorpyrifos, a common agricultural pesticide. Environ Health Perspect. 119(8): 1196–1201.

Rauh VA, Garfinkel R, Perera FP, et al. 2006. Impact of prenatal chlorpyrifos exposure on neurodevelopment in the first 3 years of life among inner-city children. Pediatrics. 118(6): e1845-e1859. Available at: www.pediatrics.org/cgi/content/full/118/6/e1845.

Roberts E. M., English P. B., Grether J. K., Windham G. C., Somberg L., and Wolf C. 2007. Maternal Residence near Agricultural Pesticide Applications and Autism Spectrum Disorder among Children in the California Central Valley. *Environmental Health Perspectives,* 115(10): 1482-1489.

Roberts J. R. and Karr C. J. 2012. Pesticide Exposure in Children. Council on Environmental Health. *Pediatrics*, 130(6): e1757-e1763. Available at: http://pediatrics.aappublications.org/content/130/6/e1757.full.html

Rosenstock L, Keifer M, Daniell WE, McConnell R, Claypole K. 1991. Chronic central nervous system effects of acute organophosphate pesticide intoxication. Lancet 338: 223–227.

Rull R. P., Gunier R., Von Behren J., Hertz A., Crouse V., Buffler P. A., and Reynolds P. 2009. Residential Proximity to Agricultural Pesticide Applications and Childhood Acute Lymphoblastic Leukemia. *Environmental Research*, 109(7): 891-899.

Salam MT, Li YF, Langholz B, Gilliland FD; Children's Health Study. 2004. Early-life environmental risk factors for asthma: findings from the Children's Health Study. Environ Health Perspect. 112(6): 760–765.

Sass, JB and Colangelo, A (2006). European Union bans atrazine, while the United States negotiates continued use. Int. J. Occup. Environ. Health 12(3): 260-267.

Schinasi L, Leon ME. 2014. Non-Hodgkin lymphoma and occupational exposure to agricultural pesticide chemical groups and active ingredients: a systematic review and meta-analysis. Int. J. Environ. Public Health 11: 4449-4527.

Senthilselvan A, McDuffie HH, Dosman JA. 1992. Association of asthma with use of pesticides—results of a cross-sectional survey of farmers. Am Rev Respir Dis 146: 884–7.

Shelton, JF et al. 2014. Neurodevelopmental Disorders and Prenatal Residential Proximity to Agricultural Pesticides: The CHARGE Study. *Environmental Health Perspective*, 122(10): 1103-1110.

Simcox NJ, Camp J, Kalman D, et al. 1999. Farmworker exposure to organophosphorous pesticide residues during apple thinning in central Washington State. *Am Ind Hyg Assoc J.* 606: 752–61.

Simcox NJ, Fenske RA, Wolz SA, Lee IC, Kalman DA. 1995. Pesticides in household dust and soil: exposure pathways for children of agricultural families. *Environmental Health Perspective.* 103: 1126–34.

Skolnick, A. 2013. "GMOs are Tearing a Tropical Paradise Apart." *Salon*, September 5. 2013. http://www.salon.com/2013/09/04/a_battle_in_paradise_how_gmos_are_tearing_a_tropical_utopia_apart/

Smith, JR. 1982. Hawaiian Milk Contamination Creates Alarm. *Science* 217: 137-140.

Stallones L and Beseler C. 2002. Pesticide poisoning and depressive symptoms among farm residents. Annals of Epidemiology 12: 389-394.

Steingraber, S. 2010. Living Downstream, Da Capo Press, 2nd edition, 2010.

Strina A, Barreto ML, Cooper PH, Rodrigues LC. 2014. Risk factors for non-atopic asthma/wheeze in children and adolescents: a systematic review. Emerging Themes in Epidemiology 11:5. http://www.ete-online.com/content/11/1/5.

Swan S. H., Kruse R. L., Liu F., Barr D. B., Drobnis E. Z., Redmon J. B., Wang C., Brazil C., Overstreet J. W., and the Study for Future Families Research Group. 2003. Semen Quality in Relation to Biomarkers of Pesticide Exposure. *Environmental Health Perspectives* 111(12): 1478-1484.

Tanner et al. 2011. Rotenone, paraquat and Parkinson's disease. Environmental Health Perspectives 119(6): 866-872.

Tanner CM, Ross GW, Jewell SA, Hauser RA, Jankovic J, Factor SA, Bressman S, Deligtisch A, Marras C, Lyons KE, Bhudhikanok GS, Roucoux DF, Meng C, Abbott RD, Langston JW. 2009. "Occupation and risk of Parkinsonism: A multicenter case–control study," Archives of Neurology 66(9): 1106–1113.

Thomison, PR. undated. "Cultural practices for optimizing maize seed yield and quality in production fields," p. 49. http://www.seedconsortium.org/PUC/pdf%20files/16-%20Cultural%20practices%20for%20optimizing%20maize%20seed....pdf

Thompson et al. 2003. Pesticide Take-Home Pathway among Children of Agricultural Workers: Study Design, Methods, and Baseline Findings. *Journal of Occupational and Environmental Medicine*. 45: 42-53.

USDA NASS 2011. Agricultural Chemical Use: Corn, Upland Cotton and Fall Potatoes 2010. USDA's National Agricultural Statistics Service, May 25, 2011. http://www.nass.usda.gov/Surveys/Guide_to_NASS_Surveys/Chemical_Use/FieldCropChemicalUseFactSheet06.09.11.pdf

USGS 2003. "USGS releases study on toxic rainfall in an Joaquin Valley," US Geological Survey, August 18, 2003.

van den Mark, M et al. 2012. "Is pesticide use related to Parkinson Disease? Some clues to heterogeneity in study results," Environmental Health Perspectives 120(3): 340-347.

van Wijngaarden E, Stewart PA, Olshan AF, Savitz DA, Bunin GR. 2003. Parental occupational exposure to pesticides and childhood brain cancer. Am J Epidemiol. 157 (11): 989–997.

Watts M. 2011. Paraquat. Pesticide Action Network Asia & the Pacific, August 2010.

Weisskopf MG, Moisan F, Tzourio C, Rathouz PJ, Elbaz A. 2013. Pesticide exposure and depression among agricultural workers in France. Am J Epidemiol. 178(7): 1051-8.

Wesseling, C, van Wendel de Joode B, Ruepert C, Leon C, Monge P, Hermosillo H, Partanen T. 2001. Paraquat in developing countries. International Journal Occupational and Environmental Health 7: 275-286.

Wigle DT, Turner MC, Krewski D. 2009. A systematic review and meta-analysis of childhood leukemia and parental occupational pesticide exposure. Environmental Health Perspectives 117(10): 1505-1513.

Wolff MS, Engel S, Berkowitz G, et al. 2007. Prenatal pesticide and PCB exposures and birth outcomes. Pediatr Res. 61(2): 243–250.

Zahm, S.H.; Weisenburger, D.D.; Babbitt, P.A.; Saal, R.C.; Vaught, J.B.; Cantor, K.P.; Blair, A. 1990. A case-control study of non-Hodgkin's lymphoma and the herbicide 2,4-dichlorophenoxyacetic acid (2,4-D) in eastern Nebraska. Epidemiology 1: 349–356.