ADAPTIVE VARIETY SELECTION AND THE UNCERTAIN ROLE OF IMIDACLORPIDS IN THE FUTURE OF SOUTHERN GRAPE CULTURE: STANDING AT THE CROSSROADS

> Veiled in this fragile filigree of wax is the essence of sunshine, golden and limpid, tasting of grassy meadows, mountain wildflowers, lavishly blooming orange trees, or scrubby desert weeds. Honey, even more than wine, is a reflection of place. If the process of grape to glass is alchemy, then the trail from blossom to bottle is one of reflection. The nectar collected by the bee is the spirit and sap of the plant, its sweetest juice. Honey is the flower transmuted, its scent and beauty transformed into aroma and taste.
>
> Stephanie Rosenbaum

By: R.L. Winters
Master Horticulturist/Ampelographer
Fairhaven American Hybrid Research Foundation

In the late winter of 2005 Florida beekeeper Bill Rhodes was busy working his usual day's tasks, the drudgery of preparing hundreds of his bee colonies for shipment to the vast almond groves of California, where they would set about the daunting business of facilitating the production of an important national crop. He noticed an unusual behavior while conducting regular maintenance of the hives. The bees responded to the routine splitting of the hives by refusing to occupy the split sections! Something he hadn't seen before in decades of experience working with these tiny denizens of nature.
Once the broods arrived in California, Rhodes went out to inspect the hives, marking them and returning within the week to check again. “The hive would look entirely different,” Rhodes said. “It was like something just had them by the throat and was just pulling the strength from them. We had no idea what it was.” Of the sixteen semi-loads of pollinators Rhodes had shipped to the location only two were serviceable. The rest had perished.
Backtracking to where the brood had been previously contracted, led Rhodes back to a fruit farming operation in South Dakota. The farm was located immediately adjacent vast acres, overflowing with shades of yellow and black. There, thousands of acres of sunflowers that were literally dripping with imidacloprid.
What this simple, thoughtful, beekeeper had identified is what was to become known later as Colony Collapse Disorder. Overnight, Rhodes had become the unwilling Paul Revere of the basis of world crop production, shouting a warning that would circle the globe!

This is the story:

Imidacloprid is a type of neonicotinoid insecticide that was first registered for use in the United States in 1994. The chemical compound, a synthetic form of nicotine, was originally approved for use in ornamentals, and turf application to control sucking or chewing insects. The product is

marketed in commercial packaging under many different names such as Alias, Merit, Gaucho, Provado, Montana, Nuprid, Marathon, Adonis, Macho, and Dominion.

The neonicotinoid compound is a highly systemic product that is readily absorbed by the crop plant. When applied, it enters the plant vascular system, undergoes complete distribution in the tissues, and renders all parts of the plant toxic to insects. One of the most remarkable aspects of the imidacloprid family of chemical compounds is its dual mode of absorption. Soil drench (or drip) application results in rapid transmission of the compound to the cell structure of the plant via the root system, where it is absorbed with normal water uptake.

Imidacloprid is also unusually persistent, lasting many weeks after foliar application and with repeated applications can persist for up to 6 years in the woody parts of the treated plants and is detectable for many years after soil applications. The amazing potency of the systemic action of the compound is clearly demonstrated by seed treatments of corn and sunflower crops that, after germination, produce a highly toxic seedlings that are virtually impervious to insect attack.

A quick tour of the pesticides on the shelf at any garden center will reveal that imidacloprid has become the chemical of choice in an extensive line of products. The product is also available under various labels by direct internet sales. Imidacloprid has been tasked for a wide spectrum of applications, everything from general use insecticide for killing ants, household insects, pet flea control, to use on ornamentals, and garden food crops. Homeowner use of this compound allows for rates as high a 32 times greater than that of agricultural labeling, and none of the existing labeling warns of severe toxicity to pollinators.

This wide spread label approval is largely due to yet another unusual aspect of the compound. While it is severely toxic to insects, it has a relatively low toxicity to mammals. It works by interfering with the transmission of stimuli in the insect nervous system causing irreversible blockage of acetylcholine receptors, which are found in a type of neural pathway that is more abundant in insects than in warm-blooded animals. These receptors are rendered incapable of receiving acetylcholine molecules (an important neurotransmitter) and an accumulation of acetylcholine occurs, resulting in the insect’s paralysis and eventual death. It is effective by both contact and ingestion modes.

Conversely, this same compound has extraordinary high LD50 (the amount of the substance needed to kill 50% of a test sample group, measured in milligrams per kilogram of body weight) characteristics in mammalian tests with most tests yielding results of 4800-5000 mg/kg for dermal exposure, and an oral LD50 of 450 mg/kg.. To better understand the human toxicity for acute exposure, for a 200 lb. human male, the amount needed to produce lethal effects, would be right at 1lb (of active ingredient) for dermal, and 1.4 ozs. oral. In the world of insecticides imidacloprids have a surprisingly low toxicity to humans, which explains how this relatively unknown compound has seen it use skyrocket to nearly 1 billion pounds worldwide!

The effect on insects is another story. Lethal doses for many flying insects (including Sharpshooters) is an amazingly tiny amount of the product that ranges (depending on the target insect) from a contact application of .024 ug (that's micrograms) to an oral dose of .005 ug - .07 ug. When you consider the minute oral dose that produces a kill, it becomes clear why the compound is so devastating to the Sharpshooter!

From a grape growers standpoint, we tend to say "Wow.....here's the answer to problem of Pierces Disease!" "I'll just keep spraying and my vines and they will be Pierces free!"

But there's a darker, more sinister side to this story.

Beginning in 2005 an apiary farmer in Florida began to notice something unusual in his business of supplying honey bees to large industrial farms for the purpose of pollination. His name was Bill Rhodes, and what he discovered will likely shape the future of grape culture in a wide ranging area of America.

With an increasing frequency, the boxes that were used to house the bee colonies were turning up empty during the course of the pollination contracts. At first these incidents were being written off to the standard list of bee maladies (mites, fungal disease etc) but later tests revealed that the failures were not caused by these diseases or other pollinator pests. Another highly suspicious aspect of the failures was the nearly complete lack of dead bees in or around the boxes! Many of these supers (the correct name for the boxes) contain up to 30,000 foragers and if they had died due to the usual issues then areas around the supers would have been several inches deep in dead bees!

So where did they go?

THE COMMON THREAD

The common thread that runs through this mystery is the close proximity of the pollinators to agricultural (usually mono-culture operations) applications of imidacloprid.
Use of the product began in earnest after agricultural label approval for use on corn in 2000, and later neoniconoids were phased into approval for other crops such as sunflowers in 2005. But how was it, if the product wasn't being sprayed directly on the pollinators, that the chemical was destroying the colonies? All of the early testing of imidacloprid showed no immediate issues with its use as long as the pollinators were protected from the spray drift.

As in many laboratory testing programs involving label approval by the EPA, the responsibility to test and verify the safety of the new chemical compound was left to the producers of the product (a strange dance that seems to have the fox watching the hen house). In the developmental phases, imidacloprid toxicity to pollinators was clearly established under existing guidelines, but a critical mistake was made in understanding, not the acute results of spraying the pollinators, which reveals the usual LD50 (acute lethal) rating, but rather the effects of what is called a sublethal dose.

NO WAY HOME

In the case of sublethal dosing the pollinators aren't killed directly, but absorb or transfer enough of the chemical to the brood to produce toxicity that overlaps into successive generations. In the process of unraveling this paradox, it has become clear that the neonicotinoid was being transferred back to the colony in the form of contaminated pollen. The pollen is then consumed and or conveyed to the developing larvae. As the larvae matured the ingested pollen delivers the sub-lethal dose that would manifest itself by damaging the foragers neurons to the degree that their ability to master the vital, million year old skills of colony behavior, was severely damaged.
As is detailed in the report published by Ecotoxicology 2012 May 21(4) 973-992; "Bees trained to forage on artificial feeders, Bortolotti et al. (2003) noticed that a 500 meter distance between the hive and the feeding area resulted in no foragers at the hive/feeding area up to 24 hours after treatment when foragers were fed with imidacloprid at 500 and 1,000 µg l–1. The latter authors also found that a lower concentration (100 µg l–1 imidacloprid) caused a delay in the returning time (to hive or feeding area) of the foragers".
At the core of the mystery of the disappearance the explanation seems to be a macabre set of symptoms that include the inability to navigate correctly. The foragers use a complex system to find food and nectar sources and chart a return course to the colony. When these instinctive systems were damaged the foragers simply couldn't find their way back to the colonies! The maximum life span of the foragers outside the colony is a mere three days so their fate was sealed, not just a few of them, but rather, by the millions.

Because of imidacloprid's lightning ability to penetrate the plant cell structure, minute quantities of neonicotinoid were metabolized by the crop plants and that micro dose was ultimately transferred to both the pollen and nectar from the flower structure. The mobility of the neonicotinoid to the pollen grains has been clearly demonstrated. Several studies have examined the translocation of imidacloprid from seed treatment to different parts of sunflower (Helianthus annuus) plants.
In a greenhouse experiment with sunflowers treated with 0.7 mg 14C-imidacloprid per seed (Gaucho WS, 700 g kg–1) average imidacloprid concentrations amounted 3.9 ± 1.0 µg kg–1 in pollen and 1.9 ± 1.0 µg kg–1 in nectar (Schmuck et al. 2001). Nectar contained only imidacloprid and in pollen 85% of the 14C-residues were present as imidacloprid. The latter study also determined imidacloprid residues in pollen samples of maize and sunflower that received a seed treatment. In 58% of the pollen samples imidacloprid was found with an

average concentration of 3 µg kg−1 (range 1–11 µg kg−1) for sunflower. In 80% of the maize pollen samples imidacloprid was found at an average concentration of 2 µg kg.
It was this minuscule dose that is at the root of the unforeseen effects that have ravaged hundreds of millions of pollinators on a global scale. The mechanism of the neural damage to the foragers isn't fully understood, but tests on both pollen and nectar samples have shown the presence of the neonicotinoid molecule, and the effects of sublethal dosing is well documented; the rest of the puzzle is coming into clear perspective.

In Europe where regulatory agencies tend to act on the side of caution, the removal of imidiacloprid from the market has ended the death spiral of Colony Collapse Disorder.

Simply dismissing the effects that this product has on pollinators on a global scale is a path fraught with peril. Fully 35% of all food crops, 15 billion dollars of production, including common fruits and vegetables, rely on the intervention of pollinators to complete the cycle of production. The prospect of produce, grains, and fruit products reaching astronomical prices due to scarcity, and then simply disappearing from the shelves isn't a fiction. But rather a looming reality.

THE KNOWN FACTORS

The follow eight points are some of the currently documented facts regarding neonicotinoid use and are supported by numerous research papers:

1. Neonicotinoid residues found in pollen and nectar are consumed by flower-visiting insects such as bees. Concentrations of residues can contain both lethal and sublethal dose levels.
2. Neonicotinoids can persist in soil for months or years after a single application. Measurable amounts of residues were found in woody plants up to six years after application.
3. Untreated plants may absorb chemical residues in the soil from the previous year.
4. Products approved for home and garden use may be applied to ornamental and landscape plants, as well as turf, at significantly higher rates (potentially 32 times higher) than those approved for agricultural crops.
5. Neonicotinoids applied to crops can contaminate adjacent weeds and wildflowers.
6. Imidacloprid, clothianidin, dinotefuran, and thiamethoxam are highly toxic to honey bees.
7. After plants absorb neonicotinoids, they slowly metabolize the compounds. Some of the resulting breakdown products are equally toxic or even more toxic to honey bees than the original compound. Some of the metabolites have higher toxicity to humans.
8. Honey bees exposed to sublethal levels of neonicotinoids experience problems with flying and navigation, reduced taste sensitivity, and slower learning of new tasks, which all impact foraging ability.

PAIRING WITH IMIDACLOPRID

In 2009, some 34,000 pounds of imidacloprid was applied to 182,000 acres of wine grapes, about 36% of vineyards. Since this use data was collected and published in the 2010 Report On Pesticide Use By Commodity the total poundage and demographic of use has expanded exponentially.

As a grape grower, I have spent a great deal of time contemplating the potential of the systemic imidacloprid to affect the safety and integrity of the finished wine. During various seminars I've

attended, I have posited the simple question, several times, to the learned speakers; "Does imidacloprid cross the developmental boundaries during the early stages of berry development and is it present at harvest?" The majority of the responses were either evasive or dumbed-down answers that asserted that the product was "safe" and therefore the issue wasn't something that the grower need be concerned with (it's labeled for grapes so it's okay!). The answer to the question regarding its distribution in fruit becomes rather obvious when current testing reveals that not only is the product present in the plant body for extended periods of time, but is able to invade even the most remote tissues of vine anatomy, right down to the guttation of nectar and to the pollen bodies themselves!

Current testing conducted by SPEC CertiPrep and published in a report by Patricia Atkins, has revealed that the neonicotinoid molecule is present in 35% of samples tested in a far ranging demographic of finished wines at an alarming level of 3 ppm, and in certain samples as high as 35 ppm (using mechanical sample agitation). Current EPA food safely tolerances for residues of imidacloprid and its metabolites in food range from 0.02 ppm in eggs to 3.0 ppm in hops. (the discrepancy of this wide range of safety tolerance isn't clear). Safety tolerances for finished wines are non-existent.

All of this raises some serious questions regarding the effects of imidacloprids in human metabolism. Most frightening of all is the lingering question regarding the testing of this complex, and aggressive agent, and it's safety for human consumption. Even though it's toxicity to insects is well researched the guanidine metabolite of imidacloprid is significantly toxic to humans and the knowledge base surrounding the health effects of this component of the molecule is nearly nonexistent.

Is the same faulty evaluation of "sub-lethal" dosing that has has seen the decimation of the pollinators operative when it comes to human consumption?

The omnipresence of this designer systemic has been the dirty little secret of wines produced throughout the "Pierces Belt" of the southern tier of states. The deformed child that the wine industry has kept carefully hidden in the cellar.

OPPORTUNISTIC FORAGERS

Grapes are regularly visited by three types of pollinators, Solitary bees, Honey bees, and several species from the order Diptera (flies). It is quite correct that most grape flowers are self-pollenating, a characteristic that is considered desirable in grape breeding, and in the development of cultivars for production for the last 3000 years. All of the prominent varietals are self-pollenating and are adequately fertilized by wind action and mechanical dispersal of pollen. The roll that pollinators play is largely in facilitating the distribution of pollen, assuring better and more uniform pollination. Certain varieties of American hybrids are dependent on the assistance offered by these diligent visitors to the vineyard and are greatly enhanced by this boost.

Grape vines produce an abundance of both pollen and nectar. And, in understanding the behavior of pollinators, one must comprehend that they are opportunistic foragers. That is, they don't visit any specific flowering plant with some intrinsic understanding of whether or not the species actually requires their assistance to produce fruit, they simply go about collecting pollen and consuming nectar. It's what they do.

Don't be fooled by the many myopic comments that are posted to the various grower sites that state that "pollinators aren't required for grapes", so therefore, they are to be generally disregarded. The fact remains that they are an integral part of the annual cycle in the vineyard and have a vital (ancient) role in all aspects of grape vine ecology, including the preservation and hybridization of native grape species in the various ecotomes across the Western Hemisphere.

OCCAMS RAZOR

This issue, eventually, is reduced to its common denominator. And that is the issue of Pierces Disease, and how to combat its devastating effects. Imidacloprid has offered a workable, even if somewhat ungainly, alternative to watching vines shrivel up and die. But isn't the real issue a matter of selection? As far back as the post civil war era Thomas Munson had observed that while European grapes died by the thousands, the native grapes in the New World were largely unaffected by the rigors of the Texas climate, and at the same time were immune to a mysterious disease that was then called "Grape Vine Decline" (Pierces). Munson narrowed his focus on crossing inside this group of native vines to improve the varieties and enhance the juice quality. In this astounding group of hybrids, he handed us many selections that demonstrate nearly complete tolerance of Pierces, while still maintaining extraordinary juice qualities. By simple observation, he had crossed the intellectual rubicon that has continued to escape modern viticulture in the Pierces prone areas, in its headlong plunge to be something it may have never been meant to be.

At the core of the issue is varietal selection and the misguided belief that, in order to compete, Texas must produce grapes and wine that meet the lofty standards set by the California wine business. Somehow they can't be (truly) Texan, but rather they must be Texaforinan!
To a degree, some Texas vineyards have approached that level of quality. But most of those production areas are located well outside of the Pierces Belt and are graced with dryer, generally cooler conditions than the rest of the state. Which leaves the balance of the growers (the majority of the state and the rest of the south) struggling with cultivars that will never fully succeed in their growing areas, and present nothing short of a maintenance nightmare.

The various appellations in Europe don't seem to have much trouble letting better adapted varieties represent the culture and history of their respective regions. Maybe it has somehow escaped me, but I just haven't noticed any hand wringing by the folks in the Ribera del Duero wine region of Spain because they can't grow Cabernet just like the growers of Bordeaux! "Darn that Tempranillo....if we just could grow Cabernet we would be just as good as those guys!"
In our society, noted for its abbreviated historical knowledge (social ADD), its little wonder that few growers are aware that American varieties such as Lomanto, Extra, and Hussman were once the prize red wine grapes of the South in the years leading up to the Volstead Act. With the death of Thomas Munson, the wealth of knowledge, and the source for the vines disappeared. A legacy forgotten, placed on the dusty shelves of history.

We have set unrealistic standards for grape culture that has seen a million year old bacteria outwit us at every turn (they aren't very smart...which makes us seem even dumber).
Rather than accepting that varieties such as the American Hybrids may offer an answer to problem of grape culture for most of the south, and afford us a way toward a unique regional group of cultivars, we have, instead, chosen a path to chemical oblivion.

We have, unwittingly, become part of a process that undermines the universal basis of food production through the use of imidacloprids, in the name of forcing poorly adapted, physiologically deficient varieties (Vinifera) into production, we have lost our sense of reason.

WHAT YOU CAN DO - 10 STEPS

1. **Immediately terminate the use of the nitro-group form of imidacloprid and switch to the less toxic cyano-group.** The newer, safer (for bees) form of imidacloprid is sold under commercial names such as Assail, and Tristar
2. Mitigate imidacloprid contamination of the soil and ground water by switching to foliar application only.
3. Reduce cross contamination of native wild flowers by eliminating flowering weed growth in the vineyard
4. Avoid co-mixing imidacloprid with other insecticides until current research clarifies the effects.
5. **Do not apply products while pollinators are present**. Allow sufficient time prior to daylight exposure for spray material volatilization to complete and spray drift to settle.
6. Plan for a future where imidacloprid is either removed from the market or becomes highly restricted by developing alternative spray routines.
7. Modify existing vineyard IPM programs to increase Sharpshooter monitoring with the aim of maintaining control by contact application of non-neonicotiniod products.
8. Plant adapted varieties that are either tolerant or resistant to Pierces Disease.
9. Lobby your congressman to immediately force the EPA to suspend label approval for homeowner use of neonicotinoid products.
10. Lobby your congressman to immediately force the EPA to suspend label approval for the use of all nitro-group neonicotinoid products.

Written by;
R.L. Winters
Master Horticulturist/Ampelographer
Fairhaven American Hybrid Research Foundation