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THE INHERENT UNCERTAINTY OF RISK ASSESSMENT: HOW PESTICIDE RESIDUE TOLERANCES FALL SHORT ON SAFETY

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I. INTRODUCTION

In the United States, pesticides are important.[1] They are used in astounding quantities and varieties to battle the pests that wreak havoc on our food.[2] Food is cheaper and more bountiful precisely because pesticides are used with such ubiquity.[3] Although pesticides are beneficial for food production, the exact nature of these chemicals makes their use hazardous[4]—pesticides are meant to kill pests.[5] So when the effects of pesticides are felt beyond their intended recipients, substantial harm can result.[6] Fortunately, the United States has a pesticide

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1. *See* Clevo Wilson & Clem Tisdell, *Why Farmers Continue to Use Pesticides Despite Environmental, Health and Sustainability Costs*, 39 ECOLOGICAL ECON. 449, 457 (2001) (explaining how attaining high crop yields in commercial agriculture would be extremely difficult without the use of pesticides).

2. *See* David Pimentel, *Environmental and Economic Costs of the Application of Pesticides Primarily in the United States*, 7 ENV'T DEV. AND SUSTAINABILITY 229, 229 (2005) (noting that the United States uses "approximately 500 million kg of more than 600 different types of pesticides" each year).

3. While this is true in the short term, long-term use of pesticides decreases soil fertility, destroys beneficial pests, and fosters resistance among harmful pests, leading to increased costs of food production. *See* Wilson & Tisdell, *supra* note 1, at 455 (explaining that increasingly larger amounts of pesticides are needed to maintain crop yield levels).

4. *See* Gilles Forget, *Pesticides: Necessary But Dangerous Poisons*, INT'L DEV. CTR. RES. REP., July 1989, at 4, 5 (1989), *available at* http://idl-bnc.idrc.ca/dspace/bitstream/10625/24094/1/108896.pdf (explaining how organophosphates, which is the most commonly used type of pesticide, were actually developed as a chemical weapon during the Second World War).

5. *See* Steven Geoffrey Gieseler, *On a Viable and Effective Future for the Food Quality and Protection Act*, 9 ALB. L. ENVTL. OUTLOOK J. 345, 347 (2004) (explaining the distinctive treatment of pesticides as both inherently toxic, yet undeniably necessary for food production).

6. *See Pesticides and Food: Why Children May be Especially Sensitive to Pesticides*, ENVTL. PROT. AGENCY, http://www.epa.gov/pesticides/food/pest.htm (last updated Sept. 12, 2011) (highlighting

regulatory system that seeks to eliminate this harm.[7] Pesticides cannot be manufactured, sold, or applied to food unless their use meets stringent health-based safety standards.[8] Nevertheless, harm from these chemicals continues.[9] Apart from intentional misuse or imperfect implementation of the law, it is the very design of current pesticide regulation that fails to appropriately assess and measure the risk of continued pesticide use.[10] Current methods of quantitative risk assessment reflect neither the toxicological complexity nor practical realities of human pesticide residue exposure.[11] In this way, the United States' regulatory system fails to achieve "a reasonable certainty that no harm will result from aggregate exposure to pesticide chemical residue" on food.[12]

However, there are several alternatives that consumers and regulators alike can pursue to reduce the overall risk of harm from pesticide residues.[13] One is a plain admission of uncertainty; governmental disclosure and acknowledgement that assurances of safety cannot always be met would increase public awareness of pesticide harm and could, in turn, stimulate the development of pesticide alternatives.[14] This could help grow the already existent—yet small—portion of domestic agriculture committed to organic practices,[15] which has demonstrated an ability to quickly and significantly lower a person's pesticide exposure.[16] These

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the particularly dire health effects children can experience from ingesting pesticide residues); *see also* Michael Eddleston et al., *Pesticide Poisoning in the Developing World–A Minimum Pesticides List*, 360 LANCET 1163, 1163 (2002) (stating that an estimated 798,000 people died from purposeful pesticide self-poisoning in 1990).

7. *See* 7 U.S.C. § 136a(a) (2006) (providing EPA the authority to register all pesticides for sale and use); 21 U.S.C. § 346a(a) (2006) (setting health-based pesticide residue tolerances for food).

8. *See* 21 U.S.C. § 346a(b)(2)(A)(ii) (requiring EPA to ensure "that there is a reasonable certainty that no harm will result from aggregate exposure to pesticide chemical residue" from food and any other potential exposures).

9. *See* Alan R. Boobis et al., *Cumulative Risk Assessment of Pesticide Residues in Food*, 180 Toxicology Letters 137, 138 (2008) (explaining that the most significant health risks from pesticides on food occur from the ingestion of multiple pesticides on a single food item or single pesticides ingested successively on separate food items); *see also* Eddleston et al., *supra* note 6, at 1164 (discussing pesticide harm resulting from occupational and accidental exposure).

10. *See* Michael C.R. Alavanja et al., *Health Effects of Chronic Pesticide Exposure: Cancer and Neurotoxicity*, 25 ANN. REV. PUB. HEALTH 155, 156 (2004) (arguing that if pesticide regulatory policy "were completely effective, the only disease associated with pesticide use would" be associated with accidental or intentional misuse).

11. *See* Emily Monosson, *Chemical Mixtures: Considering the Evolution of Toxicology and Chemical Assessment*, 113 ENVTL. HEALTH PERSP. 383, 383 (2005) (explaining that people are almost always exposed to a multitude of pesticides, rather than individual chemicals in isolation, and that because current risk assessment methodologies generally ignore chemical interactions, they often fail to address the full effects of pesticide exposure).

12. 21 U.S.C. § 346a(b)(2)(A)(ii).

13. *See infra* Part V.

14. *See infra* Part V.A.

15. *See infra* Part V.B.

16. *See* Carl K. Winter & Sarah F. Davis, *Organic Foods*, 71 J. FOOD SCI. R117, R119 tbl.1 (2006), *available at* http://citeseerx.ist.psu.edu/viewdoc/download?doi=10.1.1.128.6360&rep=rep1&type=pdf

steps could help the public simultaneously understand the true risk of harm from pesticides and offer them ways to limit it.[17]

II. THE REGULATORY SYSTEM FOR PESTICIDE FOOD RESIDUES

A. The Problem With Pesticides

Any regulation of chemicals that affects human health must necessarily confront the complex relationship between exposure and harm.[18] Pesticides are no exception, even considering that most of their users specifically desire their resultant harm.[19] Pesticides are designed to kill "pests,"[20] which—statutorily—encompasses all organisms that are perceived to affect human health or welfare.[21] The difficulty in regulating pesticides to achieve safety is that pesticides are chemicals inherently designed to cause harm.[22] Furthermore, the most commonly used pesticides affect biochemical mechanisms that pests and humans share,[23] making harm exceedingly likely for those people exposed to unregulated pesticide chemicals.[24] Indeed, the potential for serious harm in humans due to acute and chronic pesticide exposure is thoroughly documented.[25] The task of eliminating this risk, while simultaneously allowing for the beneficial use of pesticides, falls upon the U.S. Environmental Protection Agency ("EPA") in the form of two

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(summarizing the detection of pesticide residues in conventional and organic produce according to different monitoring programs).

17. *See infra* Part V.

18. *See infra* Part III (outlining how the risk assessment process confronts this complex relationship).

19. *See supra* note 4 and accompanying text.

20. 7 U.S.C. § 136(u) (2006).

21. *See id.* § 136(t) (including all insects, rodents, fungi, weeds, bacteria, and viruses). The EPA Administrator may also elect any other organisms that are "injurious to health or the environment." *Id.* § 136w(c)(1).

22. *See id.* § 136(u)(1) (defining a "pesticide" as "any substance or mixture of substances intended for preventing, destroying, repelling, or mitigating any pest"). In fact, pesticides are prohibited from being sold or used if they fail to effectively eliminate pests. *See id.* § 136j(a)(1)(E) (banning the sale of adulterated or misbranded pesticides).

23. *See* NAT'L CTR. FOR ENVTL. HEALTH, CTR. FOR DISEASE CONTROL AND PREVENTION, GLOSSARY OF CLASSES OF NON-PERSISTENT PESTICIDES 3, *available at* http://www.cdc.gov/nceh/clusters/fallon/Glossary-Non%20Pers.pdf ("Organophosphate pesticides are the most widely used insecticides available today."); *see also* Michael O'Malley, *Clinical Evaluation of Pesticide Exposure and Poisonings*, 349 LANCET 1161, 1161 (1997) (explaining how organophosphate pesticides disrupt the nervous system by inhibiting the enzyme-catalyzed breakdown of the neurotransmitter acetylcholine, leading to disruptions in breathing and normal heart rhythm).

24. *See* O'Malley, *supra* note 23 (explaining that misuse of pesticides or insufficient control measures can lead to pesticide poisonings).

25. In acute poisonings, they have been found to cause everything from headache and dizziness to bronchospasm and coma. Alavanja et al., *supra* note 10, at 175–77. Low-level chronic exposure has been implicated in changes of mood, as well as deficits in neurobehavioral performance. *Id.* at 176–77; *see also* O'Malley, *supra* note 23, at 1161.

monumental responsibilities: pesticide registration[26] and the establishment of pesticide tolerances for food.[27]

B. The Federal Insecticide, Fungicide, and Rodenticide Act: Pesticide Registration

The EPA's primary function regarding pesticides is to serve as a gatekeeper for their legal manufacture and use.[28] Under authority granted to it by the Federal Insecticide, Fungicide, and Rodenticide Act ("FIFRA"), EPA administers a pesticide registration program to determine which pesticides are permitted in the United States,[29] and prohibits any pesticides not approved through this process.[30] The EPA makes these determinations with the help of product information submitted to it before any pesticide can be manufactured for sale or distribution.[31] Accurate product information allows the EPA to evaluate the product's stated claims and intended uses, and the scientific data on which these claims are based.[32] This information is extremely important, as it is later used to calculate the pesticide residue tolerances that are allowed to be present on food.[33]

If the EPA Administrator determines a pesticide's application is complete and accurate, FIFRA compels the Administrator to approve it so long as its intended use will not have "unreasonable adverse effects on the environment."[34] In determining what adverse effects are actually "unreasonable," the Administrator is to take into account "the economic, social, and environmental costs and benefits" of a pesticide's use.[35] By subjecting the registration of pesticide chemicals to a broad standard of cost-effectiveness, this process becomes an important first step to minimizing individuals' pesticide exposure.[36]

C. The Federal Food, Drug, and Cosmetic Act: Setting Tolerances for Food

During the course of its use and application, a registered pesticide is never confined to pests alone, and it will inevitably cross paths with innocent bystanders,

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26. 7 U.S.C. § 136a (2006); *see also infra* Part II.B.

27. 21 U.S.C. § 346a(a)(1)(A) (2006); *see also infra* Part II.C.

28. *See* Gieseler, *supra* note 5, at 350 (discussing EPA's role in registering, approving, and licensing pesticides).

29. *See* 7 U.S.C. § 136a (outlining EPA's pesticide registration program).

30. *Id.* § 136a(a). Pesticides used in emergencies by Federal or State agencies, pursuant to § 136p, and subject to an experimental use permit, pursuant to § 136c, are exempted from the registration process. *Id.*

31. *See* Gieseler, *supra* note 5, at 351; *see also* 7 U.S.C. § 136a(c)(1).

32. *See* Gieseler, *supra* note 5, at 351; *see also* 7 U.S.C. § 136a(c)(1)(A)–(F).

33. Gieseler, *supra* note 5, at 351.

34. *See id.* at 351; *see also* 7 U.S.C. § 136a(c)(5).

35. *See* 7 U.S.C. § 136(bb); *see also* Gieseler, *supra* note 5, at 351.

36. *See* Gieseler, *supra* note 5, at 351 (explaining how, in contrast to other environmental regulatory schemes, the test for approval of pesticide registrations applied by the EPA weighs costs and benefits).

such as children at home,[37] applicators on a farm,[38] or people eating vegetables.[39] When used in agriculture, pesticides can remain present on food throughout its growth, harvest, and consumption.[40] Because of the potential harm exposure to pesticides can cause, it is imperative that exposure routes through food residues are carefully understood and accounted for.[41] This responsibility falls once more upon the EPA, yet under authority granted to it by the Federal Food, Drug, and Cosmetic Act ("FFDCA").[42]

The EPA Administrator must decide which pesticides, and in what amounts, may be permitted for use on food.[43] To fulfill this duty, the Administrator shall set acceptable tolerance levels,[44] to the extent necessary to protect public health, considering whether a given pesticide is necessary for food production and how it may affect consumers through exposure.[45] Tolerances must be set for "safe"[46] pesticide residue levels, which means there is a "reasonable certainty that no harm will result from aggregate exposure to the pesticide chemical residue."[47]

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37. *See, e.g.*, Somia Gurunathan et al., *Accumulation of Chlorpyrifos on Residential Surfaces and Toys Accessible to Children*, 106 ENVTL. HEALTH PERSP. 9, 10 (1998) (examining pesticide exposure routes for children in residential settings).

38. *See, e.g.*, Won Jin Lee et al., *Cancer Incidence Among Pesticide Applicators Exposed to Alachlor in the Agricultural Health Study*, 159 AM. J. EPIDEMIOLOGY 373, 373 (2004) (surveying disease incidence rate of registered restricted use pesticide applicators).

39. *See, e.g.*, Cynthia L. Curl et al., *Organophosphorus Pesticide Exposure of Urban and Suburban Preschool Children With Organic and Conventional Diets*, 111 ENVTL. HEALTH PERSP. 377, 377 (2003) (measuring pesticide exposure in a diet-controlled group experiment).

40. *See* U.S. DEPT. OF AGRIC., PESTICIDE DATA PROGRAM, ANNUAL SUMMARY 3 (2008), *available at* http://www.ams.usda.gov/AMSv1.0/getfile?dDocName=STELPRDC5081750 (explaining that pesticides can be applied both during crop production and after harvest).

41. *See* Natalie C.G. Freeman et al., *Quantitative Analysis of Children's Microactivity Patterns: The Minnesota Children's Pesticide Exposure Study*, 11 J. EXPOSURE ANALYSIS ENVTL. EPIDEMIOLOGY 501, 501 (2001) (explaining that with a greater concern about environmental contaminants comes a concomitant need for a greater understanding of exposure routes).

42. 21 U.S.C. § 346a(b)(1) (2006) (providing the "Administrator" with the authority to set health-based pesticide residue tolerances for food); *see also id.* § 321(hh) (defining "Administrator" to mean "the Administrator of the United States Environmental Protection Agency").

43. *Id.* § 346a(b)(1). The decision to presume that pesticides are harmful, which the process of registration seeks to disprove, is similarly reflected here, where pesticide additives to food are presumed unsafe unless a tolerance is approved by the EPA Administrator. *Id.* § 342(a)(2)(B).

44. Any pesticide residue present on food in excess of its tolerance is deemed "unsafe" under the FFDCA. *Id.* § 346a(a)(1)(A).

45. *Id.* § 346 (2006). In determining how consumers may be affected by exposure, the Administrator must consider such factors as the nature of the toxic effect, dietary consumption patterns, and the cumulative effects of residues. *See id.* § 346a(b)(2)(D).

46. *Id.* § 346a(b)(2)(A)(i).

47. *Id.* § 346a(b)(2)(A)(ii). The Food Quality Protection Act (FQPA) of 1996, which amended the FFDCA's tolerance setting procedure, required that this health-based safety standard be retroactively applied to all tolerances in effect as of August 3, 1996. Pub. L. No. 104–170, § 405(q)(1), 110 Stat. 1489, 1534 (1996) (codified as amended at 21 U.S.C. § 346a(q)(1)). As of 2006, EPA has fulfilled this obligation. *See Accomplishments Under the Food Quality Protection Act*, ENVTL. PROT. AGENCY,

While this strict standard is undoubtedly encouraging, it nevertheless leaves ample ambiguity, considering it needs to be applied with regard to minute quantities of chemicals in attenuated causal chains.[48] Fortunately, however, Congress provides some guidance.[49] The House Report for the Food Quality Protection Act of 1996 indicates that tolerances for harmful pesticides should be at a level where the "aggregate exposure to the pesticide chemical residue will be lower by an ample margin of safety than the level at which the pesticide chemical residue will not cause or contribute to any known or anticipated harm to human health."[50] During the course of application, this has come to require a one hundred-fold safety factor beyond the determined level of no observed adverse effect ("NOAEL").[51] Calculating this level, and therefore where residue tolerances should be set, is accomplished through quantitative risk assessment ("QRA").[52]

III. CALCULATING SAFETY: QUANTITATIVE RISK ASSESSMENT

A. The Process of Assessing Risk

Defined as "the characterization of the potential adverse health effects of human exposures to environmental hazards,"[53] the EPA uses QRA to assess the risk of harm from pesticide exposures and to set an upper level of acceptable risk.[54] This process generally involves four steps: (1) hazard identification, (2) dose-response assessment, (3) exposure pathway assessment, and (4) risk characterization.[55] The first step asks whether a given chemical is putatively

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http://www.epa.gov/pesticides/regulating/laws/fqpa/fqpa_accomplishments.htm (last visited Oct. 28, 2011).

48. For instance, how "reasonable" would factual certainty be if it involved understanding the exact quantities of multiple applied pesticides on a certain crop, the percentage of which remain through growth, harvest, and consumption, and finally how (if at all) those chemicals adversely affect human health? *See generally* Monosson, *supra* note 11, at 383 (explaining how toxicology has historically focused on the effects of just one substance at a time, making it ill-prepared to answer complex questions involving a multitude of active agents).

49. H. R. REP. NO. 104-669, pt. 2, at 29 (1996), *reprinted in* 1996 U.S.C.C.A.N. 1268, 1268 (explaining Congress' purpose in drafting of the Food Quality Protection Act of 1996).

50. *Id.* at 41, *reprinted in* 1996 U.S.C.C.A.N. 1268, 1280.

51. *Id.* ("[T]he Administrator will interpret an ample margin of safety to be a 100-fold safety factor applied to the scientifically determined 'no observable effect' level when data are extrapolated from animal studies.").

52. QRA for pesticide residue exposure is conducted using the paperwork and data submitted by registrants pursuant to 7 U.S.C. § 136a(c). Gieseler, *supra* note 5, at 357–58.

53. NAT'L RESEARCH COUNCIL, RISK ASSESSMENT IN THE FEDERAL GOVERNMENT: MANAGING THE PROCESS 18 (1983).

54. *See Assessing Health Risks from Pesticides*, ENVTL. PROT. AGENCY, *available at* http://www.epa.gov/pesticides/factsheets/riskassess.htm (last visited Oct. 28, 2011) [hereinafter *EPA Assessment Website*].

55. NAT'L RESEARCH COUNCIL, *supra* note 53, at 19–20.

dangerous.[56] In the realm of pesticides—since they are intentionally designed to harm—the answer is almost always "yes."[57]

The second step, a dose-response assessment of the chemical, is conducted with the goal of determining the relationship between the levels of exposure and the severity and frequency of the effects from exposure.[58] The result of this assessment is the NOAEL.[59] The EPA then takes each pesticide's NOAEL and divides it by the aforementioned safety factor, which is usually one hundred, to arrive at the Acceptable Daily Intake ("ADI").[60] This is the maximum "safe" daily residue exposure that a person can safely consume under the FFDCA.[61] However, it is important to keep in mind that because dose-response studies are performed using high doses of chemicals on animals, these assessments typically require "extrapolation from high to low dose and extrapolation from animals to humans."[62] In many cases, converting human toxicity data from animal studies involves broad assumptions and creates wide margins of error.[63]

The third step is the exposure assessment, which requires that the manner in which people may be exposed to the pesticide residue, as well as the intensity and duration of these exposures, shall be measured and identified.[64] During this part of QRA, conclusions are made about the quantity of pesticide residues present on food and the probability those residues will be consumed.[65] Statistical analysis is used to calculate the total residue exposure of a given pesticide based on the registrant's proposed uses, the end result of which is the theoretical maximum residue contribution ("TMRC").[66]

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56. *See EPA Assessment Website*, *supra* note 54.

57. *See supra* notes 4–5. If the answer is "no," then the EPA can exempt the pesticide from the tolerance requirement under 21 U.S.C. § 346a(c)(2)(A)(i).

58. *EPA Assessment Website*, *supra* note 54.

59. *See* Regulation of Pesticides in Food: Addressing the Delaney Paradox Policy Statement, 53 Fed. Reg. 41,104, 41,118 (1988) (illustrating that EPA arrives at the NOAEL by determining how low a dosage must be for a substance–that otherwise causes harm at higher doses–to have no appreciable adverse effects).

60. *Id.* at 41,118. The NOAEL is scaled down by a factor of one hundred because two adjustments, each represented by a factor of ten, need to be made to the dose-response data: one for the fact that humans may be more susceptible to a given chemical than the test animals, and a second to account for the potentially wide variability of chemical susceptibility among humans. Gieseler, *supra* note 5, at 360.

61. *See* Regulation of Pesticides in Food: Addressing the Delaney Paradox Policy Statement, 53 Fed. Reg. 41,104, 41,118 (1988) (explaining how this process is aimed at estimating the level of pesticide exposure that is not believed to cause noticeable harm during the course of a person's life).

62. NAT'L RESEARCH COUNCIL, *supra* note 53, at 19–20.

63. *See infra* Part IV.B.1.

64. NAT'L RESEARCH COUNCIL, *supra* note 53, at 20.

65. Gieseler, *supra* note 5, at 360.

66. *See* Regulation of Pesticides in Food: Addressing the Delaney Paradox Policy Statement, 53 Fed. Reg. 41,104, 41,118 (1988).

The final part of QRA, characterizing the risk, is more or less a summation of the first three steps.[67] In order to characterize the risk, the first three steps are considered together in order to determine the ultimate severity of harm a pesticide's use would cause.[68] This is accomplished in large part by calculating whether the TMRC exceeds the ADI; if estimated exposure (the TMRC) is greater than the acceptable intake of pesticide residue (the ADI), then the use of that pesticide is generally considered to be unsafe.[69]

B. The Inherent Uncertainties

While seemingly reliable and deliberate, QRA is, in fact, based largely on conjecture and guesswork.[70] Indeed, "[a] typical risk assessment consists of about fifty separate assumptions and extrapolations."[71] And it is these myriad assumptions throughout the process of assessing risk that can produce hazard estimations with varyingly protective margins of safety.[72] What is primarily at issue is the great uncertainty of "estimates of the types, probability, and magnitude of health effects associated with a chemical agent."[73] Moreover, toxicological data may be sparse or incomplete.[74] There are still large deficiencies in our knowledge of the causal mechanisms of carcinogenesis and neurotoxicity, which inherently limits our ability to derive conclusive evidence of health hazards associated with specific chemical exposures.[75]

What is disheartening still is that these problems have no discernable solutions.[76] In its comprehensive report on risk assessment procedure in the federal government, the National Research Council ("NRC") identified the "inherent limitations on the power of analysis" as a major limitation on agencies' ability to conduct accurate risk assessment.[77] While the NRC specifically mentioned the "complexity" of the assessment and the "limited analytical resources" agencies

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67. *See EPA Assessment Website*, *supra* note 54 ("[Risk characterization] is the process of combining the hazard, dose-response and exposure assessments to describe the overall risk from a pesticide.").

68. *Id.*

69. *Id.*; *see also* Regulation of Pesticides in Food: Addressing the Delaney Paradox Policy Statement, 53 Fed. Reg. 41,104, 41,118 (1988).

70. *See* Mark Eliot Shere, *The Myth of Meaningful Environmental Risk Assessment*, 19 HARV. ENVTL. L. REV. 409, 413 (1995) ("[The] environmental risk assessment as currently practiced is anything but scientific, objective, and credible.").

71. *Id.*

72. *See* NAT'L RESEARCH COUNCIL, *supra* note 53, at 36 (explaining how many of the components of risk assessment neither have definitive scientific answers nor receive consensuses among scientists).

73. *Id.* at 11.

74. *Id.*

75. *Id.*

76. *Id.*

77. *Id.*

have at their disposal to evaluate such complex chemical risks, it found the greatest inherent limitation on risk assessment to be, simply, "pervasive uncertainty."[78]

The sheer scale of ambiguity in the risk assessment process means scientists and risk assessors within the EPA must make assumptions to fill gaps in scientific knowledge when conducting risk assessments of pesticide residue exposure.[79] For example, while conducting hazard identification and dose-response assessment, a risk assessor must make certain fundamental assumptions about the available toxicological data.[80] These assumptions include that humans and animals will react to pesticide chemicals in similar ways and that low doses in humans will yield predictably small adverse effects from high dose data in animals.[81] New understanding of dose-response curves has fueled targeted criticism to these fundamental risk assessment assumptions.[82] It is perhaps not altogether unsurprising this uncertainty is acknowledged by those intimate with the field, stating that "current methods of conducting . . . health risk assessments . . . generally rely on default assumptions whose validity is unknown."[83]

IV. THE COMPLICATIONS OF ASSESSING RISK FROM PESTICIDE RESIDUES

A. Hazard Identification

After determining whether or not a pesticide residue is putatively dangerous, assessing the type of potential harm is also an important early phase of risk assessment.[84] Residues for which there is no discernable safe level of exposure are classified as "nonthreshold effect" residues.[85] All other residues, for which a safe level of exposure can be determined, are classified as "threshold effect" residues.[86] When the EPA identifies a given pesticide residue as exhibiting threshold toxicity,

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78. *Id.*

79. *See* Celia Campbell-Mohn & John S. Applegate, *Learning from NEPA: Guidelines for Responsible Risk Legislation*, 23 HARV. ENVTL. L. REV. 93, 97 (1999) ("Operating in a world of uncertainty, incomplete data, and genuine differences between scientists in interpretation of and inferences from the available data, risk assessors must make many assumptions and estimates.").

80. *See id.* at 100 ("The basic data needed to perform risk evaluations of chemicals, activities, and sites are severely limited, and the uncertainties in the extant data are profound.").

81. *Id.* at 102.

82. *See* Edward J. Calabrese, *Hormesis: From Marginalization to Mainstream – A Case for Hormesis as the Default Dose-Response Model in Risk Assessment*, 197 TOXICOLOGY AND APPLIED PHARMACOLOGY 125, 125–26 (2004) (arguing that risk assessment assumptions need to be reconsidered because certain chemicals exhibit higher toxic effects at lower doses than higher doses).

83. *See* Monosson, *supra* note 11, at 388 (quoting Linda Teuschler et al., *Support of Science-Based Decisions Concerning the Evaluation of Toxic Mixtures: A New Beginning*, 36 REGULATORY TOXICOLOGY AND PHARMACOLOGY 34, 34 (2002)).

84. *See EPA Assessment Website*, *supra* note 54 (stating that part of the hazard identification is to "identify potential health effects that may occur from different types of pesticide exposure").

85. 21 U.S.C. § 346a(b)(2)(B)(i)(I) (2006).

86. *Id.* § 346a(b)(2)(B)(i)(III).

it then attempts to define a point at which the chemical will not cause any adverse effects.[87]

The inherent scientific difficulty of determining the level at which pesticide residues produce no adverse effects is made more complex by the limited information of biological mechanisms and the dearth of chemical-specific information.[88] This is particularly worrisome because pesticide residues can produce subtle and widely variable health effects.[89] Moreover, some pesticides have been shown to exhibit synergistic[90] effects,[91] calling into question the reliability of many NOAEL determinations.[92] Indeed, the accuracy of the EPA's preferred method of risk assessment—which examines chemicals on an individual basis—is particularly suspect given the fact that people are almost always exposed to pesticides in concert.[93] Even if they are not exposed to multiple residues simultaneously, the majority of people may have lingering presences from past exposures,[94] making both the mixture and the timing of residue exposures important for accurately assessing risk.[95] Furthermore, the effect of a pesticide residue can be difficult to measure.[96] Some pesticides induce immunotoxicity,[97]

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87. *Id.* § 346a(b)(2)(B)(i)(III).

88. *See* William Boyd, *Controlling Toxic Harms: The Struggle Over Dioxin Contamination in the Pulp and Paper Industry*, 21 STAN. ENVTL. L.J. 345, 351--52 (2002) (discussing the lack of basic understanding about the associated health effects of the vast majority of chemicals released into the environment).

89. *See* Alavanja et al., *supra* note 10, at 162–79 (examining the potential carcinogenic and neurotoxic effects of acute and chronic pesticide exposure).

90. EPA defines "synergism" as "[w]hen the effect of the combination is greater than that suggested by the component toxic effects." RISK ASSESSMENT FORUM TECHNICAL PANEL, ENVTL. PROT. AGENCY, SUPPLEMENTARY GUIDANCE FOR CONDUCTING HEALTH RISK ASSESSMENT OF CHEMICAL MIXTURES B-4 (2000), *available at* http://www.epa.gov/ncea/pdfs/chem_mix/chem_mix_08_2001.pdf [hereinafter *EPA Mixture Guidance*].

91. *See, e.g.*, Pamela A. Pape-Lindstrom & Michael J. Lydy, *Synergistic Toxicity of Atrazine and Organophosphate Insecticides Contravenes the Response Addition Mixture Model*, 16 ENVTL. TOXICOLOGY & CHEMISTRY 2415, 2417 (1997) (demonstrating the synergistic effect of atrazine and various other organophosphate insecticides).

92. *See id.* at 2418 (explaining that the study's findings do not conform to traditionally-used models of chemical interaction).

93. Monosson, *supra* note 11, at 383.

94. *See* Daniel Smith, *Worldwide Trends in DDT in Human Breast Milk*, 28 INT'L J. EPIDEMIOLOGY 179, 181–83 (1999) (finding pervasive, detectable concentrations of DDT in human breast milk in the United States twenty-seven years after it was banned).

95. This seems particularly true for exposures of chemicals with hormone-modulating effects, where the extent of chemical B's effect on a person depends upon the timing and amount of that person's exposure to chemical A. *See* Nissanka Rajapakse et al., *Combining Xenoestrogens at Levels Below Individual No-Observed-Effect Concentrations Dramatically Enhances Steroid Hormone Action*, 110 ENVTL. HEALTH PERSPECTIVES 917, 917–18 (2002) (testing the mixture effects of thirteen different sets of common xenoestrogrens).

96. *See* Monosson, *supra* note 11, at 384 (explaining how the few studies that have tried to measure the health effects of chemical mixtures at environmentally relevant concentrations have found contradictory results).

which could potentially make a person more susceptible to illness[98] or more vulnerable to the effects of other pesticides.[99] The causality of immunotoxic effects would certainly go undetected by the EPA's chemical-by-chemical risk assessment procedure.[100]

B. Dose-Response

1. Inherent Problems of Toxicity Measurement

In the current method of assessing risk, determining what relationship exists between the dose of a pesticide residue and its potential effect is extremely important.[101] Typically, studies are conducted that involve administering high doses of pesticides to animals in order to observe the type and severity of any potential adverse effects.[102] The low-level doses at which these adverse effects do not occur are extrapolated from high-dose data and applied to humans.[103] The assumption of this process is the adverse effects of chemical exposure diminish linearly, eventually reaching a point at which low exposures produce no effects.[104] Other research, however, suggests a wide range of chemicals behave antithetically to this assumption.[105] Hormesis, "a dose-response phenomenon characterized by low-dose stimulation and high-dose inhibition," has been frequently encountered and broadly

97. *See* Peter T. Thomas, *Pesticide-induced Immunotoxicity: Are Great Lakes Residents at Risk?*, 103 ENVTL. HEALTH PERSPECTIVES 55, 56–58 (1995) (illustrating the multitude of immunosuppressive and allergic effects of certain pesticide exposures).

98. *See* Elizabeth B. Baldwin, *Reclaiming Our Future: International Efforts to Eliminate the Threat of Persistent Organic Pollutants*, 20 HASTINGS INT'L & COMP. L. REV. 855, 858 & n.14 (1997) (explaining that certain synthetic chemicals have been shown to lead to immune system disruption in humans).

99. *See* Mary O'Brien, *Our Current Toxics Use Framework, Our Stolen Future, and Our Options*, 11 J. ENVTL. L. & LITIG. 331, 340 (1996) (discussing the effect of past pesticide exposure on future pesticide exposure).

100. *See id.* ("Chemical-by-chemical risk assessment does not easily account for contributory roles of multiple chemicals.").

101. *See* Ting-Chao Chou & Paul Talalay, *Quantitative Analysis of Dose-Effect Relationships: The Combined Effects of Multiple Drugs or Enzyme Inhibitors*, 22 ADVANCES IN ENZYME REGULATION 27, 27 (1984) (describing the importance of dose-response relationships in advancing our knowledge of biological systems). The idea that the dose alone determines whether or not a substance is harmful has been a tenet of toxicology since the 16th century. *See* Kirk R. Smith, *Place Makes the Poison: Wesolowski Award Lecture – 1999*, 12 J. EXPOSURE ANALYSIS & ENVTL. EPIDEMIOLOGY 167, 167 (2002) (quoting the oft repeated adage of Renaissance physician Paracelsus that "[s]olely the dose determines that a thing is not poison").

102. *See* NAT'L RESEARCH COUNCIL, *supra* note 53, at 19–20.

103. *Id.* at 19–20.

104. *Id.* at 20.

105. *See* Calabrese, *supra* note 82, at 127–28 (exemplifying the hormetic dose-response phenomenon, whereby low–as opposed to high–doses of a chemical can produce an excitatory).

represented across biological models, endpoints, and chemical agents.[106] Receptor systems that affect a wide range of biological functions—from basic muscle function, to DNA transcription, to inflammatory responses, and hormone communication—have been reported to exhibit hormetic dose-response relationships.[107] Such responses can even occur at exceedingly minute doses.[108] The importance of this phenomenon is underscored by the fact that hormetic dose responses manifested immediately below the toxicological NOAEL,[109] with potentially staggering variability.[110] This finding indicates the QRA safety determinations for pesticides acting on certain receptor systems are simply inaccurate for a large portion of the population, and that the EPA is not meeting the statutorily-mandated standard of safety.[111]

Indeed, growing evidence has suggested that xenoestrogens (chemicals with hormone disruptive properties) cause adverse effects at levels below their established NOAELs.[112] Moreover, the presence of several sub-NOAEL xenoestrogens (which is a probable pesticide exposure scenario) can cause synergistic effects, whereby adverse responses exceed those predicted by the simple addition of each substance's potency.[113] This illustrates a predictive weakness of current methods of assessing risks from chemical mixtures.[114] Such inadequacies may lead to underestimations of potential pesticide harm and an inability to meet the FFDCA's strict standard of safety.[115]

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106. *Id.* at 127–28. Of all implicated endpoints, immune reactions displayed the highest average stimulatory response. *Id.* at 126.

107. *Id.* at 128 (identifying several such receptor systems, such as adrenergic, bradykinin, corticosterone, estrogen and testosterone).

108. *Id.* at 126 (stating that the immune system displayed hormetic stimulation at doses smaller than 1000 times below the toxicological NOAEL).

109. *Id.* at 126. These responses could cause harmful immune alterations, endocrine alterations, and tumor cell proliferation. *Id.* at 132. This is particularly troublesome considering that the hormetic dose-response model was found to represent chemical behavior two and a half times more often than the common threshold model. *Id.*

110. *Id.* at 128 (suggesting that such variability can be accounted for in part by the heterogeneity of the study population, which, in accordance with good scientific practices, mirrors the population at large).

111. *See* 21 U.S.C. § 346a(b)(2)(A)(ii) (2006) (requiring EPA to ensure "that there is a reasonable certainty that no harm will result from aggregate exposure to pesticide chemical residue" from food and all other exposures).

112. Rajapakse et al., *supra* note 95, at 920 (finding dramatic modulation of hormone action from xenoestrogens at levels below pre-determined no-observed-effect).

113. *Id.* at 920.

114. *See id.* at 921 ("[B]y not taking combination effects into account, significant underestimations of the effects associated with exposure to xenoestrogen are likely.").

115. *Id.*

2. The Problem of Chemical Mixtures

While current chemical risk assessment is conducted on an individual basis,[116] the reality is that most people are never exposed to such substances in isolation.[117] In fact, "the great majority of people are exposed to mixtures of both organics and inorganics" at low concentrations.[118] The question of how these chemicals interact is where difficulty arises, given there are nearly an infinite number of combinations.[119] Such an enormous scope disguises which combinations are most important, which dosages should be examined, and which biological systems should be focused on.[120] Of the many chemicals in use today, only a few studies have examined the interactions of even two of these chemicals at a time.[121] Compounding the complexity of mixture assessment is the range of possible biological mechanisms that chemicals can affect.[122] When introduced to the body, toxic pesticides can "act through some combination of altering gene expression, changing levels of intracellular concentrations of ions, alternating cellular metabolism or production of cellular regulators," all on a variety of biological targets.[123] Moreover, when the target organ of a pesticide regulates other organs or cells (i.e. the thyroid or pancreas), the overall impact of the chemical can be large.[124]

In recognition of this astounding complexity, the EPA developed guidance documents for assessing the human health impacts of chemical mixtures.[125] Therein, it compiled information on several different approaches to chemical mixture risk assessment.[126] One approach involves complex mixtures, common industrial and commercial chemical combinations, for which mixture-specific data actually exists.[127] However, even when such data exists, the mixture itself often

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116. *See* Raymond S. H. Yang, *Introduction to the Toxicology of Chemical Mixtures*, *in* TOXICOLOGY OF CHEMICAL MIXTURES: CASE STUDIES, MECHANISMS AND NOVEL APPROACHES 1 (Raymond S. H. Yang ed., 1994) (finding that, in a review of papers on chemical mixtures published in 1992, 95% of the papers were devoted to single-chemical studies).

117. David O. Carpenter et al., *Understanding the Human Health Effects of Chemical Mixtures*, 110 ENVTL. HEALTH PERSPECTIVES (SUPP. 1) 25 (2002).

118. *Id.*

119. *Id.*

120. *Id.*

121. *Id.* It is estimated that there are upwards of 80,000 chemicals in use today. The National Toxicology Program has published only 605 reports on the long and short-term effects of these. *Id.*

122. *Id.*

123. *Id.*

124. *Id.* Xenoestrogen chemicals that affect hormone function likely target these endocrine organs first. *Id.* at 30-32.

125. *See EPA Mixture Guidance*, *supra* note 90, at 2.

126. *Id.* at 6–9.

127. *See id.* at 1. This method is ideal for diesel fuel and some polychlorinated biphenyl (PCB) mixtures. *See* Monosson, *supra* note 11, at 386.

changes or degrades once introduced into the environment.[128] Any secondary metabolites from the original mixture will not be reflected in the commercial data.[129] In any case, mixture-specific data exists for so few chemical combinations that this assessment approach is seldom used.[130]

Without adequate data on the chemical interactions of mixtures, the EPA may apply a "components" approach, which adds the data for each individual chemical and analyzes it to estimate the resultant interactions.[131] The rationale for such an approach rests on the premise that chemical interactions either do not occur or are toxicologically unimportant, given the miniscule concentrations at which they are typically present.[132] As a matter of fact, the EPA's default presumption is that chemical interactions do not occur.[133] However, this is often not the case, so when chemical interactions do take place, one of two risk assessment methods are used: dose addition or response addition.[134]

If multiple chemicals act on the same biochemical pathway and affect similar targets, then a dose addition approach is usually appropriate.[135] This method "assumes that the potency of each chemical in the mixture can be calculated relative to each other or to one common chemical" and seeks to determine the contribution of each chemical in the mixture to its overall toxicity.[136] In this way, dose addition effectively standardizes similar chemicals, so that they can be treated alike and added together, with the resultant figure representing the total toxicity of the mixture.[137] Because pesticide residue tolerances are calculated with a wide safety factor,[138] the cumulative toxicity of biochemically similar pesticides is usually well within the margin of safety.[139] However, as mentioned above, two very notable exceptions to this trend are xenoestrogens[140] and chemicals with hormetic

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128. Monosson, *supra* note 11, at 386. This process is called "weathering" and results in unpredictability even where ample data exists. *Id.*

129. *Id.* (explaining that the toxicological data for complex mixtures does not accurately describe those mixtures after they are introduced to the environment).

130. *Id.* (explaining that because toxicity data for chemical mixtures is rarely available, risk assessors are forced to apply a "components approach").

131. *Id.*

132. *Id.*

133. *Id.* However, this approach is problematic given the lack of data available for chemical interactions at low concentrations. *Id.* EPA is unlikely to detect chemical interactions if the default presumption is that they don't exist. *Id.*

134. *Id.* at 386–87 (explaining the dose addition and response addition assessment methods).

135. *Id.* at 386.

136. *Id.* at 387.

137. *Id.*

138. *See supra* notes 50–51 and accompanying text.

139. For instance, the simultaneous consumption of ten pesticide residues at their maximum allowable tolerance (without any interaction) would still be below the NOAEL by a factor of ten. *See supra* note 60 and accompanying text.

140. *See supra* notes 112–113 and accompanying text.

dose-response curves,[141] each of which has the potential to exceed their established NOAELs.[142]

A different approach to assessing chemical mixtures is the response addition method, which the EPA uses when multiple chemicals have entirely separate modes of action and whose toxicity is not interactive.[143] The EPA uses this method precisely because each chemical acts on a different biological endpoint.[144] Thus, in order to evaluate the cumulative toxicity of the mixture, the EPA suggests simply measuring the percentage of laboratory animals responding adversely to each chemical and then combining those portions to arrive at a cumulative risk estimate.[145] This method in no way resembles an exact science, partly because it is assumed—as with dose addition—that no chemical interactions occur.[146] The EPA itself proclaims that "additivity assumptions are expected to yield generally neutral risk estimates."[147] However there is evidence to suggest that this is not always true; interactions between various dilute environmental estrogens have been shown to create adverse effects greater than the sum of their combined concentration.[148] Similarly, synergistic chemical interactions have been observed in the EPA's own MIXTOX database.[149] Intended to serve as a database for the interactive effects of chemicals, an analysis of MIXTOX revealed that about twenty-five percent of the chemical combinations demonstrated consistent synergism.[150] This figure is likely an underestimation of synergistic chemical relationships since it did not include chemicals that exhibited intermittent synergism—instances where synergistic behavior was observed but not reliably occurring.[151] Rather than diluting the persuasive weight of chemical synergism, these instances of intermittent synergism may indicate there were differences in specific timing, sequence, and endpoints involved in the synergistic chemical interactions.[152] It becomes clear, in any case,

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141. *See supra* notes 106–109.

142. *See* Monosson, *supra* note 11, at 388 (noting the importance of increasing scientific understanding regarding chemical interactions that fall below the NOAEL).

143. *See EPA Mixture Guidance*, *supra* note 90, at 11–12.

144. Monosson, *supra* note 11, at 387.

145. *See EPA Mixture Guidance*, *supra* note 90, at 29.

146. *See* Monosson, *supra* note 11, at 387 ("Like dose addition, response addition is a 'no-interaction' approach.").

147. RISK ASSESSMENT FORUM, ENVTL. PROT. AGENCY, GUIDELINES FOR THE HEALTH RISK ASSESSMENT OF CHEMICAL MIXTURES 15 (1986), *available at*: http://www.epa.gov/raf/publications/pdfs/CHEMMIX_1986.PDF.

148. *See* Rajapakse et al., *supra* note 95, at 920 (finding dramatic modulation of hormone action from xenoestrogens at levels below pre-determined no-observed-effect).

149. *See* Monosson, *supra* note 11, at 387 (citing Richard C. Hertzberg & Margaret M. MacDonell, *Synergy and Other Ineffective Mixture Risk Definitions*, 288 SCI. OF THE TOTAL ENV'T 31, 32–33 (2002)).

150. *Id.*

151. *Id.* The intermittent occurrence of synergistic interactions in the MIXTOX database studies is most likely resultant from the variability of study designs for a given chemical interaction. *Id.*

152. *Id.*

the assumptions made by the EPA's cumulative risk assessment procedure may systematically underestimate the risk of harm from chemical interactions.[153]

C. Exposure Assessment

Exposure assessment is "one of the most difficult problems facing environmental health scientists."[154] Risk assessors are forced to make many assumptions in order to estimate potential risk because of the attenuated causality between the application of pesticides on food and its ultimate consumption.[155] For instance, an exposure assessment of pesticide food residues requires an assessor to make assumptions about the percentage of crops treated, the amount of pesticide residues on food when purchased by consumers, the quantity of the product consumed, and how readily such a residue is actually absorbed by the consumer.[156] The variables that are a part of the exposure pathways of pesticide residues force many estimations to fill their gaps.[157] The parity of any given assumption used in exposure assessments does not lend confidence to their conclusions, especially when "almost every single number in this area may be modified, with relative impunity, by the risk assessor."[158] In order to improve the methods of exposure assessment, the U.S. Government Accountability Office ("GAO") urged the EPA to use and examine more biomonitoring data so that its risk assessments can be verified by actual population exposure to chemicals.[159] However, the EPA can hardly be faulted, since this data exists for only 148 of the 6,000 chemicals used and deposited in the environment.[160] Such limited availability prevents risk assessors from using an otherwise ideal source of information for determining exposure.[161] Yet even biomonitoring data cannot always uncover a person's total chemical exposure.[162] While it is true such data often provides reliable estimates about a person's exposure, many chemicals are either quickly metabolized or just

153. *See supra* notes 148–152 and accompanying text.

154. *See* Carpenter et al., *supra* note 117, at 26.

155. *Id.*

156. *See* Junius C. McElveen, Jr. & Chris Amantea, *Legislating Risk Assessment*, 63 U. CIN. L. REV. 1553, 1588 (1995).

157. *See* NAT'L RESEARCH COUNCIL, *supra* note 53, at 27 ("For new chemicals with no measurement data at all, rough estimates of exposure are necessary.").

158. McElveen & Amantea, *supra* note 156, at 1586.

159. U.S. GOV'T ACCOUNTABILITY OFFICE, BIOMONITORING: EPA NEEDS TO COORDINATE ITS RESEARCH STRATEGY AND CLARIFY ITS AUTHORITY TO OBTAIN BIOMONITORING DATA 21–22 (2009), *available at* http://www.gao.gov/new.items/d09353.pdf.

160. *Id.* at 3.

161. *See id.* at 2 ("[B]iomonitoring measurements are the most health-relevant assessments of exposure because they measure the amount of the chemical that actually get into people from all environmental sources . . . combined.").

162. *See* Carpenter et al., *supra* note 117, at 26 (explaining how even concrete biomonitoring data isn't entirely reliable because in most cases it doesn't reflect a substance's excretion and/or metabolism).

fundamentally not persistent.[163] This leads both to the under estimation of chemical exposure from biomonitoring data and the degradation of its reliability.[164] Consequently, a risk assessor is left to deal with incomplete data and an excess of uncertainty.[165]

V. RESPONDING TO THE UNCERTAINTIES OF PESTICIDE RESIDUE RISK ASSESSMENT

A. Admitting and Disclosing Uncertainty

Because the science of environmental toxicology is relatively new, there is an inherent limit to the accuracy and wisdom of the current chemical regulatory system.[166] There is much uncertainty in the causal chain linking agricultural pesticide application and the consumption of pesticide residues.[167] When people are exposed, it is still unclear how pesticides affect human biology,[168] what interactions the pesticides might produce,[169] and the doses at which pesticides cause harm.[170] Since much is still unknown about pesticides, it is important that regulators and the public alike acknowledge our current understanding of pesticide toxicity prevents us from providing a reasonable assurance of safety.[171] "Labeling ignorance as ignorance, rather than safety, is an important first step."[172]

This approach is not without precedent.[173] With the passage of the Emergency Planning and Community Right-to-Know Act ("EPCRA")[174] of 1986, Congress required industrial facilities to report and compile any substantial environmental releases of specified chemicals.[175] In the years following EPCRA's enactment, that resultant compilation, the Toxics Release Inventory ("TRI"), is credited with

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163. *Id.* While not fully detectable, many toxic substances nevertheless continue to cause harm. *Id.*

164. *Id.* (explaining how many toxic chemicals are either fundamentally not persistent, yet exert long lasting harmful effects, or are partially metabolized and/or excreted).

165. *Id.* ("[A]ll too often the environmental health scientist is left with only interview reports of exposure, occupational history, or other less rigorous evidence from which to draw conclusions about relationship to disease.").

166. *See generally* ENVT'L HEALTH PROGRAM, ENVT'L. DEF. FUND, TOXIC IGNORANCE: THE CONTINUING ABSENCE OF BASIC HEALTH TESTING FOR TOP-SELLING CHEMICALS IN THE UNITED STATES 45–47 (1997), *available at* http://www.edf.org/sites/default/files/243_toxicignorance_0.pdf (arguing for several changes to the legal status for chemicals where safety screening is unreliable).

167. *See supra* Part IV.C.

168. *See supra* Part IV.A.

169. *See supra* Part IV.B.2.

170. *See supra* Part IV.B.1.

171. *See* ENVT'L HEALTH PROGRAM, *supra* note 166, at 7 (stating that toxicity testing hasn't been performed for nearly seventy-five percent of the most popular chemicals in commercial use, and as such, there can be no definitive determination on the safety of their use).

172. *See id.* at 42.

173. *See id.* at 42 (explaining how a similar approach was pioneered with the creation of the Toxics Release Inventory).

174. 42 U.S.C. §§ 11001-11050 (2006).

175. *Id.* § 11023.

reducing chemical releases from covered facilities by forty-four percent.[176] Importantly, EPCRA contained no mandatory reduction criteria,[177] and so the success of this procedure is wholly attributable to the power of public disclosure.[178] Likewise, if the true uncertainty of pesticide harm were publicly disclosed, then manufacturers would invariably try to find alternatives to conventional pesticides and avoid the condemnation of discerning consumers.[179]

B. Encouraging Organic Agriculture

It is certainly acknowledged that our cheap and fecund food production system depends in large part on conventional pesticides.[180] Yet, as the environmental and health costs associated with their use becomes more transparent, the inability to sustain this current system is hard to ignore.[181] Ever increasing food demands, as well as environmental, economic, and social goals,[182] demand innovative policies and new farming approaches. Organic agriculture can serve as an important first step by immediately reducing pesticide residue exposures[183] and creating opportunities for the production of conventional pesticide alternatives.[184]

Though the farm sector remains diverse, there has been a steady and consistent trend toward crop specialization and economizing scale, leading to a vast

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176. Addition of Reporting Elements; Toxic Chemical Release Reporting; Community Right-to-Know, 61 Fed. Reg. 51,322, 51,322 (Oct. 1, 1996).

177. *See* 42 U.S.C. § 11023(a) (requiring only that toxic chemical production information be made public).

178. *See* ENVT'L HEALTH PROGRAM, *supra* note 166, at 36.

179. *See id.* at 36 (explaining that, between 1988 and 1994, despite any mandatory reductions, chemical manufacturing facilities whose releases were disclosed in the TRI reduced such emissions by 1.6 billion pounds).

180. *See* David Pimentel et al., *Benefits and Costs of Pesticide Use in U.S. Food Production*, 28 BIOSCIENCE 772, 772 (1978) (relaying the proposition that pesticide use contributes significantly to the production of cheap and bountiful food in the U.S.).

181. *See, e.g.*, Wilson & Tisdell, *supra* note 1, at 450–51 (explaining that while pesticide use is initially beneficial, it destroys natural predators and creates insect resistance, leading to the economic futility of pesticide).

182. *See* DIV. ON EARTH AND LIFE STUDIES, NAT'L RESEARCH COUNCIL, TOWARD SUSTAINABLE AGRICULTURAL SYSTEMS IN THE 21ST CENTURY 54 (2010) ("As productivity in agriculture continues to increase, the natural resources used to support agriculture are being depleted . . . Such economic concerns as farm sector profitability and rising input costs and such social concerns as labor justice, food quality and safety, animal welfare, and community well-being are also becoming more prominent.").

183. *See id.* at 223, 225–26 (detailing the principles of organic farming and what strategies are used to tackle typical agricultural problems without the use of synthetic pesticides).

184. *See, e.g.*, *id* at 137–39 (explaining how certain farm practices–such as tillage, crop rotation, and the applications of fertilizers and pesticides–can be modified in ways that either create unintended adverse consequences on crop yield and pesticide reliability or foster an environment that is supportive of beneficial arthropods and soil-borne pathogens).

increase in the production amount of the largest farms in the country.[185] The Organic Foods Production Act ("OFPA") of 1990[186] responded to the increased demand for organic foods and helped to commercially revitalize organic agriculture by giving it a standardized structure.[187] By creating uniform national standards[188] and strict compliance procedures for certification,[189] the OFPA has created an organic foods production system on which both producers and consumers can rely.[190] This system is essential for increasing the viability of organic food producers, as well as the corollary benefits that come with organic agriculture.[191]

One of the fundamental requirements for obtaining organic certification is that a producer abstains from using synthetic pesticides.[192] For consumers, this qualification is increasingly desirable; not only do consumers seek out organic foods in greater and greater numbers,[193] but they do so because of its perceived deficit in pesticide residues.[194] Largely to the credit of regulators, consumers' perception about organic food is entirely accurate.[195] In one of the few studies looking at the relationship between pesticide residues in conventional and organic foods, significantly lower residue amounts were found in organic produce than on

185. *See id.* at 48–51 (explaining that in 2002, nearly half of total U.S. farm sales came from so-called "million-dollar" farms–those with annual sales in excess $1 million–yet such farms represented only two percent of all U.S. farms).

186. Organic Foods Production Act of 1990, Pub. L. No. 101-624, 104 Stat. 3935 (codified as amended at 7 U.S.C. §§ 6501-6523 (2006)).

187. *Id.* *See* CAROLYN DIMITRI & CATHERINE GREENE, ECONOMIC RESEARCH SERVICE, U.S. DEPT. OF AGRICULTURE, RECENT GROWTH PATTERNS IN THE U.S. ORGANIC FOODS MARKET 1 (2000), *available at* http://www.ers.usda.gov/publications/aib777/aib777c.pdf ("Since the early 1990s, certified organic acreage has increased as producers strive to meet increasing demand for organic agricultural and food products in the United States.").

188. *See* 7 U.S.C. § 6504(1) (requiring that organically produced agricultural products must be produced without synthetic chemicals).

189. *See id.* § 6505 (providing that a person may only sell or label an agricultural product as organically produced if it is produced and handled in accordance with the statute).

190. *See* Winter & Davis, *supra* note 16, at R117 (delineating the three main goals of the OFPA).

191. *See* David Pimentel et al., *Environmental, Energetic, and Economic Comparisons of Organic and Conventional Farming Systems*, 55 BIOSCIENCE 573, 573 (2005) (stating that the third-party verified practices managed by the USDA's National Organic program give consumers assurance on how organic foods are produced).

192. *See* 7 U.S.C. § 6504(1) (providing that products can only be labeled as organically produced if they "have been produced and handled without the use of synthetic chemicals, except as otherwise" allowed by the statute).

193. *See* Winter & Davis, *supra* note 16, at R117 (stating that organic food sales have increased nearly twenty percent annually since 1990).

194. *See Nearly Two-Thirds of Americans Have Tried Organic Foods and Beverage*, WHOLE FOODS MARKET NEWSROOM (Nov. 18, 2005), *available at* http://wholefoodsmarket.com/pressroom/blog/2005/11/18/nearly-two-thirds-of-americans-have-tried-organic-foods-and-beverages/ (finding that of those consumers purchasing organic products, seventy percent cited a desire to avoid pesticides as their primary reason).

195. *See* Winter & Davis, *supra* note 16, at R119 (illustrating the significantly reduced quantities of pesticide residues on organically-grown food).

conventional produce.[196] The data from the U.S. Department of Agriculture's ("USDA") Pesticide Data Program ("PDP"), for example, found that seventy-three percent of conventional foods contained one or more pesticide residues, whereas organic foods contained just twenty-three percent.[197] The numbers of both groups were likely inflated due to the detectable presence of environmentally-persistent banned pesticides (such as DDT).[198] By omitting these pesticide residues from the analysis, conventional levels dropped to seventy-one percent and organic levels dropped to thirteen percent.[199] This study helped to illustrate an important reality—even organic foods are not free from pesticides. Whether it is from persistent organic pollutants, environmental drift, or fraudulent mislabeling, organic certification can only reduce pesticide residues on food, not eliminate them.[200]

Nevertheless, this reduction is real and it is essential for minimizing the potential health risks associated with pesticide exposure.[201] For example, several scientific studies examining the negative health effects that pesticides cause in children have nearly all reflected the trends evident in the USDA's data.[202] Both short and long-term studies have found statistically significant relationships between a child's organic diet and the reduction of their exposure to pesticides.[203] Moreover, the diets of these groups demonstrate the primary source of pesticide exposure for children is through their diet, which can be significantly reduced by eating organically produced food.[204] While it is important to note that the residue reductions one would achieve by eating organic food has not yet been found to result in measureable health benefits,[205] the pesticide exposure reductions are

196. Brian P. Baker et al., *Pesticide Residues In Conventional, IPM-Grown and Organic Foods: Insights from Three Data Sets*, 19 FOOD ADDITIVES AND CONTAMINANTS 427, 428–29 (2002). This finding was consistent across multiple data sets with markedly different sensitivity, analytical, and sample collection techniques. *Id.* at 427–28.

197. *Id.* at 428.

198. *Id. See* Winter & Davis, *supra* note 16, at R117 (noting that in one study "[s]ome of the residues encountered in all of the sample pools represented environmentally persistent chlorinated hydrocarbon insecticides that have been banned for use for several decades but are still present in small amounts in many agricultural fields and can result in food residues").

199. Baker et al., *supra* note 196, at 429.

200. *See id.* at 430 (explaining why "organic foods are not pesticide free").

201. *See* Winter & Davis, *supra* note 16, at R117 (illustrating the significant difference in pesticide levels between conventional and organic produce).

202. *See, e.g.*, Curl et al., *supra* note 39, at 381 (discussing how an organic diet results in a significant reduction in children's pesticide exposure).

203. *See, e.g.*, Curl et al., *supra* note 39, at 377 (looking at short term organophosphate exposures in children); Chensheng Lu et al., *Dietary Intake and Its Contribution to Longitudinal Organophosphorus Pesticide Exposure in Urban/Suburban Children*, 116 ENVTL. HEALTH PERSPECTIVES 537, 537 (2008) (examining the longitudinal effects of organophosphorus pesticide exposure in children).

204. *See* Lu et al., *supra* note 203, at 541.

205. *See* Winter & Davis, *supra* note 16, at R121.

real.[206] It is likely the same methodological difficulties which arise during the assessment of pesticide risks[207] present themselves once more during attempts to determine the safety of alternatives.[208] In this way, the uncertainty of the risk assessment process is precisely why concrete reductions in one's pesticide exposure are, most likely, the best way to minimize one's risk of harm.

VI. CONCLUSION

Because of the inherent toxicity of pesticide chemicals—and their current ubiquity in U.S. agriculture—Congress has long found the need to protect people from the health and safety hazards resulting from their use.[209] Under the current regulatory scheme the EPA has authority to administer and enforce pre-manufacture registration requirements for pesticides and set allowable residue tolerances for those permitted for sale and use.[210] Both of these tasks are undertaken with the primary purpose of ensuring safety,[211] yet the process used to accomplish this quantitative risk assessment contains inherent uncertainties that prevent the statutorily mandated safety standard[212] from being fulfilled.[213] Most of these shortcomings are attributable to the developing nature of environmental toxicology and the limited scope of past chemical research,[214] but the fact remains that current methods of assessing risk are ill suited for the task of examining pesticide residues.[215] Current dose-response assessments simply do not consider potential hormetic properties of or the complex interactions between pesticide chemicals.[216] Similarly, exposure route assessments do not have nearly enough data to make accurate evaluations of the ways in which people are exposed to pesticides.[217]

Nevertheless, there are ways in which the negative impact of the uncertainty of this process can be minimized.[218] Admitting and disclosing the true variability of pesticide exposure—instead of maintaining that our regulatory structure actually

206. *See id.* at R121 ("Organic fruits and vegetables posses fewer pesticide residues and lower nitrate levels than do conventional fruits and vegetables.").

207. *See supra* Part IV.

208. This seems likely given the need to ensure that the FFDCA's safety standard is maintained for all pesticides in use, no matter their initially perceived innocuousness. *See supra* Part IV.

209. *See supra* Parts II.B-C.

210. *See supra* notes 27–28 and accompanying text.

211. *See supra* notes 21–27 and accompanying text.

212. *See* 21 U.S.C. § 346a(b)(2)(A)(ii) (defining "safe" as it relates to pesticide residue as "that there is a reasonable certainty that no harm will result from aggregate exposure to the pesticide chemical residue"); *see also supra* Part III.B.

213. *See supra* Part III.A.

214. *See supra* notes 75–78 and accompanying text.

215. *See supra* Part III.B.

216. *See supra* Parts IV.B.1–B.2.

217. *See supra* Part IV.C.

218. *See supra* Part V.

results in a "reasonable certainty that no harm will result from aggregate exposure to the pesticide chemical residue"[219]—would be an important first step for empowering consumers and motivating producers to find pesticide alternatives. Similarly, the continued proliferation of organic agriculture will help redirect our current food production system toward a more sustainable future and significantly reduce our collective pesticide exposure.[220] While current methods of risk assessment are undoubtedly based on immature science and imperfect regulation, it is nevertheless encouraging to know there are immediate ways to reduce one's risk of harm.[221]

219. 21 U.S.C. § 346a(b)(2)(ii) (2006).

220. *See supra* Part V.B.

221. *See* Chensheng Lu et al., *Organic Diets Significantly Lower Children's Dietary Exposure to Organophosphorus Pesticides*, 114 ENVTL. HEALTH PERSPECTIVES 260, 262 (2006) (stating that the protection against pesticide exposure that organic dietary choices provides is both "dramatic and immediate").