Biotechnology Regulatory Services
Animal and Plant Health Inspection Service
United States Department of Agriculture
4700 River Road
Riverdale, MD 20737

RE: Docket APHIS-2021-0062
Guidance for Requesting a Regulatory Status Review (RSR) under 7 CFR part 340

Center for Food Safety (CFS) and the undersigned organizations and individuals appreciate the opportunity to comment on APHIS’s guidance for requesting a regulatory status review (RSR) under the SECURE Act, 7 C.F.R., Part 340.

As detailed in pending litigation,¹ the U.S. Dept. of Agriculture’s (USDA’s) new genetically engineered organism regulations, the SECURE Act, are unlawful in numerous respects. Nothing expressed in these comments should be construed as contrary to the claims raised in this lawsuit. USDA should withdraw the current regulations and promulgate new ones that protect American agriculture by properly implementing the Plant Protection Act.

The proposed scope of the regulatory status review is far too narrow to adequately capture significant adverse effects ensuing from cultivation of some genetically engineered (GE) plants, effects that are cognizable under the Plant Protection Act (PPA). As proposed, APHIS’s plant pest risk assessment (PPRA) would focus first on the biological properties of the plant; then train a magnifying glass on the GE trait, and finally a microscope on the biochemical minutia of the mechanism of action that determines the trait. Missing in this proposed scheme is any systematic consideration of the higher levels of organization where many PPA-relevant adverse effects will manifest. APHIS must also pay greater attention to the unintended effects and phenotypes that will occur with most GE plants.

Assess Real-World Impacts
First, APHIS must assess the GE plant in the context of its agricultural landscape and associated cultivation practices. One would think that assessing a crop as and where it is cultivated by farmers is so obvious a necessity as not to need mentioning, but APHIS appears intent on divorcing GE plants from these real-world contexts to the greatest extent possible, perhaps to continue to avoid confronting their many harms. Context is particularly important when the GE trait is agronomic in nature – that is, explicitly designed to change agricultural practice – which is the case for virtually all commercially cultivated GE crops.

¹ Nat’l Fam. Farm Coal. v. Vilsack, No. 21-5695 (N.D. Cal.) (filed July 26, 2021).
To take the most obvious and prevalent example, GE herbicide-resistant (HR) crops profoundly alter weed control practices through deleterious simplification: namely, excessive reliance on post-emergence application of the HR crop-associated herbicide(s) to the exclusion of other methods of weed management. This change in herbicide use patterns has demonstrably led to two major outcomes: an epidemic of glyphosate-resistant weeds (with dicamba-resistance developing rapidly), and the most extensive and severe herbicidal drift damage episodes in the history of U.S. agriculture from dicamba. APHIS cannot simply defer to EPA’s authority over weed-killers, because the HR crop is designed for and invites the herbicide use pattern which has generated these problems. Moreover, APHIS deregulation of HR crops forces the hand of EPA, which is then under tremendous pressure to approve post-emergence use of the companion herbicide(s), even if it/they cannot be used safely.2

Second, APHIS creates artificial constraints by limiting its assessment to the GE plant and its sexually compatible relatives. While plants that harbor or can acquire the GE trait via gene flow should of course be assessed, APHIS’s analysis should also include the universe of other plants that may be impacted by cultivation of the GE crop. For instance, intensive selection pressure from HR crop-associated herbicide use patterns has generated massive herbicide resistance in many weeds that are not sexually compatible with the GE crop. These weeds have emerged due to herbicide selection pressure, but then they have also subsequently spread through gene flow to conspecifics, and by seed dispersal to new regions via wind and water (Liu et al. 2012, Nordby et al. 2007, Sosnoskie et al. 2012, Bennett 2011, Dauer et al. 2009). Although glyphosate-resistant weeds from HR crop cultivation have imposed billions of dollars in costs on U.S. farmers (EPA 2017, USDA 2015), APHIS has done nothing to stem them, despite the ample authority over weeds granted to it under the PPA, and its supposed consideration of “weediness” as one element of its PPRA. Accelerated emergence and spread of herbicide-resistant weeds is a direct and/or indirect consequence of HR crop cultivation.

APHIS’s usual rejoinder here – that herbicide-resistant weeds are not unique to GE crop cultivation – is an irrelevant distraction. HR weeds need not be a risk that is unique to GE crops; it is enough that GE HR crop systems result in vastly accelerated evolution and spread of resistant weeds, and that they have imposed major costs on U.S. agriculture. APHIS is welcome to take action on HR weeds in other contexts, but their emergence in HR crop systems must be its top priority. APHIS’s stance is akin to the National Highway Traffic Safety Administration signing off on a poorly-made new SUV with an unacceptably high risk of rollover on the grounds that currently driven cars with low rollover risk have nevertheless occasionally been known to roll over. Magnitude matters.

APHIS’s concept of the occurrence pattern of the GE plant and sexually compatible relatives should be expanded to include the occurrence pattern of other plants that are affected by cultivation of the GE crop, for instance weeds whose proliferation is fostered by HR crop-associated herbicide use patterns, including but not exclusively herbicide-resistant biotypes. To take one example, Palmer amaranth is a weed native to the desert Southwest and northern Mexico that has undergone extremely rapid spread to the Midwest, South and Eastern United States, where it infests cropland, “especially cotton, corn [and] soybean” (USDA 2017). Among

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the factors that have facilitated its spread is its ability to rapidly evolve herbicide-resistance (Steckel 2007), particularly in conjunction with intensive selection pressure from frequent, every-year application of HR-crop associated herbicides. This has occurred on a massive scale with glyphosate (Pucci 2018), and is starting to occur after just a few years of dicamba application to dicamba-resistant soybeans and cotton, with dicamba- and glyphosate-resistant Palmer amaranth documented in Tennessee (Steckel 2020). Just one year later, this Palmer amaranth biotype has evolved far higher resistance to dicamba, and in many cases exhibits resistance to 2,4-D as well (Steckel and Foster 2021). Palmer amaranth is also evolving resistance to glufosinate from post-emergence application of this herbicide to cotton and soybean varieties genetically engineered for resistance to it, the very first broadleaf weed to have developed resistance to this herbicide anywhere in the world (Barber et al. 2021).

Another scenario where occurrence patterns require consideration is when a GE crop’s trait enables it to expand into environments previously unsuitable for it, thereby displacing other crops. For instance, drought tolerance in maize might permit it to be cultivated under irrigation in more arid regions, and displace non-irrigated crops like wheat or sorghum. Such a shift would entail increased demand on already overtaxed water resources, such as the Ogallala Aquifer in Nebraska, and thus harm to an agriculturally important and increasingly scarce resource. In this case, the expanding occurrence pattern of the GE maize would be a PPA-cognizable effect.

It is also possible that the reduced occurrence pattern of the crop thereby displaced would be an adverse consequence of the GE trait that requires consideration. For example, if the GE maize displaced wheat, this would exacerbate a long-term trend of declining U.S. wheat acreage and production (see graph below). Wheat is a staple crop, and is also a valuable rotation partner that can (and once did to a much greater extent than today) diversify simple rotations based on corn and soybeans, and thereby provide agronomic and environmental benefits such as reduced weed pressure, as well as less pesticide and fertilizer use (Liebman and Dyck 1993, Liebman et al. 2008). While the U.S. has always produced far more wheat than is consumed domestically, recent years have seen a narrowing of the production/consumption ratio for wheat, to a predicted 60-year low of 1.42 in the 2021/2022 crop year, with reduced production/acreage driving the trend.³ The potential for a GE trait to reduce production of a staple crop that also provides substantial agronomic benefits is a PPA-cognizable effect that APHIS should consider in its assessments.

³ See USDA Foreign Agricultural Service at https://apps.fas.usda.gov/psdonline/app/index.html#/app/advQuery. Under Commodity, choose Wheat; under Attributes, choose Production and Domestic Consumption), then choose United States, and All Market Years.
In addition to the four classes of effect that are named on page 7 of the RSR Guidance, APHIS should also consider the effect of the GE plant, as typically grown by farmers, on agriculturally important resources like water, and overall sustainability of US crop production.

**Unintended Phenotypes Must be Assessed**

For the initial review, APHIS proposes to require information on the “intended trait” and “intended phenotype,” implying that its review will assess only the GE plant developer’s intended change. Due to the multifunctionality of genes and their protein and regulatory products, however, a GE plant modified for one intended purpose will often exhibit unintended changes that require assessment in the initial review and PPRA.

Scientists have been slow to appreciate and understand the multifunctionality of genes and proteins (Pritykin et al. 2015), due in part to the tremendous influence of the one gene – one protein – one function dogma (Jeffrey 1999), dating to Watson and Crick. It is also difficult to sort out the roles played by a protein, because those with multiple functions comprise a diverse group that do not share characteristic sequence or structural features, making the discovery of multifunctionality a matter of serendipity (Chen et al. 2021, Su et al. 2019).

However, as more and more proteins are discovered to have several different roles, scientists increasingly see multifunctionality as the norm rather than the exception (Moore 2004), with many proteins presently identified as single-function only because their additional “hidden” functions have yet to be discovered (Zaretsky and Wreschner 2008). Zaretsky and Wreschner
(2008) discuss proteins from 14 different functional groups that display multifunctionality in the broadest sense.

So-called moonlighting proteins represent a subset of multifunctional proteins: those capable of executing two or more distinct biochemical and/or biological functions; but excluding those that result from gene fusion, alternate RNA splicing, or variable post-translational modifications, and excluding also homologous proteins (Jeffery 1999, 2003, 2009; Su et al. 2019). Moore (2004) focuses on plant moonlighting enzymes with multiple roles, and divides them into four categories: 1) Enzymes that catalyze one reaction but have different physiological roles when expressed in different tissues; 2) Promiscuous enzymes, which either recognize multiple substrates or produce multiple products from a given substrate; 3) Enzymes that have two or more sites that catalyze independent reactions; and 4) Enzymes that also have independent non-catalytic role(s), for instance regulatory or structural.

Even within this subclass, the numbers discovered have exploded. Chen et al. (2021) have established the MoonProt Database, which currently catalogues over 500 moonlighting proteins found in animals, plants, other eukaryotes and bacteria. As in every field of molecular biology, far more is known about animals than plants, given the far greater resources directed to research of medical relevance than to plants. This explains why “plant moonlighters are under-represented” among current moonlighting protein databases (Su et al. 2019). Even so, over 100 plant moonlighting proteins have been identified (Ibid.), versus just seven 15 years ago (Moore 2004).

There is no a priori reason to believe that an unintended phenotype ensuing from the genetic modification is any less likely to have plant pest-relevant adverse consequences than the intended phenotype. Therefore, besides investigating the PPA-relevant effects of the GE plant’s intended phenotype, APHIS must also make efforts to discover whether the GE plant has unintended phenotypes, and if so, their potentially adverse and PPA-relevant effects. To this end, APHIS should consult available databases, such as MoonProt, MultitaskProtDB and PlantMP, to determine whether the genetic modification eliminates, silences or otherwise alters a gene/protein product that has one or more functions beyond the one targeted by the GE plant developer to generate the intended phenotype. Because these databases currently capture only a small percentage of multifunctional genes/proteins, especially in “under-represented” plants, APHIS should also consult the scientific literature. Then any unintended phenotypes can be assessed for PPA-relevant impacts.

We briefly discuss one example to illustrate our points.

Silencing of Polyphenol Oxidase (PPO) Genes
There have been a number of crops genetically engineered to suppress the browning of damaged tissue, such as occurs with bruising. One example is the Arctic apple, the intended phenotype of which is a trivial cosmetic one – eliminate browning of the apple’s flesh when sliced. This browning reaction is catalyzed by the enzymes of the polyphenol oxidase (PPO) family, which in the presence of oxygen transform monophenols to diphenols, and diphenols to quinones, which latter bind to amino acids to form dark-colored, lignin-like polymers. PPOs are responsible for most of the browning of damaged fruits and vegetables. In the Arctic apple, this non-browning phenotype is produced via silencing polyphenol oxidase genes in all the plant’s tissues by means of RNA interference (CFS 2013).
While little is known about the natural functions of PPO enzymes in apple trees, there has been considerable research in other plants. High PPO activity levels in leaves is associated with significantly higher yield of red clover, relative to a mutant population with low leaf PPO activity (Boeckx et al. 2017). PPO also appears to be critical for normal nodule development in red clover, as established in experiments in which RNA interference was used to silence PPO in leaves, root and nodules (Webb et al. 2014). PPO activity is associated with reduced nitrogen loss during ensilage of red clover, and better protein quality and nitrogen-use efficiency when the silage is fed to ruminants; as well as increased deposition of beneficial C₁₈ polyunsaturated fatty acids in animal products (Lee 2014). The mechanism seems to involve the o-quinone end product of the browning reaction, which readily binds to many functional groups of proteins, thereby deactivating proteases and lipases in silage (Ibid). Interesting here is the necessity of going beyond the mechanism of action of the enzyme, and assessing the properties and activity of the enzyme product and the consequences of stopping production of enzyme product (o-quinone) by silencing the (PPO) genes that express it. There has also been research demonstrating that PPOs are involved in the biosynthesis of specialized plant metabolites, via both tyrosinase and catechol oxidase activities (Sullivan 2015). The author details biosynthetic roles for PPOs in walnut, snapdragon and creosote bush, and notes that these examples “could represent the tip of the iceberg with respect to PPO enzymes that have specific roles in biosynthesis of specialized metabolites” (Ibid.).

Perhaps most importantly for a PPRA, however, is the well-established role of PPO enzymes in plant defenses against pathogens and herbivores (reviewed in Constabel and Barbehenn 2008, Zhang and Sun 2021; see also CFS 2013). To take just a few examples, tomato PPO is induced by systemin and jasmonate, herbivore defense signals, and there is an inverse correlation between PPO levels in tomato plants and Heliothis zea growth. Bacterial pathogens have also been shown to induce PPO in tomato, and high PPO levels correlate with high pathogen resistance. PPO enzymes are also induced in dormant wild oat seed by seed-decaying Fusarium fungi, and are thought to play a critical role in the seed’s biochemical defenses against decay (Fuerst et al. 2011, 2014). Taranto et al. (2017) review additional examples of the physiological and in particular plant defense roles of PPOs in other plants.

Any PPRA of a GE plant in which PPO enzymes have been silenced must go well beyond a narrow examination of the intended non-browning phenotype in the plant tissue of interest to the crop developer (e.g. apple fruit, potato tuber). APHIS should assess whether elimination of the other roles played by PPO enzymes and/or their products, in various tissues, has any other adverse consequences. If a legume is engineered to silence PPOs, for example, EPA should consider whether nodule development is impacted (Webb et al. 2014); or if a forage is PPO-silenced, protein and lipid quality should be assessed (Lee 2014). The potential for PPO-silencing to disrupt biosynthesis of specialized plant metabolites should be analyzed (Sullivan 2015). The potential for impairment of plant defenses in the PPO-silenced plant has clear plant pest risk implications, particularly the creation or enhancement of a reservoir for one or more plant pests (RSR Guidance, p. 7). PPO silencing presents similar concerns as plants genetically engineered for reduced lignin content (Ibid., p. 7) – they become more vulnerable to attack by insect pests and/or plant pathogens. In assessing such GE plants as enhanced pest reservoirs, APHIS must also consider several likely scenarios: 1) The interrelated impact of increased pesticide use to compensate for increased pest susceptibility; 2) The effect of no pesticide use on the GE plant’s capacity to act as a plant pest reservoir, as might occur if the crop (particularly if
it is a perennial) is abandoned, in consequence of a grower’s management decisions, or for other reasons; and 3) The potential for the GE plant pest reservoir to increase pest and/or disease pressure in other crops, particularly but not only organic crops for which synthetic pesticide use is prohibited.

**Data Needs for Comparator Plant**

Because the PPRA standard is defined in relative rather than absolute terms – as whether the modified plant poses an increased plant pest risk relative to the comparator plant (RSR Guidance, p. 4) – the choice of and knowledge about the comparator plant become extremely important. With respect to all intended and unintended phenotypes, the comparator plant must be understood thoroughly enough to permit a meaningful comparison with the GE counterpart. To use the PPO-silenced Arctic apple as an example, there is more than sufficient evidence of the physiological role of PPO enzymes in plants to form the reasonable scientific hypothesis that the Arctic apple will have increased susceptibility to insect pests and pathogens. To judge whether or not this constitutes a plant pest risk requires knowledge of the plant defense role(s) of the PPO enzyme family in various tissues of the comparator apple tree, as well as pathogen susceptibility testing of the comparator plant(s) and the Arctic apple.

In general, then, if requisite data on either the GE or comparator plant is lacking, then it is impossible to conduct a meaningful evaluation, and the regulatory status review should be halted for lack of data. We emphasize that data on the intended phenotype does not suffice; APHIS must go further and assess unintended phenotypes suggested by a close review of the scientific literature as well as any information the requestor might provide, and demand additional studies as needed if the “best available information” does not satisfactorily answer questions. In addition, APHIS should retain the authority to choose a different comparator plant than the requestor.

**Empirical Data Necessary**

Many questions that need answers in the initial review and PPRA proper will require empirical data. APHIS’s approach here – to base decisions on “the best information available” – is entirely inadequate and indeed irresponsible, if the information that happens to be “available” fails to provide an adequate basis for making a competent plant pest risk determination. APHIS should revise this section of the Guidance to make it clear that no plant pest determination will be made until all needed data, whether then available or not, are collected. Otherwise, APHIS’s PPRA becomes little more than a rubber-stamp exercise.

As discussed above, enough must be known about the GE and comparator plant to assess both the intended as well as unintended phenotypes. To take just two examples. When the modification involves the potential for increased pest or pathogen susceptibility, for example, APHIS should demand controlled tests (e.g. pathogen challenge) to inform its assessment. Pesticidal GE plants should also always be assessed for adverse effects on non-target organisms.

APHIS should not impose unnecessary constraints on itself. For instance, a PPRA should not necessarily be constrained to issues or potential pathways identified in the initial review, nor should either the initial review or PPRA be conducted in the absence of empirical data. APHIS has said that under the old Part 340, field trial observations on a GE plant being assessed for nonregulated status have not influenced its deregulation decisions. This is not surprising, since observations do not provide rigorous evidence. The empirical data we are suggesting here
involves controlled scientific testing whose results can provide actionable information for regulatory decision-making.

**Do Not Exempt Breeding or Molecular Stacks From Regulatory Status Review**

The new Part 340.4 exempts a GE plant from regulatory status review if another GE plant of the same species, with the same trait(s) determined by the same mechanism(s) of action, has already been exempted from Part 340.4. This raises the question of whether, for GE plants with multiple traits, the exemption applies regardless of whether the given trait combination was generated as a molecular or as a breeding “stack.” APHIS has stated that a novel trait combination would be exempted if it was the result of conventional breeding of different GE events that had already been evaluated as not subject to Part 340.4 (breeding stack); but that a novel trait combination generated all at once via genetic engineering (molecular stack) would not be exempted, even if GE plants with the component traits had previously cleared Part 340.4 review.⁴

APHIS’s rationale for this distinction is not persuasive, or even coherent. APHIS believes that breeding stacks involve traits with mechanisms of action (MOA’s) that typically do not interact and so can always be exempted from the RSR, while molecular stacks are not exempted because such interactions may occur (Ibid). However, this distinction is entirely specious. APHIS is loosely associating supposedly “non-interacting” GE plant traits in current GE crops with breeding stacks, on the one hand, and potentially “interacting” (but entirely unspecified) GE plant traits in future GE crops with molecular stacks, on the other. Such fuzzy thinking cannot justify what amounts to a very large RSR exemption category that is not supported by Part 340.4.

An example of a breeding stack that could very well pose plant pest risks, even though it is the product of GE plants that do not, is one involving multiple insect-resistance traits. Imagine a crop that expresses twelve different insecticidal proteins, the result of breeding twelve different GE crop events, each with one insecticidal protein; and assume as well that the insecticidal proteins have similar, overlapping pest spectra (as in the pyramid strategy to stave off resistance), and that the expression levels of each of the twelve proteins is the same in the 12-protein breeding stack as in the parental events. Clearly, the breeding stack has the potential to cause greater and perhaps far greater harm to non-target organisms (including those beneficial to agriculture) than any of the twelve constituent GE events. This could be because there are synergizing interactions between the proteins, or it could be due simply to dose addition – that the combined effect of the 12 insecticidal proteins pose greater risks to non-target organisms than any of the twelve parents does independently. This follows from the risk = hazard x dose principle. And we would note that with respect to resistance management of insecticide-producing GE crops, EPA has promoted a “high-dose” strategy involving the maximum possible mortality of target pests, a strategy that relies in part on maximizing the expression level of the insecticidal protein to kill as high a percentage of pests as possible. The same principle would be expected to apply for non-target organism impacts. This example is by no means far-fetched. SmartStax corn, which expresses six insecticidal proteins, was introduced over a decade ago.

A breeding stack could also pose PPA-cognizable risks in the field that no amount of GE plant, trait or MOA-level scrutiny would reveal. For instance, the most commonly stacked traits in GE crops are herbicide- and insect-resistance. Field research has shown that glyphosate-resistant corn stacked with Bt that expresses Cry3Bb1 targeting corn rootworm has a strong potential to accelerate the evolution of resistance to this insecticidal protein in corn rootworm, exacerbating

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this serious plant pest and its damage to corn (Krupke et al. 2009). Volunteers of this stacked corn in subsequently planted soybean fields provide sustenance to corn rootworm larvae, which are able to feed on their roots whether or not the volunteer plant tests positive for Cry3Bb1. This suggests lower-level Cry3Bb1 production in volunteers, and exposure to sublethal toxin levels accelerates evolution to the toxin in corn rootworm. Such volunteer corn has become more prevalent because it is resistant to glyphosate, the most commonly used soybean herbicide.

This same dynamic becomes still more concerning with newer varieties of GE corn that combine Bt toxin(s) with resistance to multiple herbicides, for instance glyphosate, 2,4-D, aryloxyphenoxy propanoate (AOPP) grass herbicides like quizalofop, and glufosinate; or GE corn with stacked resistance to dicamba, glufosinate, AOPP herbicides, 2,4-D and glyphosate, the latter of which is currently being considered for deregulation by APHIS (CFS 2021). The prevalence of such corn volunteers – and hence their Bt resistance-promoting effects in follow-on soybeans – will likely increase with the number of herbicides to which the corn is resistant, given limitations on the spectrum of weeds confronting farmers and the amount they are willing to spend on herbicidal weed control.

Sequence Information
To better detect and understand the effects of the genetic modification, including unintended phenotypes, APHIS should at a minimum always demand the full sequence of the inserted or modified DNA, and/or the sequence that bridges gene deletions; and for inserts, the sequence as integrated into the crop genome rather than merely the “intended insertion” (RSR Guidance, p. 11). DNA sequencing has become a routine affair, and the cost of sequencing inserts trivial. Indeed, the cost of whole plant genome sequencing has plummeted in recent years. Li and Harkess (2018) report that an Arabidopsis thaliana genome can be sequenced for less than $1,000, with a genome as large as 1 billion base pairs sequence-able for roughly $10,000.

Sequence information can be used to assess frequent mishaps during the genetic engineering process, such as fragmentation of the insert and any resulting fusion proteins (Freese and Schubert 2004); multiple copies of the inserts and/or fragments thereof; insertional mutagenesis that is regularly observed, often far from insertion site(s) (Wilson et al. 2006, Jupe et al. 2019); any off-target effects of gene-editing (Wolt et al. 2016); and interruptions to native plant genes. Sequence information can help inform troubleshooting when the GE plant exhibits unintended phenotypes.

Conclusion
The rapid advances in technologies to manipulate crop genomes for development of food and feed crops call for application of the latest science in their regulation. Instead, APHIS appears to be more interested in inventing excuses for not applying modern science in support of its regulatory mission. This is evident in numerous ways. In its recent proposal to open broad new exemptions from its Part 340 regulations, for instance, APHIS showed far more interest in radiation mutagenesis than in gene-editing, citing four papers on the former subject, one dating back to 1977, with not a single citation to a study of gene-editing or a gene-edited crop. As noted above, APHIS does not even propose requiring the nucleotide sequence of the inserted genetic material, but rather gives the option of submitting the sequence of the intended insertion (RSR Guidance, p. 11).

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Like other aspects of the new GE plant regulations, APHIS’s Regulatory Status Review Guidance is all-too-directed at “regulatory relief,” from a misguided notion of its own activities in this arena as a “burden” on GE crop developers that must be “relieved.” In fact, the history of APHIS GE plant regulation is a history of risks and harms that APHIS ignored or discounted by construing its broad PPA authority far too narrowly. We urge APHIS to regulate GE plants in the true interests of U.S. agriculture and the public, and shed the debilitating conception of its own regulatory program as a rubber stamp wielded to increase public acceptance of GE plants, and thus to be minimized to the greatest extent possible.

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