GMOs, Glyphosate and Tomorrow

A recently discovered infectious agent, as small as a virus, is implicated in causing diseases in crops treated with glyphosate as well as significant health problems in animals fed with these contaminated GM products. A concerned scientist made a stand before it's all too late.

An interview with
Don Huber, PhD
by Chris Walters © 2011

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Seeds evolved for millions of years before humans invented corporate agribusiness. Genetic selection to improve crops began only when people invented farming. Early on, there was a vast germ pool from which to select differences in vigour, growth, quality characteristics, yield or disease resistance. Even after years of extensive selection and later blending into hybrids by diligent researchers during the past century, most of this inheritance is unpatentable and therefore useless as a source of power or corporate-style profit.

Genetic engineering to modify crops exists because most of the world's farmers depend on seeds; and as a novel way to manipulate genes, it offers inviolate proprietary control. Two traits account for practically all of the genetically modified [GM] crops grown in the world today. One deploys herbicide tolerance enabled by a glyphosate-insensitive form of the EPSPS gene coding (key to this GMO is the soil bacterium *Agrobacterium tumefaciens*). The other uses insect resistance due to one or more toxin genes derived from the soil bacterium *Bacillus thuringiensis*.

It is the former that concerns us here, for without glyphosate the biotech industry would be an orphan, all dressed up with nowhere to go. Glyphosate, often known as Roundup® after the popular Monsanto product but available in many guises since its patent expired in 2000, is the partner which GMOs must bring to the dance. It is a broad-spectrum herbicide that ingeniously ties up nutrient access rather than killing unwanted plants directly. It was heralded for many years as a relatively benign replacement for the horrific dioxin-based herbicides of the past. The figures don't lie; GMOs drive glyphosate sales.

Enter Dr Don Huber, a plant pathologist of 50 years' standing, now an Emeritus Professor at Purdue University and enjoying an active post-academic life. Huber is an international authority on nutrient deficiency diseases of plants and is particularly well situated to comment on glyphosate as it functions through nutrient tie-up, not inherent toxicity.

His retirement turned hyperactive when a letter he wrote to Secretary of Agriculture Tom Vilsack was leaked. Although much of the mainstream media ignored it, the letter [dated 16 January 2011] was an immediate sensation. Huber informed Vilsack that a new infectious agent had been discovered. It is "widespread, very serious, and is in much higher concentrations in Roundup Ready (RR) soybeans and corn," he wrote. He appealed to the Secretary for help with resources and research capability. The letter unleashed a storm of alarm and denial, and, as Huber tells below, the US Department of Agriculture [USDA] is looking into the matter despite its ill-advised approval of genetically modified alfalfa. We asked him to comment on his letter and share his own thoughts and opinions on this ubiquitous farm chemical.

— Chris Walters, Contributing Editor, *Acres* U.S.A.

INTERVIEW WITH DR DON HUBER

Acres U.S.A.: How does glyphosate differ from herbicides that were popular before it came along?

Don Huber: There are a number of ways that glyphosate is different from most other herbicides. Most of our herbicides are mineral chelators that act to physiologically immobilise a specific mineral nutrient that is required for a specific critical enzyme. When that physiological pathway is shut down, the weed or the plant it's applied to dies. Glyphosate also is a chemical chelator that can grab onto mineral nutrients and immobilise them physiologically so they're no longer available for those physiological functions that they regulate. The difference with glyphosate is that it is not specific to just one mineral nutrient, but immobilises many of them and doesn't affect a primary mechanism to cause death by itself. It merely turns off the plant's defence mechanisms so that soilborne fungi that would normally take weeks to months to damage a plant can kill it in just a few days after glyphosate is applied. When they use the glyphosate-tolerant technology, they insert another gene that keeps that plant's defence mechanism going somewhat so you can put the glyphosate directly on the crop plant without having it killed. But the technology doesn't do anything to the glyphosate, which is still tying up mineral nutrients. Any time you put the gene in, you reduce the nutrient efficiency of the plant, though not to the point that it destroys the ability of the plant to survive. It does leave it physiologically impaired.

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Acres: Before glyphosate-tolerant genes were introduced, how did farmers cope with the danger of possibly killing the crop plant?

Huber: They took care of their weed control before planting or before the crop emerged. Back then, there weren't too many herbicides that you could apply directly to the plant. We had a few, 2,4-D and a few others, that were semi-selective and very effective against broadleaves, which have a different physiology than grass plants. A similar thing with Tordon®: you can put Tordon right on a grass pasture and it will kill the broadleaf weeds for three or four years. It has pretty good residual activity, but grass looks like you'd just fertilised it when you got rid of all of those broadleaved weeds.

Acres: The innovation that gave glyphosate its market clout had to do with concentrating the whole arsenal into one weapon? No more multiple herbicides?

Huber: There was selective activity in our herbicides. Glyphosate on plants without the new gene inserted has a very broad-spectrum effect so that all weeds are affected. They're all killed by the soil fungi. It's not quite analogous, but you could say that what you're doing with glyphosate is you're giving the plant a bad case of AIDS. You've shut down the immune system or the defence system.

Acres: How does glyphosate's immobility as a strong metal chelator or nutrient chelator translate into the long-term effects of glyphosate build-up after years of steady use?

Huber: As long as it's bound very tightly with those mineral elements, it is not available or not in an active form for plant damage. If there is something that happens to break that binding, then it can again be released and available for root uptake and plant damage. It depends on how long it survives in the soil, and that will depend on two primary factors. Soil pH is a big factor in stability, and the other is clay content. The higher the pH, the less stable it is; and the higher the clay content, the more stable it will be. In a high-clay soil, it may survive for a number of years. In water solution, it can degrade fairly rapidly and not have a lot of residual activity. I think that's probably one reason why the French Supreme Court ruled two years ago that it would be fraud to claim biodegradability of glyphosate in soil—because it's not always really predictable. For some soils it can survive for a long period of time, and in others it may have a much shorter period. With the information that's currently available, it's not really possible to have a good predictable figure. We do know that even though glyphosate is immobilised rapidly in most soils, it can then be reactivated or desorbed and reactivated to damage future crops.

Acres: What must happen to reactivate it?

Huber: One of the things that's recently been shown to do this is to apply phosphorus fertiliser on the crop. From a nutritional standpoint, it can actually desorb the glyphosate so that it's again reactivated as an active chemical for plant uptake and damage.

Acres: Has this been demonstrated by researchers to impact the crops when it's desorbed?

Huber: Yes. That can be quite damaging to the crop and actually limit uptake of nutrients required by the crop as much as 60 to 70 per cent, and that's pretty much across the board. Most elements will be reduced around 60 per cent, and a few of them in the 70 per cent range. In this way, the plant can be placed under a fairly significant nutrient deficiency, even though the nutrients may be in the soil. The plant can't utilise them because of glyphosate's toxicity.

Acres: Have your colleagues found similar impacts?

Huber: Yes. A number of soil microbiologists are all

reporting the same type of impact on the soil biology. One paper mentions that it's a very powerful herbicide, but also a very potent biocide. It's a little bit selective in that it stimulates some soil organisms and is very toxic to other organisms. It's toxic to your legume nodule bacteria for nitrogen fixation; also quite toxic to the organisms that make manganese and iron available for plant uptake, and those are critical nutrients. It stimulates the soil pathogens that do the killing from a weed control standpoint, but it also stimulates some so that you're essentially making a super-pathogen to kill a weed. Then you leave that super-pathogen in the soil, which also attacks other plants later on in the rotation.

Acres: The letter you sent to Secretary of Agriculture Tom Vilsack in January not surprisingly is being attacked on a number of fronts. Since the pathogen that has been discovered hasn't been detailed in a journal, its existence has been questioned. How was this pathogen discovered, who did the research, and is research being readied for publication?

Huber: The letter to the Secretary wasn't for public dissemination. It was a request for help. It was meant to bring to his attention the things that many of us are seeing out in the field, both from the veterinarians and animal producers as well as agronomists, plant pathologists and our crop producers. I wanted to bring the situation to his attention and request help so we could move the science along faster than we can individually. It's because of the seriousness of the situation that many growers are experiencing. The work to date has been very well done, very scientifically conducted, but there is still much to do. Much of it hasn't been published on the animal side, but Koch's postulates—the scientific criteria used to establish a cause–effect relationship—have been completed, and much of the science on the animal side has been done. That's not a concern or a question. The veterinarians have been very thorough. They split their samples, sent them to a number of different labs to rule out all of the other known causes of those conditions; and when they check for this new organism, that's what they find. They find it with cattle and pigs and horses and poultry. So it has a pretty broad host range. In trying to identify how the animals were being infected, they began looking at the feed and found that soybean meal was just loaded with it. They also found it in silage and corn products.

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Any fermented product seems to encourage this organism. It's also a very good synergist with other pathogens. The *Fusarium* fungus that causes sudden death syndrome (SDS) is very compatible with this new organism. Another interesting thing is that it appears very compatible with *Clavibacter*, which causes Goss's wilt of corn, as well as other bacteria. Over the past two years we've had extensive SDS and Goss's wilt epidemics, and that's where we really see the higher titre with this organism. The two diseases and the newly discovered pathogen appear to be very synergistic. This new organism may be an opportunist that is able to take advantage of a weakened condition and then really move forward.

Acres: What is "higher titre"?

Huber: Higher population. Just a lot more of it. It seems to grow better for possibly a higher infection potential.

Acres: Is this the first appearance of this pathogen in nature? Or is it something that was there all along, waiting for discovery?

Huber: We're fairly convinced it's something that's always been there, very benign, not really a problem until we changed something that has either increased its virulence or its opportunity. I think the research to date would indicate that it's probably more a change in the susceptibility of the crops, in the population of the pathogen, and in the potential for animal infection. There are many organisms new to science that have been around forever, which is something you see with the prions. We didn't know they existed either until we had to look a lot further to find an answer to a problem, and then they were discovered. This organism was discovered pretty much the same way. When they ruled out all other known sources, then the veterinarians just kept looking and found this one, and then verified it as the cause by doing Koch's postulates. Then they took it a step further to find out: where was it coming from? How are the animals getting it? That led them to check the feed and they found it there. In science, you go from one thing to another, sometimes in a process, and you don't necessarily stop and publish each little bit that is found until you have a better understanding of how it all fits together. In agriculture, we're really talking about a system; we're not talking about silver bullets.

Acres: People have an easier time understanding single-factor analysis and silver bullets, but that's not how it works in nature, is it?

Huber: We're talking about how parts of this system interact and fit together. That's been the real emphasis in this research, not how to get that publicity and meet the popular demand by publishing each little bit of information. You try to get enough research so you can really understand its scope and what its impact is in the overall production system. That's really my plea to the Secretary in that letter: we need resources, and we need some commitment of those resources and personnel that are available to the Secretary but aren't available to each individual scientist. It was for alerting him to the problem so he would be interested, as he has been, in passing it on to those who would be able to provide additional resources. We need to understand how it fits into the overall ecological scheme and agricultural production system.

Acres: Despite the recent rapid approval of genetically modified alfalfa, do you find a silver lining in indications that USDA resources or commitment are forthcoming?

Huber: Well, I certainly hope so.

Acres: This pathogen doesn't have a name. What do you call it?

Huber: That's been a bit of a stumbling block. In the letter I called it a microfungus. That was a mistake, because when you think of a microfungus you automatically think of a mould-type organism, and it certainly isn't that. It's many thousands of times smaller than a mould, much smaller than a bacterium, approximately the size of a virus. It's in that category, except that it self-replicates and can be passive.

Acres: But it is certainly not a virus?

Huber: Not by our current definition.

Acres: Could your theory be summarised thusly: this is not the result of a mutation in an existing pathogen; rather, a change in the conditions has caused an existing pathogen to multiply and become a problem, with pathways being created that were not common in the past?

Huber: Right. The organism appears to be prominent in the environment but new to science. On a much larger scale, it would be like when they bred the Texas male-sterile gene into corn. We got away with it for a few years. Then all of a sudden we realised we had an organism out there that was new to science with the Southern corn leaf blight epidemic of 1970–71. We'd previously had that experience with the Victoria gene in oats.

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Acres: Can you name some of the researchers who are involved? Specifically, who discovered the pathogen?

Huber: No. Because there's no need for them to have the harassment or be inundated the way I've been. We've got too much work to do.

Acres: But you can vouch for them?

Huber: They are very well established scientists. There's no need to attack everybody else, and that's exactly what happens when you come up with something that's new.

Acres: In other words, naysayers are assured that there is more than one person involved with this research, they're reputable people, the results are going to be published as soon as they're available, and these plant and animal afflictions are not going away?

Huber: *Clavibacter* survives in corn residue for three to four years at least; so if we continue to do the same things, we should anticipate the same result. There's research that shows that when you apply formulated glyphosate to a glyphosate-tolerant corn plant that normally is resistant, some hybrids become fully susceptible to that organism. Glyphosate can nullify the genetic resistance for *Clavibacter* just like it can for *Rhizoctonia* in sugar beets or *Fusarium* in some plants.

Acres: What other results do you anticipate?

Huber: High infertility and abortions in animals fed with corn and soybean feeds containing high populations of this organism.

Acres: Some of your critics reject the whole idea that sudden plant death and spontaneous cattle abortions are even an increasing problem.

Huber: It isn't a universal phenomenon, just as most disease outbreaks can be limited. I think the criticism goes against the statistics, though. If you look at the USDA's anticipated yield on corn that they put out in August and then subtract the actual yields reported in January, you come up with almost a billion bushels less, even though we had near-ideal conditions for harvest. Where did those billion bushels go? All you have to do to document that there was a short crop last year is look at the price. We're no longer talking about $3 per bushel of corn, we're talking about $6 per bushel. That's not from increased ethanol use; that's from a major shortage in the crop produced. How do you get soybeans from $5 up to $12? You have a short crop because you have an inelastic supply–demand

relationship in agriculture. I think the figures document that. In some areas they didn't have those problems this year as some had last year, and that's because environmental conditions are also important for disease…

Acres: What was the major focus of your work during the years before you became a retired, or emeritus, professor?

Huber: For 50 years my research was focused heavily on the biology and control of soilborne pathogenic fungi, microbial ecology, biological control, microbial interactions and host–parasite physiology, trying to understand resistance and susceptibility from a physiological standpoint. I was heavily involved in the whole development of nitrification inhibitors and also in identifying nutrient pathways in corn, soybeans and wheat. I served as one of the editors of the American Phytopathological Society's book, *Mineral Nutrition and Plant Disease*, which came out in 2007. I initially got involved with glyphosate, thinking that when glyphosate-tolerant soybeans were released it would probably be a win-win situation for a lot of our growers who didn't want to make a separate trip across the soybeans to meet the nutritional demands for manganese. If they could just add manganese as a tank mix, it would be a pretty good time to remedy the manganese deficiency we saw in a number of areas in Indiana, and they could get the weeds controlled at the same time. It only took one trial to realise that it wouldn't work, because glyphosate immobilised the manganese that we were trying to make available for the plant. The last 15 to 16 years were primarily devoted to understanding and finding ways to remedy the nutrient inefficiency that the technology and the chemistry were imposing on the plant. Of course, that brought me right back to looking at a lot of those soil–microbial interactions that are so essential to making nutrients available to plants to start with…

"It only took one trial to realise that it wouldn't work, because glyphosate immobilised the manganese that we were trying to make available for the plant."

Acres: Genetic engineering is relatively new to science. Does that bring this problem to a new level of seriousness, because you can't just remove those traits, that is, the way you would if you simply stopped a hybrid program that was making something you didn't like?

Huber: It's certainly easier to put it in than to get it out. Each time you put a foreign gene in, you're adding another stress to the plant—commonly referred to as a yield-drag aspect, which is very well documented. There's powerful technology here, and usually, with a little bit of time, we can find a way to make that work more compatibly. Genetic engineering is a tool we may need for specific situations, but it's also been easy to abuse. I believe that when we start putting all of our eggs in one basket, it increases our vulnerability and potential risk factors dramatically. I believe we should try to follow scientific principles and use a lot of caution until we understand what's going on in the whole process…

Acres: Despite the difficulty American researchers in particular have experienced, can you now cite much data that wasn't around when GMOs were introduced?

Huber: There's a fair amount of toxicological data indicating that there are very serious concerns with some of the products. That's also one of the things that has been looked at with infertility and spontaneous abortions. There is an increasing level of glyphosate in our food chain, and, with the toxicological data that's now available, the levels are often many times the level that would send up a very serious concern from a clinical laboratory standpoint. Some of that data shows that quite low levels of glyphosate are very toxic to liver cells, kidney cells, testicular cells and the endocrine hormone system, and it becomes important because all of the systems are interrelated. We're finding fairly significant levels of glyphosate in manure. You have to ask how the chicken got it or how the hog or cattle got it, and, of course, that's through their feed. Is it all moving through the animal or is it also into their meat and other tissues? We really don't have a lot of that data. Some of the other countries are collecting it and doing the analysis, and we're just starting to do some in this country. But for the most part it's just been considered so safe that we closed our eyes and said there's no need to do any of that work… ∞

About the Interviewee:
Dr Don M. Huber is Professor Emeritus of Plant Pathology at Purdue University, Indiana, USA. He received BS and MS degrees from the University of Idaho (1957, 1959) and a PhD from Michigan State University (1963), and is a graduate of the US Army Command and General Staff College. He was a cereal pathologist at the University of Idaho for eight years before joining the Department of Botany and Plant Pathology at Purdue University in 1971. He also has done advisory work for the US Department of Agriculture. He can be contacted at huberd@purdue.edu.

About the Interviewer:
Chris Walters is Contributing Editor of *Acres U.S.A.*, founded by his father Charles Walters (1926–2009). Based in Austin, Texas, *Acres U.S.A.* has a 40-year track record of reporting on sustainable agriculture and related issues. For more information, visit http://www.acresusa.com.

DR DON HUBER'S LETTER TO US SECRETARY OF AGRICULTURE TOM VILSACK

January 16, 2011

Dear Secretary Vilsack:

A team of senior plant and animal scientists have recently brought to my attention the discovery of an electron microscopic pathogen that appears to significantly impact the health of plants, animals, and probably human beings. Based on a review of the data, it is widespread, very serious, and is in much higher concentrations in Roundup Ready (RR) soybeans and corn—suggesting a link with the RR gene or more likely the presence of Roundup. This organism appears NEW to science!

This is highly sensitive information that could result in a collapse of US soy and corn export markets and significant disruption of domestic food and feed supplies. On the other hand, this new organism may already be responsible for significant harm (see below). My colleagues and I are therefore moving our investigation forward with speed and discretion, and seek assistance from the USDA and other entities to identify the pathogen's source, prevalence, implications, and remedies.

We are informing the USDA of our findings at this early stage, specifically due to your pending decision regarding approval of RR alfalfa. Naturally, if either the RR gene or Roundup itself is a promoter or co-factor of this pathogen, then such approval could be a calamity. Based on the current evidence, the only reasonable action at this time would be to delay deregulation at least until sufficient data has exonerated the RR system, if it does.

For the past 40 years, I have been a scientist in the professional and military agencies that evaluate and prepare for natural and manmade biological threats, including germ warfare and disease outbreaks. Based on this experience, I believe the threat we are facing from this pathogen is unique and of a high risk status. In layman's terms, it should be treated as an emergency.

A diverse set of researchers working on this problem have contributed various pieces of the puzzle, which together presents the following disturbing scenario:

This previously unknown organism is only visible under an electron microscope (36,000X), with an approximate size range equal to a medium size virus. It is able to reproduce and appears to be a micro-fungal-like organism. If so, it would be the first such micro-fungus ever identified. There is strong evidence that this infectious agent promotes diseases of both plants and mammals, which is very rare.

It is found in high concentrations in Roundup Ready soybean meal and corn, distillers meal, fermentation feed products, pig stomach contents, and pig and cattle placentas.

The organism is prolific in plants infected with two pervasive diseases that are driving down yields and farmer income—sudden death syndrome (SDS) in soy, and Goss' wilt in corn. The pathogen is also found in the fungal causative agent of SDS (*Fusarium solani* fsp glycines).

Laboratory tests have confirmed the presence of this organism in a wide variety of livestock that have experienced spontaneous abortions and infertility. Preliminary results from ongoing research have also been able to reproduce abortions in a clinical setting.

The pathogen may explain the escalating frequency of infertility and spontaneous abortions over the past few years in US cattle, dairy, swine, and horse operations. These include recent reports of infertility rates in dairy heifers of over 20%, and spontaneous abortions in cattle as high as 45%.

For example, 450 of 1,000 pregnant heifers fed wheatlage experienced spontaneous abortions. Over the same period, another 1,000 heifers from the same herd that were raised on hay had no abortions. High concentrations of the pathogen were confirmed on the wheatlage, which likely had been under weed management using glyphosate.

Recommendations

In summary, because of the high titer of this new animal pathogen in Roundup Ready crops, and its association with plant and animal diseases that are reaching epidemic proportions, we request USDA's participation in a multi-agency investigation, and an immediate moratorium on the deregulation of RR crops until the causal/predisposing relationship with glyphosate and/or RR plants can be ruled out as a threat to crop and animal production and human health.

It is urgent to examine whether the side-effects of glyphosate use may have facilitated the growth of this pathogen, or allowed it to cause greater harm to weakened plant and animal hosts. It is well-documented that glyphosate promotes soil pathogens and is already implicated with the increase of more than 40 plant diseases; it dismantles plant defenses by chelating vital nutrients; and it reduces the bioavailability of nutrients in feed, which in turn can cause animal disorders. To properly evaluate these factors, we request access to the relevant USDA data.

I have studied plant pathogens for more than 50 years. We are now seeing an unprecedented trend of increasing plant and animal diseases and disorders. This pathogen may be instrumental to understanding and solving this problem. It deserves immediate attention with significant resources to avoid a general collapse of our critical agricultural infrastructure.

Sincerely,

COL (Ret.) Don M. Huber

Emeritus Professor, Purdue University

APS Coordinator, USDA National Plant Disease Recovery System (NPDRS)