GMOs In Animal Agriculture
COMMON QUESTIONS FROM THE PUBLIC ANSWERED BY EXPERTS
GMO Answers (GMOAnswers.com) was created to do a better job answering your questions — no matter what they are — about GMOs. The biotech industry stands 100 percent behind the health and safety of the GM crops on the market today, but we acknowledge that we haven’t done the best job communicating about them – what they are, how they are made, what the safety data says.

GMO Answers is an initiative committed to responding to your questions about how food is grown. Its goal is to make information about GMOs in food and agriculture easier to access and understand. The members and partners of GMO Answers commit to five core principles:

**FIVE CORE PRINCIPLES**

1. Respecting people around the world and their right to choose healthy food products that are best for themselves and their families;

2. Welcoming and answering questions on all GMO topics;

3. Making GMO information, research and data easy to access and evaluate and supporting safety testing of GM products; including allowing independent safety testing of our products through validated science-based methods;

4. Supporting farmers as they work to grow crops using precious resources more efficiently, with less impact on the environment and producing safe, nutritious food and feed products;

5. Respecting farmers’ rights to choose the seeds that are best for their farms, businesses and communities and providing seed choices that include non-GM seeds based on market demands.

Find GMOAnswers on these social channels:

@GMOAnswers  Facebook.com/GMOAnswers  Pinterest.com/GMOAnswers
Table of Contents

Meat, Milk & Eggs  1 - 3

Food Safety  4 - 7

Animal Feed & Nutrition  8 - 11

Animal Health  12 - 21

GMO Animals  22-27
In the United States, livestock have been fed genetically engineered crops since these crops were first introduced in 1996. In 2005, 87 percent of the U.S. soybean crop and 52 percent of the U.S. corn crop were grown from genetically engineered seed (see the USDA ERS Briefing Room website). Because the majority of corn (72 percent) and soybeans (60 percent) are used for livestock feed, it is clear that the livestock industry is a major user of genetically engineered crops.

**Are genetically engineered feeds safe for livestock?**

Over 100 digestion and feeding studies examining the effects of feeding genetically engineered crops to various food-producing animal species (e.g., beef cattle, swine, sheep, fish, lactating dairy cows, water buffalo and chickens) have been reported in the scientific literature (see the Federation of Animal Science Societies Communications website for a comprehensive listing by species and crop). Results have revealed no significant differences in the nutritional value of feedstuffs derived from commercially grown genetically engineered crops compared with their conventional counterparts, nor have any peer-reviewed studies documented alterations in feed intake, growth or other livestock production parameters as a result of including currently available genetically engineered feedstuffs in diets of animals (for a comprehensive review, see Flachowsky et al. 2005). The published literature also contains no indication of any disturbance to food animal health or the quality of resulting animal products as a result of long-term consumption of genetically engineered feeds. Current scientific evidence confirms the concept of “substantial equivalence” for currently available genetically engineered feedstuffs. “Substantial equivalence” is a comparative approach to the assessment of food safety that involves comparing the feed value and safety of genetically engineered crops with those in existing crops (usually the genetically unmodified parent line) that have known feed values and a history of safe use.

**Does genetically engineered DNA or protein get into milk, meat or eggs?**

Genetically engineered crops are digested by animals in the same way as conventional crops. Numerous scientific studies have examined the digestive fate of genetically engineered DNA and protein introduced into genetically engineered feed (see the Federation of Animal Sciences Communications website for a comprehensive listing). Genetically engineered DNA, or the novel proteins encoded therein, have never been detected in the milk, meat or eggs derived from animals fed genetically engineered feedstuffs. Several studies have documented that small fragments of plant-derived, but not genetically engineered, DNA can pass into the tissues of animals that consume the plants (see, for example, Aumaitre et al. 2002). Multicopy plant-specific DNA sequences have been found in various tissues (e.g., muscle, spleen, liver and kidneys) of chicken, cattle and pigs. There has even been a report on the transient presence of rabbit DNA in blood samples derived from human volunteers after they ate a cooked rabbit meal (Forsman et al. 2003). The biological importance of these findings is unclear because the transient DNA fragments are generally too small to encode a protein, and it is unclear whether they possess any biological activity.
Are nutrients in the meat, milk or eggs different?

Nutrients in meat, milk and eggs from livestock fed genetically engineered feeds have been found to be the same as the nutrients from livestock fed conventional feeds. The metabolic processes involved in digestion, absorption and the use of feed proteins by livestock species make it very unlikely for a protein of any plant gene to be found intact in food of animal origin, and none have been detected. For this reason, products derived from animals that have been fed feedstuffs containing the current commercially approved genetically engineered crops do not require specific labeling in the United States. Labeling is required only when genetically engineered food products have a detectable difference in nutritional composition or safety when compared with comparable non-genetically engineered products. In addition, labeling that details the process(es) used to create compositionally equivalent foods is currently not required.

What if I choose not to eat products from animals given genetically engineered feed?

Consumers seeking to purchase products from animals that have not been fed genetically engineered feed can do so by purchasing organic livestock products. The USDA National Organic Program requires that livestock sold, labeled or represented as organic be fed organic feed sources only, unless organic feed sources are commercially unavailable. Even if organic livestock producers are statutorily permitted to use nonorganic feed sources for their livestock, the National Organic Program standards specifically prohibit the use of feed grains from genetically engineered sources.

PERSPECTIVE

Evidence to date strongly suggests that feeding livestock with genetically engineered crops is equivalent to feeding unmodified feed sources in terms of nutrient composition, digestibility and feeding value. Over one hundred scientific studies have found no difference in the productive performance or health of livestock that have been fed genetically engineered feedstuffs, and they found no presence of genetically engineered DNA or proteins in the milk, meat or eggs from animals that have eaten genetically engineered feed. Since it is not possible to distinguish any differences in the nutritional profile or components of animal products following inclusion of currently available genetically engineered feedstuffs in the animal diets, labeling of such animal products is not required in either the United States or Europe.

References Provided by Expert: bit.ly/2mgVygN
Q: If a cow eats GMO corn or soy is there any way to tell or is there any difference in that animal’s meat or milk as opposed to an animal that consumed only organic feed?

Answered By: Bruce M. Chassy, Professor Emeritus of Food Safety and Nutritional Sciences, Department of Food Science and Human Nutrition, University of Illinois at Urbana-Champaign

No! There is no way to detect whether an animal has been fed GM feed because there is no content from any part of the GM feed in the flesh, milk or eggs. The GM feed is digested and assimilated in exactly the same way as any other feed. There is in fact no meaningful measurable difference between GM and convention feeds.

Actually that’s not exactly true. It turns out that conventional and organic feeds can have more of the mycotoxin fumonisin in them than does insect resistant GM feed. Specifically, studies show that insect protected corn can have less fumonisin. Molds that produce mycotoxin often grow at sites of insect damage on corn ears and because the GM grain has less insect damage it has less mold and consequently lower levels of fumonisin. Thus GM grain can in this instance be safer than non-GM. Fumonisin causes esophageal cancer, neural tube defect babies (NTDs) and other illness so this is an important benefit that is largely being over-looked because of the hysteria that has been created around the safety of GM crops.

Read More: bit.ly/1N48xxX

ARTICLE: WSU researchers find U.S. breast milk is glyphosate free

By: Community Manager

This article is excerpted from WSU News:

“Washington State University scientists have found that glyphosate, the main ingredient in the herbicide Roundup, does not accumulate in mother's breast milk.

“Michelle McGuire, an associate professor in the WSU School of Biological Sciences, is the lead researcher of the study, which is the first to have its results independently verified by an accredited, outside organization.

“Her findings, presented at the Federation of American Societies for Experimental Biology Conference on July 23 in Big Sky, Mont., show that glyphosate, the most used weed-killing chemical in the world, does not accumulate over time in human milk. She conducted the study with Kimberly Lackey, Ph.D. candidate in zoology, laboratory technician Janae Carrothers and colleagues at the nearby University of Idaho.

“The U.S. Environmental Protection Agency is using the study as part of an ongoing review of glyphosate regulations prompted by public concern over a controversial report on the chemical released by the advocacy group, Moms Across America, last year.

“'The Moms Across America study flat out got it wrong,' said McGuire, who is an executive committee member for the International Society for Research in Human Milk and Lactation and a national spokesperson for the American Society for Nutrition. 'Our study provides strong evidence that glyphosate is not in human milk. The MAA findings are unverified, not consistent with published safety data and are based on an assay designed to test for glyphosate in water, not breast milk.'”

Read More: bit.ly/2m4AokS
Q: When a transGMO weird stuff that is not rice A to rice B is created say it has a protein from another nonrelated species, what kind of toxicology studies are made? For cis GMOs rice A to rice B for example, its usually something already consumed so not as much as a problem right? How do we know that the new proteins are safe to eat? how do we know a protein already tested will be express at a concentration level safe to eat?

Answered By: Joseph Jez, Professor of Biology and a Howard Hughes Medical Institute Professor at Washington University in St. Louis

To answer these questions, let’s start with some background on what is a protein, how a protein is made, and what happens when we eat.

The first rule is to remember the “Central Dogma” - the sequence of a gene (DNA) encodes the sequence of a protein. Next, proteins are made of combinations of 20 amino acids. Each amino acid shares a common chemical structure (NH2-CHR-CO2H) but with a different R-group. The amine (NH2) of one amino acid can link to the carboxylic acid (CO2H) of another to form a peptide bond. The sequence of a gene dictates the order of amino acids and how many are linked to form a particular protein. This means that proteins are diverse in both sequence and size.

Where do the different amino acid building blocks required for a protein come from? Depending on what organism you are – bacteria, plant, or human – the answer varies. Amino acid building blocks are either made de novo from other materials by an organism’s metabolism or are obtained from diet by digesting consumed proteins into amino acids. For example, plants contain all the metabolic pathways necessary to make the 20 amino acids. In contrast, humans only produce ten of the amino acids and need to obtain the other ten ‘essential’ amino acids from our diet because we lack the pathways for their production. Eating proteins made by other animals and plants leads to recycling of those amino acids for our metabolic purposes.

Consider a stir-fry plate with chicken, rice, and broccoli. That’s nearly 100,000 different proteins sitting on the plate! The chicken has approximately 25,000 genes and rice and broccoli each have approximately 35,000 genes encoding a variety of proteins. Imagine how many proteins a human consumes in a given day – and the more varied the diet, the more diverse the set of proteins.

So, when a transgene (i.e., DNA) is introduced into a plant, that plant produces the encoded protein from endogenous amino acid building blocks. This means that the building blocks of a protein encoded by a transgene expressed in genetically-engineered rice are no different than in any of the other approximately 35,000 proteins in rice.

With that in mind, the protein encoded by the transgene can undergo extensive toxicological and allergen testing, but the depth of the testing depends on the situation. A typical analysis involves bioinformatics analysis to determine how similar the protein is in sequence to known toxins and allergens; studies on the impact of digestion and heat on protein structure and function as it relates to dietary exposure; and on documenting a history of safe use.

Let’s take the case of using rice protein A as a transgene in rice B. If the protein is unrelated to toxins and allergens, is readily digested or denatured by heat, and is already eaten because we eat rice A (i.e., has a history of safe use), then there is no scientific basis to justify extensive testing. Now, if the transgene was from an organism not eaten by humans, was stable in
digestibility/heat stability tests, or shared homology with known toxins and/or allergens, then these conditions may trigger a more thorough analysis. If there is a scientific reason to justify toxicological and/or allergen testing, the protein can be produced and used for food safety experiments. These are typically 90-day feeding studies, in which the protein encoded by the transgene is feed to animals. It should be noted that in such studies, the proteins can be tested at levels equal to a 75 kg man eating 50 tons of corn in one meal! Once again, these studies are typically performed at levels that exceed expression levels of the transgene encoded proteins in the genetically-engineered plant. A detailed list of the testing of proteins for toxicity and allergenicity is too expansive to be covered here, but the extent of testing should be commensurate to the magnitude of risk associated with the new food.

For more information related to these questions, I suggest reading an article published in 2013 by Laura DeFrancesco, a senior editor at Nature Biotechnology, entitled “How safe does transgenic food need to be?” (Nature Biotechnology vol. 31, issue 9, pages 794-802). This excellent article was an independent and balanced analysis of exactly the types of questions asked here and goes into more depth on this topic.

Read More: bit.ly/2lHVhSJ

ARTICLE: Why there are no long term GMO studies on humans?

By: Layla Parker-Katirae (Independent Expert)

Why aren’t there any long term GMO studies on human? Layla Katirae explores this common question and provides the facts in this Genetic Literary project post. Read an excerpt of the post here.

A very common question or criticism of GMOs is that they are not properly tested, particularly on humans. The spouse and I had a discussion about this a while back and he asked why GMOs weren’t tested like drugs since they’re regulated by the FDA. I’ve read comments such as “I won’t believe GMOs are safe until they’re tested for 5 years on humans and we examine long-term impact”, so I thought we should explore this point.

The regulation of GMOs is based on the principle of “substantial equivalence”, meaning that the nutritional content of the GE crop and the non-GE crop that it originated from is the same. In the past, I’ve reviewed papers that have done comparisons between crops generated by transgenesis (the method used to make GMOs) vs crops generated by traditional cross breeding and mutagenesis. The transgenic crops had far fewer unintended consequences than the crops generated by traditional breeding methods. What remains to be demonstrated is that the protein introduced poses no greater risk to human health than non-GE crops, which is why studies on allergenicity and animal feeding studies are performed.

So “why don’t we do clinical trials on GMOs the same way we do for drugs?” Drugs are designed to cause a change in the human body: that’s the whole point behind them. Since drugs are altering something in humans, it’s important to know the side-effects that they may cause and whether or not they’re causing the anticipated effect (i.e. is it better than placebo). In contrast, GMOs are designed to be equivalent to their non-GE counterparts: they aren’t drugs or nutritional supplements. GE crops which ARE designed to impact human health, such as vitamin-A enriched rice, should be tested in humans to determine if the desired outcome is achieved (i.e that the rice actually delivers vitamin-A to the body). But such studies are not the same as looking for unknown long-term effects.

Curious to read more? Read the full post here (bit.ly/2I8r8Sc).

Read More: bit.ly/1T7w0A0
Q: What concentration of glyphosate remains in GMO corn on my table?

Answered By: Marian Bleeke, Fate and Metabolism Platform Lead, Monsanto Company

The use of a pesticide in any crop, whether conventional or genetically modified, can potentially lead to residues remaining in the harvested commodities. Regulatory agencies such as the EPA evaluate the safety of these residues, based on the highest concentrations that could be expected if the pesticide is used at the highest rates allowed by the label. Because residues typically vary significantly due to the environmental factors such as temperature, rainfall, and soil conditions, residue trials are conducted at multiple locations throughout the growing region of the crop in order to determine the highest potential residues.

These results are used to set a Maximum Residue Level (MRL), also called a tolerance by the US EPA, which is the maximum concentration that is allowed in the commodity. The tolerance will only be established by the EPA if the dietary exposure from consuming that crop commodity, combined with other exposures from that pesticide, is within the safety limit set by the EPA.

While the safety assessment of glyphosate is conducted assuming that all the food you consume contains residues at the maximum allowed for each commodity, in reality the food actually on your plate contains much lower levels of glyphosate, for several reasons. First, the tolerance is the highest expected residues from the highest allowed use rate. Not all growers use glyphosate, and of those who do, not all apply at the highest use rate. Second, most residues will be significantly below the tolerance even when the highest rates are used. Third, there are many processes between the time a crop is harvested and when it is eaten that reduce the amount of glyphosate remaining in the food. Glyphosate is water soluble, and does not transfer into oil, so it can be removed by rinsing or cooking in water, and is significantly reduced during the production and refining of processed foods like sugar, oil and cornstarch.

The amount of glyphosate in the GMO corn on your table, in any other food on your table, or even in your entire daily consumption of food is well below the established safe limits as determined by EPA.

Read More: bit.ly/2lWyfYu

ARTICLE: What can we say about exposure to glyphosate?

By: Michael I. Greenberg, M.D., MPH (Independent Expert)

With regard to glyphosate, it seems the IARC did not consider the overwhelming scientific and medical evidence demonstrating that glyphosate is not a human carcinogen. This includes the largest epidemiological study of farmers ever undertaken, the U.S. National Cancer Institute’s Agricultural Health Study, which failed to find a relationship between glyphosate and cancer. Agencies around the world, including the US EPA and the highly respected German BfR (Agency for Risk Assessment, conducting the EU evaluation of glyphosate) have concluded that there is no risk of cancer with glyphosate use. BfR updated its comprehensive assessment as recently as January 29, 2015 and has criticized the IARC assessment.

In short, the weight of the evidence strongly indicates that glyphosate does not cause cancer—in humans or in animals. The evidence comes from the Ag Health Study which uses a design that is considered the best epidemiology approach for examining the impact of a chemical on actual cancer rates (Blair et al., 2015), and did not find an association with cancer (De Roos et al., 2005).

It is also important to realize that the IARC assessment is what toxicologists call a “hazard assessment,” meaning that a particular chemical might cause a problem under some circumstances. Paracelsus, the “father” of modern day toxicology, said, “The dose makes the poison.” In other words, to translate hazard into risk, you need to know something about dose—a dimension that is critical to understanding potential health impacts of glyphosate on cancer or other clinical conditions. The few animal studies cited by IARC were not repeatable, had tumors at the incidence historically observed for controls and are misquoted by the IARC.

In this case, we can’t compare the human dose of glyphosate to doses that cause cancer in animals because glyphosate
does not cause cancer in animals – the evidence supports the opposite conclusion. We can compare exposure to doses shown to be safe in animals based on a “no effect level.” For glyphosate, the US EPA establishes a safe level of intake for humans, or Allowable Daily Intake (ADI), based on a no effect level, applying a 100-fold safety factor and conservatively assuming the highest possible residues in all the food you consume.

So, first, to state the obvious, the concept of dose doesn’t even come into play if there is no exposure. In other words, if one is never in contact with (i.e. exposed) a chemical, then one cannot possibly receive a dose. If there is a container of glyphosate in your garage, sitting unopened on a shelf, no one is being exposed to it. Likewise, aspirin in the bottle won’t help your headache.

If contact with a chemical does occur (inhaled, ingested or on skin), it is possible that the chemical may enter the body and result in a “dose.” But simply receiving a dose isn’t enough – that dose needs to be high enough and prolonged enough for medical consequences to occur. Likewise, an aspirin tablet may help your headache, but 1/100 of an aspirin tablet won’t.

Further, once a chemical does get absorbed into the body, it can be broken down (metabolized) by or excreted from the body. Metabolism can produce problems if the breakdown products are more toxic or reactive, and some chemicals can persist in the body – but what about glyphosate? Absorbed glyphosate is not metabolized in humans, is not fat soluble, and does not accumulate in the body over time, but rather is excreted unchanged in the urine.

What can we say about exposure to glyphosate? For the public with exposure via foods, we know that the “worst case” estimates, assuming that permitted crops are treated by all growers and have maximum allowable residues, put us at about 1/5 of the ADI. Actual use is far less, and most crops have well below maximal levels, so true exposure is far less than this. In fact, urinary monitoring data available for the US and Europe (which works very well for glyphosate) suggests typical exposures less than 1/100 of the ADI. We also have studies in farmers and farm families (Farm Family Exposure Study) that demonstrate that the majority of farmers had no detectable glyphosate in urine following application, and that wives (all the farmers in this study happened to be male) and children had very low levels and no detectable rise in levels (unless they assisted in application). Farmers who did apply glyphosate had exposures well within the ADI.

Finally, what about consumers using glyphosate products in the lawn and garden setting? Certainly, the farmer data should be very reassuring given the scope of agricultural use compared to typical home application. Even, Kate Guyton, the IARC officer responsible for the IARC session looking at glyphosate said: “I don’t think home use is the issue,” pointing out the higher exposure potential for farmers and professional applicators.

Overall, it appears that IARC has overreached in its opinion by failing to consider the vast body of literature supporting the notion that glyphosate is not a carcinogen. Further, “the dose makes the poison,” and the IARC, even if they are correct, has failed to place potential hazard into a context of actual risk. Real-life exposures to glyphosate via food and the environment are small – the ADI is 100 times less than a dose that causes no observable adverse effect in animals, and most people seem to have exposures more than 100-times less than the ADI. Even farmers applying glyphosate fall within the ADI and consumer use exposures, while not studied in the detail we have for farmers, can reasonably be expected to be similar or smaller.

The weight of evidence strongly suggests that glyphosate does not cause cancer – and if it does, a look at the dose demonstrates a lack of medically significant exposure. Users and the public can be confident that labeled uses of glyphosate products pose no meaningful risk of cancer.

Read More: [bit.ly/2lm5w2E]
Q: Have there been any studies of mineral and vitamin levels in livestock that have been fed round up ready forages and grain to determine whether roundup acts as a chelating agent?

Answered By: John Vicini, Ph.D., Food Safety Scientific Affairs Lead, Monsanto Company

As I looked at your question, there are two sub-questions that can be answered to understand your bigger question. First, does glyphosate chelate minerals in such a way as to alter mineral content of feeds? Second, does glyphosate affect the bioavailability of minerals? I’ll answer both and also base my answer on only minerals. You suggested vitamins, but there is no reason to even suspect glyphosate binds any vitamin and there is no homogeneity in the structure of vitamins that would result in binding of this class of nutrients.
**Mineral Content.** The chemistry of glyphosate is such that it will bind certain minerals. Binding does not mean irreversible (see previous answer from this site). Binding is just part of the full story and chelators actually can facilitate movement of minerals into plants. Other chelators are present in plants and soil and strong chelators, such as EDTA, when added to soil can promote uptake of minerals; however, relatively weak chelators, like glyphosate, do not affect uptake. Composition studies indicate that nutritional content, including minerals, is not affected by spraying glyphosate on corn.

**Bioavailability.** Another issue is bioavailability. In other words, are the minerals in the diet available to be digested, absorbed and available for metabolism. The experiment you suggest has not been conducted because the issue of bioavailability is answered by growth studies. Animals that lack necessary nutrition, including minerals and vitamins, will not grow as expected. In toxicology studies, feeding large amounts of glyphosate has not restricted growth.

Another way to approach this question is to do some simple calculations. We know from chemistry that a single molecule of glyphosate will bind to one divalent cation (such as Fe++) . Chickens are fed diets that typically contain just a few feedstuffs (corn grain, soybean meal and minerals), but their diets are developed to maximize gain with minimal cost. This type of diet can have 100% of the non-mineral feedstuffs coming from GM (i.e. Roundup Ready®). If making the highly-conservative assumptions that all feeds contained the maximum residues of glyphosate (see link to explain MRL) and if all of these molecules irreversibly bound a divalent cation, there would be 0.02% of the recommended dietary amounts of dietary divalent cations bound to glyphosate. If looking only at manganese, about which some claims have focused, it would represent 5 percent of the recommended intake. Even with these conservative assumptions you can see that this is a small portion of the total dietary intake for divalent cations.

We can conclude by study data and understanding the biology that glyphosate would not impact minerals, vitamins, or other nutrients in animal products when glyphosate is used according to the label.

Read More: [bit.ly/2m4RHCh](bit.ly/2m4RHCh)

**Q:** Few years back it was alleged that cattle which grazed in Bt Cotton field died in some villages in Andhra Pradesh India (However no such incidents reported anywhere else). How far this allegation by the activists sustainable?

**Answered By:** Gary Hartnell, Senior Fellow, Monsanto Company

I think the allegation that you are referring to is one from 2006 by the Andhra Pradesh (AP) Sheep Growers association, which alleged that more than 10,000 sheep/goats died after eating Bt cotton leaves.

It was unfortunate that farmers lost that number of animals. However, there are several interesting points about the allegations:

• There was no actual data presented that showed that Bt cotton was the cause of death. It was an easy correlation for the activists groups to make simply because there was Bt cotton grown in parts of the area where the animals had been grazing.

• The veterinarian who performed the autopsies on four to five sheep came to a scientifically reasonable conclusion that the sheep deaths may be due to insecticide poisoning.

• Large amounts of Bt Cotton were grown in India starting in 2002 with no adverse reports of animal deaths until this large amount in 2006. It also has not been reported since.

What is known is that safety studies on goats, cows, buffaloes, chicken and fish were conducted as part of the regulatory process for the approval of Bt cotton in India. Mahyco, an Indian seed company, conducted multiple field trials and did extensive nutritional and bio safety studies with Bt cotton in co-operation with many national institutions. All the data generated from the trials and studies were submitted to the Indian Regulatory authorities prior to approval in 2002.
In addition, a 90-day goat feeding study was conducted by Industrial Toxicology Research Center, Lucknow in 1998. The treatment groups included goats fed Bt cottonseed and control groups that were fed non-Bt cottonseed. According to Dr Vishwanathan, the scientist involved in this goat feeding study, “The feed analyses showed the similarity in nutrient and toxicant compositions between Bt & non-Bt cotton seeds, feed intake, weight gain, hematology and serum enzymes were measured for each animal during the feeding period of the study. At the end of this study, the animals were assessed for gross pathology and histopathology. It was concluded from the result by the above analyses that Bt cottonseed is as wholesome and safe for animal feed as non-Bt cottonseed. The differences observed across 48 goats in gross pathology and histo-pathology was attributed to any cottonseed feeding treatments, and was typical for goat feeding on cottonseed.”

International independent scientists also weighed in on this allegation and dismissed it. One particular Op-Ed written by Dr. Shanthu Shantharam and Dr. C. S. Prakash can be found at the following link: http://www.biospectrumindia.com/biospecindia/news/155705/bt-culprit

This is a good example of an unfounded allegation that is propagated in social media to disparage GMOs. I appreciate the opportunity to share information that hopefully dispels this myth.

Read More: bit.ly/2kHhob6

Q: I understand GM maize is grown in the US for cattle feed. Is any of this sold to the UK agricultural sector for cattle feed? And if so, it is known that maize has a deficit of certain nutrients [which necessitate cattle fed on maize being also fed supplements], is there a similar deficit in GM maize or are there any different nutrient deficits with GM maize?

Answered By: Gary Hartnell, Senior Fellow, Monsanto Company

Thanks for your question. I’m going to break down my response to make sure I answer all of your points.

• First, contrary to some Internet rumors, GM maize is not just used for animal feed in the United States. GM maize is grown for the same uses as non-GM maize, and they are not different in their nutrient composition. According to USDA, approximately 45 percent of corn grown in the United States is used as feed for animals. In animal nutrition, individual feeds that make up the total diet are selected for specific reasons. The nutritional reason animals are fed maize is that it is a good source of energy (i.e., calories), both as a result of digestible energy per pound of feed and because animals readily consume it.

According to USDA-ERS, corn processed for human consumption and industrial uses accounts for about one-third of U.S. corn utilization. Dry millers process corn into flakes for cereal, corn flour, corn grits, corn meal and brewers’ grits for beer production. It also can be wet-processed into high-fructose corn syrup (HFCS), glucose and dextrose, starch, corn oil, beverage alcohol, industrial alcohol and fuel ethanol. Approximately one-third of the corn used for ethanol production comes back as animal feed in the form of distillers’ grains.

• Second, regarding your question about the U.K., the U.K. imports most of the maize it uses, but less than 5 percent of it is used as feed. In the U.K., wheat and barley are more commonly used as energy sources in diets of animals.

• And last, regarding the nutritional composition of GM maize and maize in animal feed, diets for animals need to be balanced for many nutrients. For example, here are the numbers for protein, which often is the most expensive nutrient in their diet. Compared with other feeds, maize is relatively low in protein (9.4 percent) compared with the nutritional requirements of many animals (e.g., 18 percent for a high-producing dairy cow). Adding soybean meal (53.4 percent protein) to diets is a good way of achieving nutritional goals for protein. In fact, it stands to reason that prices of commodity feeds are related to their protein content.
As has been discussed in previous answers on this site, and contrary to Internet myths, all GM products go through comprehensive composition testing. Regardless of whether derived from GM or non-GM, the maize grain is nutritionally equivalent. This manuscript has data on the nutritional composition of SmartStax corn, which has eight genes added by GM technology, compared with near-isogenic conventional corn.

Read More: bit.ly/2m0Kfmk

Q: How do you prevent pesticide from being consumed by grazing cattle, goats, etc ?? Monsanto and friends just shift the toxins from the contaminated soil directly into the chain of the ecosystem. So insects or cattle that feed off of the toxic rich plant will also get contaminated and the heavy metals will migrate within months not years, all the way up the food chain until they reach the top predator which unfortunately happens to be us humans.

Answered By: Bryan Delaney, Ph.D., Research Fellow, DuPont Pioneer

Biotechnology has nothing to do with heavy metals, so I’m going to focus on the question it appears you’re asking about Cry proteins (i.e., Bt proteins), which have been expressed in genetically modified (GM) crops to protect them from insect damage. You offer an interesting perspective, but remember that the soil is just soil (as it has always been) and it contains bacteria (just as it always has) and some of that bacteria is Bt, or Bacillus thuringiensis (just as it always was). These bacteria are ubiquitously distributed in the environment, and therefore, their Cry proteins also are ubiquitous. Thorough testing has shown that modifying a crop plant to express Bt proteins is safe. And in fact, these insect-resistant crops mean decreased use of chemicals that are used to protect against insect damage.

With regard to a Bt protein moving “up the food chain” - it will not happen. The human digestive system treats Bt proteins the same way it treats dietary proteins. In other words, it easily breaks them down into individual amino acids and small peptides that are absorbed for nutritive purposes. Bt proteins have long been used in agriculture without any effects on mammals, birds or other non-target (non-pest) species. They have a very narrow spectrum of activity because they have to bind to specific receptors on the lining of the gut of insects, and those receptors are not present in these other organisms.

Read More: bit.ly/2m0IUW6

There are more than 400 studies of animals fed GM crops and the weight of evidence is that GM crops are as nutritious as conventional crops

John Vicini, Ph.D., Food Safety Scientific Affairs Lead, Monsanto Company
**Summary**

The short answer to your question is that long-term feeding studies with GM crops have already been conducted with both livestock and rodents (mice and rats) as research projects by public-funded organizations. None of them have convincingly shown any adverse effects.

In addition to studies with geriatric animals as mentioned in the question, multi-generational feeding studies were conducted with several generations of animals continuously fed GM crops over long periods of time. Again, no adverse findings have been observed (see review publications: Snell et al. 2012, Van Eenennaam 2013, Bartholomaeus et al. 2013, and Herman et al. 2014).

**Detailed answer**

The safety assessment of GMO seeds and agricultural products is based on a “weight-of-evidence” basis, meaning that a combination of information is collected and analyzed in order to define whether or not a GM crop is as safe as conventional products already safely consumed. To date, more than 100 regulatory submissions have demonstrated (1) compositional equivalence and (2) comparable levels of safety between GM crops and their conventional counterparts.

Despite the fact that the scientific weight of evidence from these hundreds of studies have not revealed unique risks associated with GMOs, some groups have been calling for more animal feeding studies, including long-term rodent studies. In order to address these interrogations, research projects entirely supported by public funds have addressed the following question: “Do long-term (up-to 2 years) and multi-generational GMO feeding studies provide any new evidence indicative of some adverse effect(s) that were not previously identified in the usual compositional equivalence and safety studies?”

The most-recent example of these public-funded studies is the GRACE project, funded by the European Commission (GMO Risk Assessment and Communication of Evidence, >http://www.grace-fp7.eu). One objective of the GRACE project was to assess the need and scientific added-value of (1) conducting a 90-day rat feeding study in all cases and (2) extending the duration of 90-day rat feeding study for GMOs. The project found that when the composition of the GM plant is equivalent to its conventional counterpart, or when there are no other indications of toxic effects, there is no indication that a 90-day rat feeding study would provide any new evidence of some adverse effects. In addition, extending the duration of the safety study (up to 2 years) would not bring any additional value, in the absence of a clear and reasonable scientific testing hypothesis. This conclusion is in line with the position of the European Food Safety Authority (EFSA) concerning the risk evaluation of GM crops:

“When molecular, compositional, phenotypic, agronomic, and other analyses have demonstrated equivalence of the GM food/feed, animal feeding trials do not add to the safety assessment.”

---

**Answered By:** Jean Baptiste-Rascle, PhD, Regulatory Toxicologist, Human and Animal Safety - Seeds, Bayer
A number of long-term (of more than 90 d and up to 2 years in duration) feeding trials and multigenerational studies conducted by public research laboratories using various animal models including pigs, cows, quail, and fish have also been reviewed (Ricroch, 2013; Ricroch et al., 2013; Snell et al., 2012). Significant among these studies are two thorough multigenerational studies that examined the long-term effects of feeding a genetically engineered (GE) corn variety (MON810, expressing the insecticidal Cry1Ab protein from Bacillus thuringiensis [Bt], one of the few GE corn varieties approved for cultivation in the EU) to food-producing animals, specifically, a German study in dairy cattle and an Irish study in pigs. These studies were notable in that they included appropriate controls consuming isogenic non-GE lines of corn, and both comprehensively examined a range of phenotypes and indicators of growth and health and also used sophisticated techniques to look for the presence of recombinant DNA (rDNA) and Bt protein in the tissues and products derived from these GE-fed animals (Guertler et al., 2010, 2012; Steinke et al., 2010; Walsh et al., 2011, 2012 a, b, 2013; Buzoianu et al., 2012 a, b, c, d, 2013 a, b).

Q: I have heard that the Seralini study was the first animal feeding study on GMO foods that was done over the full lifetime of the animals and looked at a broad range of health effects, and ended up published in a peer reviewed journal. I know the study has its critics and its defenders. My question is this, are there any GMO feeding studies published in a peer reviewed journal and conducted over the full lifetime of the animals that look at a broad range of health effects and show that GMO's are safe?

A: A number of long-term (of more than 90 d and up to 2 years in duration) feeding trials and multigenerational studies conducted by public research laboratories using various animal models including pigs, cows, quail, and fish have also been reviewed (Ricroch, 2013; Ricroch et al., 2013; Snell et al., 2012). Significant among these studies are two thorough multigenerational studies that examined the long-term effects of feeding a genetically engineered (GE) corn variety (MON810, expressing the insecticidal Cry1Ab protein from Bacillus thuringiensis [Bt], one of the few GE corn varieties approved for cultivation in the EU) to food-producing animals, specifically, a German study in dairy cattle and an Irish study in pigs. These studies were notable in that they included appropriate controls consuming isogenic non-GE lines of corn, and both comprehensively examined a range of phenotypes and indicators of growth and health and also used sophisticated techniques to look for the presence of recombinant DNA (rDNA) and Bt protein in the tissues and products derived from these GE-fed animals (Guertler et al., 2010, 2012; Steinke et al., 2010; Walsh et al., 2011, 2012 a, b, 2013; Buzoianu et al., 2012 a, b, c, d, 2013 a, b).

Q: If critics claim Seralinis well known study is flawed for having used the Sprague Dawley rat, doesn't that make Monsantos two year carcinogenicity studies using the same SD rat, flawed as well? Im confused about what seems to be a double standard?

A: The simple answer is: The criticism of Séralini’s use of rats is not about the fact that they were Sprague Dawley (SD) rats. It is that the number of SD rats he used was not appropriate to draw the conclusions he did. SD rats are acceptable to use in carcinogenicity tests as long as the experiment is designed to account for the fact that SD rats are known to have a high rate of certain spontaneous diseases (e.g., mammary tumors) (Brix et al., 2005).

The more technical explanation is: Spontaneous disease is not unique to SD rats; other rat strains have high background rates of different diseases as well. Thus, rodent carcinogenicity studies must be appropriately populated (i.e., have sufficient statistical power), not only to detect an increased incidence of rare tumor types, but also to discriminate treatment-related effects from spontaneous, or background, incidence of common tumor types.

Perhaps a good way to illustrate this is to consider an extreme example:
A group of researchers want to use noninvasive techniques (e.g., ultrasound) to determine if a certain diet increases heart damage in dogs, and need to pick a breed to study. One breed, the Cavalier King Charles spaniel, is known to get heart damage—usually involving the mitral valve—at a rate of approximately 90 percent. Given this high rate of background incidence, it would be difficult to pick up an increase in mitral valve damage in the animals fed the test diet. However, the researchers could address this issue through two remedies: 1) use another breed, one that does not have a high background incidence of heart damage or 2) use the Cavalier King Charles spaniel, but use more dogs. More dogs lead to greater statistical power to detect differences, even with the background incidence.

This is especially critical in the field of toxicology, where it has long been established that the dose is important (i.e., more of a toxic substance is more toxic than less of that substance). This problem can be solved 1) by using a strain with a lower expected incidence of a certain finding (this can be difficult to determine ahead of time) or 2) by using more rats (referring back to the Sérinali study using the SD rats) to improve the ability to detect the difference between treatment groups.

For the reasons provided above, “US (US EPA, 1998; FDA, 2006) and OECD (1995a) regulatory guidelines for the conduct of carcinogenicity studies in rodents specify the use of at least 50 animals per sex per treatment group. In addition, OECD states that ‘it is unlikely that a regulatory authority would find a study using a lower core number of animals per sex and per group acceptable for regulatory purposes, since a sufficient number of animals should be used so that a thorough biological and statistical evaluation can be carried out.’ (OECD, 1995b). OECD further states that ‘for strains with poor survival such as SD rats, higher numbers of animals per group may be needed in order to maximize the duration of treatment …’” (Hammond et al., 2013). The Sérinali study (2012) included only 10 rats/sex/group, and this low number is inadequate to make meaningful comparisons in tumor incidence between groups at the end of a chronic study—especially when one considers that older female SD rats are known to have high spontaneous rates of mammary and pituitary tumors (EFSA, 2012; Hammond et al., 2013). EFSA (2012) pointed out that these facts were ignored in the discussion of findings in the Sérinali publication (2012), in which the authors claimed that the mammary and pituitary tumors observed in test animals were treatment related.

These OECD guidelines are very familiar to toxicologists. Considering that all of the information above is publicly available, one can only wonder why Sérinali et al. chose not to discuss their findings in the context of existing knowledge of spontaneous disease rates in SD rats.

References Provided By Expert: bit.ly/2m4ovwq

Q: Kevin Folta doesn’t answer the question of effects of glyphosate and adjuvants on human gut microbes. Evidence of disruption is available for poultry Shehata et al., 2013, cattle Krger et al., 2013, and swine Carman et al., 2013.

Answered By: John Vicini, Ph.D., Food Safety Scientific Affairs Lead, Monsanto Company

Answer at a Glance:

• Gut microbes are an important part of gut function, but it is also know that they are dynamic and change in response to gut conditions.

• In vitro studies with single species can be done with microbes but are not always predictive of in vivo results with mixed populations.

• Measuring a change in gut microbes is not suggestive of health impacts.

• Two studies using formulated glyphosate added to mixed microbial populations (one each in vivo and in vitro) have not resulted in meaningful changes.

• The three studies cited, provide no evidence of microbial disruption in animals.
You asked about glyphosate effects on gut microbes and specifically call out three studies as providing evidence of disruption. Folta’s answer has some good points but was from a broad perspective, so this answer will be a longer and a more technical answer than you might see typically on a site like this. First, I’ll make some generic comments and then some comments about the specific studies you cited.

Currently, gut microbes are one of the hottest topics in biology. There is no doubt that they are important, but we need to also understand that microbes are dynamic and a change does not necessarily mean that there is a health impact. We’ve known for a long time that there are differences in microbiome due to an individual’s personal physiology and differences due to diets. Many animal models exist, including rodents, but ruminants (like cows and sheep) can be a good model for studying gut microbes, because much early information came from them and most of the gut microbes in ruminants and the human gut are anaerobes, meaning they do not survive in an oxygen-rich environment. It takes special skills or equipment to culture these. Also, ruminant microbes are more accessible allowing researchers to take frequent samples and determine temporal changes in great detail.

But scientists also need to determine how things work or how they are affected by the environment by eliminating noise, or all of the confounding factors that make conclusions about results difficult. This is often done in a test tube (in vitro) while recognizing that it only is part of the complexity that exists in the whole animal (in vivo) in a real world exposure. For instance, if I want to look at the impact of drinking coffee on gut microbes, I can add coffee to a microbial culture in a test tube. If it contains a single species, this is known as a mono-culture. I might find out that certain species don’t tolerate this well and with further experiments I could determine if the effects might be due to pH changes (acidity of the coffee) or due to fatty acids from cream, or due to a chemical in the coffee such as caffeine or that dreaded pumpkin spice. I could also do the same experiment with multiple species of microbes, known as a mixed-culture, and I may get a different result. That’s because each bacteria in the culture has different requirements to grow and fills a separate niche making the total metabolic activity in that tube much more complex. To paraphrase a professor of mine, “one microbe’s waste is another microbe’s food”.

There have been numerous long-term rodent studies with glyphosate fed at large amounts. If it had an impact on health, as a result of effects on gut microbes or through another mechanism, these studies are a good way to demonstrate if there is actually an impact on health. We’ve talked a lot about numbers of animals and strains of rodents on this site, but a very important thing to understand from these studies is the number of endpoints that are measured and the detailed observations. Rodents are important model animals in gut microbe research, which is well illustrated by the studies where lean mice are made obese by fecal transplants from obese humans. One unfounded claim is that glyphosate changes microbes in the gut and the pathological sequela would be gut inflammation; however, gut histology from rat toxicology has not indicated this to be an issue.

There are two studies that have examined effects on gut microbiology and microbial function that use mixed-population approaches. One is in vivo, and the other is a more sophisticated in vitro study. The first study [1] was an in vitro study that used a specialized apparatus for maintaining a normal ruminal microbial population under controlled conditions for a long period [2]. The German Federal Institute for Risk Assessment (BfR) commissioned a study with public money using this technique to investigate two objectives. First, to determine if quantitative composition of ruminal microflora or ruminal metabolism might be altered with glyphosate. Second, to determine if there is evidence of C. botulinum overgrowth (i.e., did treatments favor growth of C. botulinum over other microbial species). To meet the first objective the effects of a formulation of glyphosate containing a tallowamine surfactant on rumen fermentative parameters were studied. No major changes in rumen parameters were detected except slight decreases in ammonia nitrogen (due to microbial breakdown of amino acids) concentrations and increases in isovalerate (due to fermentation of organic compounds) production in response to the higher dosage. There was an increase in Bifidobacterium spp. (generally considered beneficial) but the Clostridia were not affected. In the second trial, growth of C. sporogenes (inoculated to the rumen fluid as a surrogate for C. botulinum) did not affect the resulting Clostridia community.

The second study was an in vivo study that used rumen cannulated sheep fed diets with formulated glyphosate added at the most conservative scenario based on the highest glyphosate residues determined in grass three to eight days after application [3]. An additional treatment had supplemental aromatic amino acids to see if they would reverse potential effects of glyphosate, assuming they would be limiting for bacterial growth. There was no indication that the rumen microbes were
affected with or without aromatic amino acids based on rumen pH, NH3-N, and VFAs. Additionally, in situ digestibility of NDF and DM were measured using Dacron bags suspended in the rumens and there was no effect of treatments. These latter endpoints indicate that rumen function was unaffected by Roundup®.

These two studies share some features that more accurately predict real world effects. As stated previously, both use mixed populations of microbes. They also used a continuous (the sheep study) or semi-continuous (the Rusitec study) turnovers. In human and animal intestines, there is a fairly continuous influx of nutrients and there is a similarly steady outflow of gut contents, including microbial cells. This results in the population of microbes being in a steady state condition. This is important not only because it affects microbial responses to treatments, but it eliminates concerns about when you take samples. You could run a system like this for indefinite periods and take samples days or weeks after initiating the experiment. Most important, these studies, which are good models of real world conditions, do not indicate that there are meaningful effects of glyphosate on gut microbes.

What about the papers you cite as “evidence of disruption”?

**Poultry - Shehata et al., 2013.** Simply put, there are no samples from a chicken given Roundup in this study [4]; therefore, there is no evidence of disruption. They grew in vitro monocultures of selected bacterial species and measure their “growth” when formulated Roundup was added to the culture media. At best, these results can demonstrate a potential but they don’t do a good job of that. The study is difficult to interpret because they leave out a lot of detail about how the study was conducted. Growth is determined from a single point in time at 48 hours. That time is probably too long, because the bacteria eventually run out of nutrients and die in their own waste products, one cannot ascertain if the value measured is from growth increasing or decreasing. This is very important since growth rate of bacteria has a major influence on the outcome of all such tests. Brown et al. [5] stated that “an under recognized but major determinant of such physiology is the rate of cell replication”. Shehata et al. suggest that Roundup differentially affects good bacteria vs. bad bacteria and this assumes that good and bad are defined by a susceptible EPSPS enzyme for which there is no basis. There are several examples among their small selection of organisms of a “bad” bacteria being more susceptible than a “good” bacteria and that also assumes that a bad bacteria is always a bad bacteria (not such a great assumption).

**Cattle - Kruger [sic.] et al., 2013.** According to their introduction, the reason the study was done was due to farms in Germany that were suspected of having a rare form of visceral botulism. Normally, cattle get clinical botulism from ingesting a toxin (BoNT) produced by Clostridium botulinum, not due to infection by the organism. Their theory was that due to dysbiosis there is an overgrowth of C. botulinum in the affected cows resulting in BoNT being produced and absorbed from the gastrointestinal tract. No reason was identified and they were hypothesizing, without direct data that this clinical outbreak may have been due to glyphosate. Seyboldt with a team of veterinarians investigated this situation [6]. They sampled 1388 animals from 139 farms in these region and found no evidence that any animal had NoBT in its feces and they considered fecal detection of NoBT to be a requirement of the hypothesis. Kruger et al. [7] did not study any animals or obtain any samples from animals suspected to clostridia or even dysbiosis. Instead, they did testing of mono-cultures to which glyphosate or formulated Roundup was added to the culture media. They then compared the minimum inhibitory concentrations (MIC) of Roundup or glyphosate added to culture media for mono-cultures of a small number of bacterial species. They found a small difference between the MIC for C. botulinum vs. E. faecalis but these MICs are greater than glyphosate intake and are not informative of similar effects in vivo or on resulting health impacts. These conclusions are a big leap of faith. Additionally, they don’t rule out if these effects are due to surfactants (formulation) or pH (glyphosate), either is a possibility. It is noteworthy that if you read the abstract, they don’t present any data whatsoever from the study and carefully limit their conclusion to glyphosate possibly contributing to a health malady that had not even been shown to occur.

**Swine - Carman et al., 2013.** This study [8] was addressed previously on GMO Answers. There are no measurements related to microbiology in this paper. The authors discuss “red” stomachs that are not statistically different when analyzed using all of the data. There were no cultures to show infections and no tissue biopsies to show inflammation. Moreover, they did not report if the pigs in this study ever consumed glyphosate. But even assuming for the sake of argument that they did, the authors state that there was no difference in body weight gains between test and control animals, suggesting there were no health effects. Other signs of clinical illness were not reported.
These studies share that fact that there was no indication among any data of an abnormal health finding related to glyphosate. The information that led to the question answered by Dr. Folta is taken from a series of correlations between glyphosate use and various diseases. The disease data are then correlated with various purported causes in the scientific literature. For an example of how misleading such correlations can be, see http://rounduproulette.com. Seneff’s work is based on supposition derived from correlations identified using data obtained from disparate sources and there is no actual demonstration of real world exposures of glyphosate to back her claims.

Q: How do you respond to the recent independent studies done by Dr. Judy Carmen of the Institute of Health and Environmental Research? The one I am referring to can be found here: http://www.iher.org.au/publications.php?pubID=16 They did a longterm (22.7 weeks, the life of a commercial slaughter pig) study on the toxicology of pigs being fed a GMO corn and soy diet. Read the study. The results showed gastrointestinal and reproductive issues in autopsy of both male and female pigs. So in the studies done by the FDA the findings were either not included in the results or the study was flawed. What is your response?

Answered By: Wendelyn Jones, Global Regulatory Affairs, DuPont Crop Protection

As an employee of the agricultural biotechnology sector, I can attest that the industry takes any new studies related to the safety of GM crops very seriously. As such, I reviewed the paper by Carman et al. with interest at the time it was published and reread in order to best answer your question. The paper reports that pigs fed GMOs had inflamed stomachs based on visual evaluation of redness. However, the paper also shows that pigs fed non-GMO diets also had inflamed stomachs. The authors chose not to comment on the table in the paper that shows there were more pigs with inflamed stomachs that had eaten the non-GMO diet than the GMO diet. Stomach inflammation is common in animals with high feed intake or feed that has been finely ground. The feed intake is not reported in this paper. Without additional information about the study (beyond that reported in the paper), other, non-feed-related factors could account for the observed results.

Swine in the United States have been consuming GM grains for more than 15 years; this includes the breeding herd. According to USDA, the reproductive performance of the breeding herd has improved steadily over that period. Without missing details and selected results analyzed in the Carman et al. paper, the paper lacks credibility in opposition to the hundreds of food and feed safety studies conducted on GMOs and the 15 years of practical experience. DuPont Pioneer has directly investigated GM and non-GM grain in livestock diets as well, and we have not detected any health or performance differences. See here for an example of one such study, or check out the Biofortified website, where independent experts are reviewing the science of plant genetics and working on a list of the more than 600 studies related to the safety of GM crops.

The Food and Drug Administration (FDA) and other regulatory authorities worldwide conduct their own peer reviews on the science provided by biotech product developers. Additionally, as scientists, FDA reviewers also review the published literature. This process helps ensure the safety of food from GM plants.

Read More: bit.ly/2kEAunc

Q: Please thoroughly explain the troubling findings of the recent, peer-reviewed journal, by Judy Carman et al., titled, “A long-term toxicology study on pigs fed a combined genetically modified (GM) soy and GM maize diet.” Can you ensure the health and safety of animals fed GMO-feed? The article is here: http://www.organic-systems.org/journal/81/8106.pdf

Answered By: Community Manager
This response by Cami Ryan, research associate at the College of Agriculture and Bioresources at the University of Saskatchewan, addresses the study referenced in your question. An excerpt from the response is included below:

“From ‘I smell a rat’ to ‘when pigs fly,’ bad science has been making the rounds of late. The multi-authored article ‘A long-term toxicology study on pigs fed a combined genetically modified (GM) soy and GM maize diet’ [referenced in the question] reports that pigs fed a diet of only genetically modified grain show a markedly higher incidence of stomach inflammation than pigs that ate conventional feed.

“However, it seems that—post-publication—the paper and its evidence fail the independent peer-review process on many fronts:

“David Tribe reviews the paper here: He says, ‘It's what some call a fishing expedition in search of a finding, and a known pitfall of animal feeding trials on whole foods…’ Tribe points out (among other things) that some of the study’s observations might be attributed to compositional differences in the variety of soybeans or corn fed to the pigs: ‘[T]here is relatively little information in the paper about nutritional formulation, methods used for producing the pig diets, storage time for the grain and which particular varieties of grain were used in the diets.’

“Anastasia Bodnar expands upon this further in her Biofortified post ‘Lack of care when choosing grains invalidates pig feeding study’: ‘The authors aimed to do a real world study, with pig feed that can be found in real life. It intuitively seems right to just go get some grain from some farms. After all, that is what pigs eat, right? Unfortunately, it’s just not that simple…To hone in on any differences that may be caused by the GM traits, they would have to use feed with one or more GM traits and feed that doesn’t have the GM traits but that is otherwise as similar as possible. If the feeds aren’t very similar, then we can’t know if any differences in the animals is due to the GM traits or due to something else.’

“Dr. Robert Friendship (via Terry Daynard)—swine expert from the University of Guelph—points to methodological problems with ‘visual scoring’ and assessment of ‘inflammation’: ‘[I]t was incorrect for the researchers to conclude that one group had more stomach inflammation than the other group because the researchers did not examine stomach inflammation. They did a visual scoring of the color of the lining of the stomach of pigs at the abattoir and misinterpreted redness to indicate evidence of inflammation. It does not. They would have had to take a tissue sample and prepare histological slides and examine these samples for evidence of inflammatory response such as white blood cell infiltration and other changes to determine if there was inflammation.’

“Andrew Kniss clearly demonstrates the failings of the statistical analysis, poking holes in the study’s evidence. He states, ‘If I were to have analyzed these data, using the statistical techniques that I was taught were appropriate for the type of data, I would have concluded there was no statistical difference in stomach inflammation between the pigs fed the two different diets. To analyze these data the way the authors did makes it seem like they’re trying to find a difference, where none really exist.’

Read More: [bit.ly/2lehE54]
Q: First, how do you respond to the recent study showing leaky gut and other health conditions in pigs over a 22.7 week period (the average length of time spent at a U.S. Piggery)? Please feel free to read through the study material here: [http://www.organic-systems.org/journal/81/8106.pdf](http://www.organic-systems.org/journal/81/8106.pdf) Second, what evidence can you provide to counter the information showing an increase in a variety of gastrointestinal diseases coinciding perfectly with the introduction of GM foods in the mid-90s? Finally, is it not factual that what Monsanto and other biotech companies call an increase in crop yield is actually due to other factors, such as replacing diverse fields (full of many different crops) entirely with cotton or wheat and then stating how many more kilos of cotton or wheat are being produced? Is it not common sense that if you plant more your yield will be more? Respectively, how do you feel that places like India are showing that organic gardening produces far more output per square foot than genetically modified crops?

Answered By: Rashmi S. Nair, Ph.D, Director, Emerging Markets, Regulatory Policy & Scientific Affairs, Monsanto Company

Answer at a Glance:

- There is no commercialized GMO wheat. The data do not support that the cotton yields are increased only because more seeds are being planted; however, there are data to support the fact that when an equivalent amount of GM cotton seed is planted side by side with an equivalent hybrid of conventional cotton, the overall yield from the GM cotton plant is higher than the conventional hybrid.

- India does have the largest numbers of organic farmers, but the reason for that could quite easily be the small area that each farmer farms. But it should be noted that no yield data exist to show that the 1 million hectares of India’s 179.9 million hectares of agricultural land that are used for organic agriculture are more productive than genetically modified crops.

Farmers adopt new agricultural technologies because they see benefits like increase in yield, reduction in input costs or overall increase in ease of farming. For example, in India, several reports from researchers both outside India and within various states have documented that the adoption of insect-protected cotton has increased from a few thousand hectares in 2002, when the technology was first approved in India, to 11 million hectares (over 90 percent of the total area where cotton is grown) in 2013 ([ISAAA](http://www.isaaa.org/)), this rapid adoption was led by an overall increase in profitability due to higher yields and reduction in pesticide costs (Kathage and Qaim, 2012; Mayee and Chaudhary, 2013).

Kathage and Qaim studied the effects of Bt cotton adoption from 2002 through 2008 and showed that adoption of Bt cotton has increased yields and profits by 24 percent and 50 percent, respectively and the impacts of Bt cotton have been stable over time. Mayee and Chaudhary (2013) recently published their report on the socioeconomic benefits of adoption of Bt cotton in three states — Punjab, Maharashtra and Andhra Pradesh — from 2002 to 2011, and their results showed doubling of yields in all three states in both rain-fed and irrigated conditions. Mayee and Chaudhary also concluded that “Bt technology has decreased pesticide usages, increased cotton productivity and increased farmers’ income and contributed significantly to poverty alleviation.” I also personally heard from cotton farmers in India, when I stayed there in 2010-11, that Bt cotton had significantly improved their lives and they would like to see additional innovative agricultural technologies to help them produce more with fewer inputs.

While I was unable to find any actual data to support your statement that yields of cotton and wheat have increased only because more seeds are planted, it should be noted that while yields of cotton have increased significantly in the last ten years in India, yields of other crops have not decreased but have shown slow to moderate increases. I totally agree with you that yield comparisons have to be done by planting fields with a given GM and conventional crop with similar agricultural practices; only then can one make the claim that there is an increase in yield. Please note that the Indian regulations for approval of GM crops require such data to be generated for every new GM event, and that the developer provided the authorities with these data. Meanwhile, farmers base their decision on what to plant every year on the overall potential profits from the crop being planted, so the sustained increase in acreage of cotton in India suggests that most farmers in India are reaping the benefits of Bt cotton.
Health conditions in Pigs: The study you are referring to is (Carmen et al., 2013) and I have personally been involved in several comprehensive animal health studies like this, in which we collaborated with teams of veterinarians, pathologists, nutritionists, etc. What I’ve learned from that experience and what I find most revealing about this study is what didn’t happen.

The first thing that didn’t happen is that pigs were not properly placed in pens. When animals are administered treatments by pen (i.e. all pigs in a pen are given the same treatment) then the pig is not the experimental unit, the pen is. They seem to have done a statistical analysis with pig as the experimental unit, which will result in false positives.

The next thing that didn’t happen is leaky gut. There was nothing measured in the entire study that would be related to or even suggest leaky gut. What they did “claim” that’s related to the gastrointestinal tract was inflammation of the stomach. Every first-year med/vet student learns that the signs of inflammation are: dolor (pain), calor (heat), rubor (redness) and tumor (swelling). The only thing they ‘observed’ was red. They could have done histopathology to really understand what they ‘saw’, but for some reason they did not do this. The first thing I noticed was those stomach pictures. Pigs are prone to gastric ulcers that are usually located in the cardia region (where the esophagus enters) and are often bloody. This occurs when pigs are stressed and there was no indication of that. Furthermore, when they looked at these stomachs, that might have been red based on the way they euthanized the pigs, they assigned them to four categories and then they did stats on two of the categories and left out the other two. But the pigs had four possible outcomes, not two. If you appropriately analyze the full data set and assume pig is the experimental unit (like they wrongly did), there is no effect of treatment.

They also reported that there was an enlargement of the uterus and that when they cut one open clear fluid oozed out. That sounds like it came from a pig in estrus (heat). This would have been simple to verify because while they were looking at the uterus they could have found a wealth of information by looking at the ovaries that are found just slightly forward of the uterus. By looking at ovarian structures you can tell if they are pre-puberal, post-puberal, in estrus, post estrus or pregnant. You can see that this is a lot of information that was also not included – and, unlike histology, it doesn’t even cost money to obtain. In our health studies, gross lesions are almost always collected for histopathology.

If you are wondering why these items are not discussed in a peer-reviewed paper, look at the editorial board of this journal and you won’t see animal scientists, toxicologists or veterinarians. It really would have benefited by a peer review of a top-tier animal science journal.

The final thing that did not happen is a change in growth rate (according to Carmen). I’ve learned in 35 years of experience that stressed or sick animals do not grow as well as healthy animals, suggesting that the health of these pigs was similar to that of the control pigs. The goal for swine producers is well known, “happy as a pig in…” . You get the picture.
Gastrointestinal Diseases: The ‘association’ between gastrointestinal diseases and glyphosate is a classic example of association vs. cause & effect. This was well illustrated by the blog post written shortly afterwards (that showed another perfect correlation between consumption of organic food and autism. For the record, I don’t buy that one either. In fact, it is unfortunate when these types of associations distract from making real progress on serious diseases like autism and celiac disease. One website posted many examples of associations and these were the result of computer programs that search thousands of databases looking for associations. The associations of things like divorce rate with margarine consumption are not obvious to people so they are more easily dismissed. But the associations that meet someone’s preconceived expectations become more believable. Your phrase “coinciding perfectly” is actually a red flag. In epidemiology there is recognition of lags that occur in data. Most of our critics think we need to move slowly on new technologies and want long-term multi-generational studies. These plots show no indication of lag. So which is it - longer studies or instantaneous cause and effect?

Recently, in July, Alison Van Eenennaam, who has answered questions on this site, gave a presentation at the Annual Meeting of the American Society of Animal Sciences that showed animal performance measurements for more than 100 billion animals in the US that have gotten diets with GM feeds, some of which are whole grains with no processing other than grinding. Her conclusions are that there are no indications that animal health and productivity have been affected by GM crops. This was published as an “Invited Review” paper in the October Issue of the Journal of Animal Science.

References Provided By Expert: bit.ly/2lDGCM1

In conclusion, long-term feeding studies with GMOs have already been conducted in livestock or rodents (mice and rat) and none of them have convincingly shown any adverse effect.

Jean Baptiste-Rascle, PhD, Regulatory Toxicologist, Human and Animal Safety - Seeds, Bayer
There is big news in the world of biotechnology…the Food and Drug Administration (FDA) recently approved genetically engineered AquAdvantage® Salmon for human consumption.

Salmon is now the first genetically modified animal to be approved for human consumption. Naturally, there are many questions about the new uses of genetic engineering in food, and we'd like to help answer some of those now.

I: What is the AquAdvantage® salmon?

This fast-growing salmon is a genetically engineered salmon that grows to market size in half the time as conventional Atlantic salmon. This salmon grows to market weight in about 16 to 18 months vs. 32 to 36 months for conventional salmon. This salmon contains a growth hormone gene from the fast growing Pacific Chinook salmon and a promoter sequence (a fragment of DNA) from the ocean pout. Combined, the gene and promoter sequence, which acts like an “on” switch, enable the salmon to grow year-round instead of seasonally like wild or farmed salmon.

Research on genetically engineered salmon started in the mid-1980s. After years of research, access to a growth hormone gene and identifying the DNA sequence, the first transgenic salmon was created in 1989, but it wasn’t until almost twenty years later, that the FDA approved this salmon.

A genetically engineered AquAdvantage salmon alongside an Atlantic salmon of the same age.
2: Why genetically engineer salmon to grow faster?

Salmon is one of the most popular fishes available. As of 2014, salmon is the second most consumed seafood in the U.S. Salmon are either caught in the wild through commercial fishing or they are raised via aquaculture (fish farming). Since the 1990's, there has been a decline in wild Atlantic salmon populations.

3: What's responsible for the decline in wild salmon?

- 90 percent of the world's fisheries are either fully exploited, over-exploited or have collapsed. And large fish, like salmon, are the first to go.

- In addition to overfishing, pollution, environmental changes, habitat deterioration and disturbances of migration routes have all contributed to the reduction of salmon populations to dangerously low levels. Many salmon populations have disappeared completely.

And yet seafood consumption will nearly double by 2050.

This means that farmed fish production will likely need to increase by 133 percent to meet projected fish demand worldwide. GM salmon will provide a sustainable and fast-growing alternative to wild salmon and enhance the production of farmed salmon.

4: Is the AquAdvantage® salmon safe?

Yes. Approximately 20 years of research, testing, evaluations, development and regulations went into getting this salmon's FDA safety approval. In fact, all GM food products must be found as safe as their non-GM counterparts before they come to market. Here's what that review process for GMO crops looks like:
5: Even though it's FDA approved, isn't this fast-growing salmon a “Frankenfish”?

As the American Council on Science and Health points out, “the term ‘Franken’ is thrown in front of a lot of biology that anti-science activists distrust and fear because they do not understand.” Scary terms and incorrect labels about GMOs are an easy way to create fear and doubts around the safety of GMOs.

But if we remove the label “Franken” and take a deep dive into biotechnology in the context of the term “Franken,” you will learn that it’s not that scary and that it’s only science and innovations. In this post from The American Council on Science and Health, it helps clarify the term “Franken” in the context of GMOs and biotechnology.

6: How does this salmon impact the environment?

This salmon has been developed to actually benefit the environment. For example:

- Conserve wild fish populations. The AquAdvantage® salmon grows to market-size using 25 percent less feed than any Atlantic salmon on the market today. This means that it requires less wild fish to be converted into salmon feed – which conserves wild fish stocks. Using GE ingredients to replace fishmeal and fish oil requirements in fish feeds are helping aquaculture to be even more sustainable. (Click here to learn more.)

- Reduce carbon emissions. Because the AquaBounty salmon is farmed in land-based facilities that are close to cities, it only needs to travel a short distance to the grocery store. This cuts down on transportation and, therefore, carbon emissions, from farm to table. Transporting the AquaBounty salmon emits 23 to 25 times less CO2 than the two major sources of US Atlantic salmon.

- Provide low impact fish farming. AquaBounty has full control over their aquaculture rearing ecosystem. This means they have total control over water input and discharge, sanitation and the ability to recycle resources. Learn more about the process on AquaBounty’s website.
Other resources on this topic include:

Dan Farber’s article, Could FDA’s Approval of GMO Salmon Actually Be Good for the Environment?, provides some excellent insights.

Reporter Tamar Haspel at the Washington Post discusses the environment risks of the GM salmon in this post.

7: Will it create new allergies?

No, this salmon will not create new allergies. In fact, no GMO crop on the market today creates new allergies, and rigorous testing ensures they never will. If a person is allergic to a non-GM plant or animal, for example salmon, he or she will also be allergic to the plant’s GMO counterpart.

8: How is GMO salmon contained; will it breed with other salmon?

The FDA requires this salmon to be grown in physically contained land-based facilities, further reducing any potential impact or breeding with wild salmon populations. The tanks that house the salmon have netting and screens to prevent escape. All water pipes going into and out of the facility have multiple physical barriers, such as metal screens, filters and pumps that fish cannot pass through.

As an added precaution, all modified salmon eggs are female and sterile, making it impossible for them to breed among themselves and with other salmon.

9: What happens next with AquaBounty’s salmon? When can we expect it to be in the stores?

Although the FDA has approved this salmon for human consumption, it may take a couple of years before it hits store shelves. There are many production planning requirements that need to be worked out before commencing commercial production.

Curious to learn more about the AquAdvantage Salmon? These resources provide more information:

Read More: [bit.ly/2jW0ok0](bit.ly/2jW0ok0)

Q: History of genetic modified salmon

Answered By: Dave Conley, Director, Corporate Communications, AquaBounty Technologies, Inc

Information about the development of the AquAdvantage® Salmon (AAS) from AquaBounty Technologies is available on our website. Initial research and development with transgenic salmon began in 1989, and you can read a full chronology of the AAS and AquaBounty Technologies here.

Dr. Anastasia Bodnar, co-executive director for the Biofortified Blog, wrote about the “science behind the salmon” and the steps taken to develop the AAS on the Biofortified website in 2010.
Dr. Alison Van Eenennaam and Dr. William Muir published a commentary in Nature Biotechnology that discusses concerns raised about AquAdvantage Salmon and whether they are scientifically justified, see Transgenic salmon: a final leap to the grocery shelf?

You might also be interested in these three videos about the production of the AAS and its eggs.

Read More: bit.ly/2mcHrMz

Q: Is genetically modifying salmon equivalent to genetically modifying plants?

Answered By: Caitlin Cooper, Ph.D Candidate, University of California Davis

Essentially, yes. In both cases, a gene that is intended to result in a new, desirable trait is inserted into the genome of the plant or animal, the gene provides the information to make a protein and the protein results in a new or enhanced trait. One difference involves the similarity of the new protein to the proteins produced by the target. Commercial GM plants today use a gene from a different source (other plant, microbe, mushroom, etc.) to make a completely new protein for the plant. Another approach is to add a gene that already exists in the plant to make more of a protein that is already made by the target plant to enhance some attribute. This is called cisgenesis and the result is that there is no new protein. In the case of the salmon that is under FDA review, a gene for growth hormone from Chinook salmon is added to Atlantic salmon to increase their rate and efficiency of growth. Like cisgenesis, the Chinook growth hormone is not new compared with the growth hormone made by Atlantic salmon. But its release profile is different, and this is enough of a difference to increase the salmon’s growth.

To further extend the comparison, GM salmon are evaluated by the FDA for food safety, and the FDA uses some of the same experimental approaches to evaluate safety.

Read More: bit.ly/2lPVozp

Q: Are there any GMO chickens in the world?

Answered By: Ashley Peterson, Ph.D., Senior Vice President of Scientific and Regulatory Affairs, National Chicken Council

There are no genetically modified chickens commercially available. In fact, there are not currently any genetically modified animals commercially available for food. In late 2015, genetically engineered salmon was the first GE animal to be approved but it will not be on the market and available for purchase for a few years. Modern chickens are bigger than ever before, which raises the question – how’d we do that? A number of factors go into raising larger, healthier birds, but genetically modifying chickens isn’t one of them.

So what factors do go into raising larger, healthier chickens? We start with good breeding. Over the years, we’ve selected chickens with the best traits (size, health, leg strength, etc.) for breeding, to help get the best start possible.

Modern advances in farming including state-of-the-art housing, nutrition, climate controls and biosecurity, coupled with good animal husbandry and cooperation between farmers and veterinarians, all help us raise larger, healthier birds.

Read More: bit.ly/25TxkJH
Q: Eduard Kac engineered a rabbit to glow in the dark, just for the sake of art. If I went into the field of genetic engineering, would I be able to do something similar? If I got the necessary qualifications, would I be able to pursue my own interests, or would I be sitting in a lab making corn grow taller every single day? I’m seriously considering studying genetic engineering, but it isn’t worth it to me if I won’t have the opportunity to become the best I can be.

Answered By: Denneal Jamison-McClung, Ph.D., Associate Director, UC Davis Biotechnology Program

Becoming the best scientist that you can be is a worthy goal. As an educator, there is nothing better than seeing students reach their intellectual potential and grow as thoughtful, contributing members of society. As much as I love to see folks having fun and enjoying their work, genetic engineering is a powerful technology and begs the question, “Because we can do this, should we do this?”

In university labs, where serving the public good is the ultimate mission (typically funded by tax payers), fun projects (glowing mammals!) would necessarily take a back seat to more weighty matters, such as crop improvement or the development of new medicines. Research universities typically have institutional biosafety committees that review and approve genetic engineering experiments before the research takes place. We also ensure that aspiring genetic engineers receive training in bioethics. For example, our UC Davis Designated Emphasis in Biotechnology doctoral degree program requires Ph.D. students to complete a course in bioethics.

If you are lucky enough to win the lotto and outfit a private laboratory, then your research priorities may be more flexible… But, even then, there are federal biosafety and bioethics guidelines governing conventional products produced by genetic engineering. In 1986, the Office of Science & Technology Policy (OSTP) issued the Coordinated Framework for Regulation of Biotechnology. The OSTP oversees the work of three federal agencies involved in regulating the products of genetic engineering (USDA, EPA, FDA). Scientists working in industry and research institutes, as well as universities, must abide by practices and policies that align with federal standards. The Law Library of Congress maintains a compilation of resources outlining restrictions on the use of genetic engineering in the United States.

Scientists and policymakers continue to work together to set bioethics and biosafety standards for the “Golden Age of Biology” we are experiencing. For example, in 2014-2015, the National Academies of Sciences, Engineering and Medicine undertook a study of current genetic engineering technologies in food and agriculture, A Science-Based Look at Genetically Engineered Crops (#GECropStudy). The NAS Agriculture committee’s comprehensive report, “Genetically Engineered Crops: Past Experience and Future Prospects” is expected in 2016. The report will provide policymakers with an expert, objective frame of reference to guide public policy decisions regarding the use of genetic engineering technologies in food and agriculture.

To follow national discussions on bioethics, see the Presidential Commission for the Study of Bioethical Issues. Recent reports related to genetic engineering include:

- Privacy and Progress in Whole Genome Sequencing
- New Directions: The Ethics of Synthetic Biology and Emerging Technologies

To learn more about biosafety and regulatory guidelines for federally funded genetic engineering projects, see:

- National Institutes of Health (NIH)
- United States Department of Agriculture (USDA) – Animal and Plant Health Inspection Service (APHIS)

With careful consideration of societal risks and benefits, scientists will continue to advance humanities' understanding of the natural world and develop technologies to solve global challenges in agriculture, healthcare and environmental sustainability.

Read More: bit.ly/2l3SKSp