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The Consequences of Consensus: Dangerous Compromises of the Food Quality Protection Act

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THE CONSEQUENCES OF CONSENSUS: DANGEROUS COMPROMISES OF THE FOOD QUALITY PROTECTION ACT

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In August 1996, President Clinton signed the Food Quality Protection Act ("FQPA"),[1] amending the Federal Insecticide, Fungicide, and Rodenticide Act ("FIFRA")[2] and the Federal Food, Drug, and Cosmetic Act ("FFDCA").[3] Amendments to the FFDCA removed pesticide residues on food from the reach of the infamous Delaney Clause.[4] In so doing, Congress enacted "one of the most significant environmental and public health bills passed in 20 years, [which] indeed may distinguish itself in time as the most significant."[5] The new FQPA contains many detailed provisions of potential import. Perhaps the most striking and significant provision is the creation of a new decision standard for regulation of risks to human health.[6] This article focuses on the new standard and its anticipated effects, with particular attention to carcinogenic substances.[7]

The FQPA is the result of a compromise between two groups. One group, the food industry, had long complained about the unreasonableness of the existing Delaney Clause standard for pesticide residues in processed foods.[8] In response, various senators and representatives made periodic attempts to modify the standard.[9] The second group, environmental interests, publicly

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1. Food Quality Protection Act of 1996 ("FQPA"), Pub. L. No. 104-170, 110 Stat. 1489 (1996).

2. Federal Insecticide, Fungicide and Rodenticide Act ("FIFRA"), 7 U.S.C. §§ 136-136y (1994).

3. 21 U.S.C. §§ 301-395 (1994).

4. 21 U.S.C. § 348(c)(3)(A) (1994). For a discussion of the Delaney Clause, see *infra* Part I.

5. Letter from Charles Benbrook, former director of the National Academy of Sciences Agricultural Board, to California State Senator Mike Thompson (July 31, 1996) [hereinafter Benbrook letter].

6. Environmental Protection Agency ("EPA") has declared that this uniform decision standard is the "most important aspect" of the statute. Notice to Manufacturers, Formulators, Producers, and Registrants of Pesticide Products, Pesticide Registration (PR) Notice No. 97-1 (January 31, 1997).

7. The FQPA was passed largely to deal with problems of the Delaney Clause regulation of carcinogenic pesticides. *See infra* Part II.A. Carcinogens present a particularly vexing regulatory problem because they are presumed to have no safe threshold of exposure. Approximately 20% of registered pesticides are suspected carcinogens. *See* Sandra O. Archibald & Carl K. Winter, *Pesticides in Our Food: Assessing the Risks*, *in* CHEMICALS IN THE HUMAN FOOD CHAIN 1, 12 (Carl K. Winter et al. eds., 1990).

8. *See* Margaret Kriz, *A Peace Treaty Over the Delaney Clause*, NAT'L J., Aug. 3, 1996, at 1642.

9. *Id.* (observing that "[f]arm-state members from both parties have long been keen to overturn the 1958 Delaney Clause and its sweeping prohibition against food additives that contain even a trace

adhered to the overly-protective Delaney Clause, but many of these groups also appreciated the shortcomings of its standard.[10] Accordingly, these groups indicated their willingness to abandon the Delaney Clause if they received a substitute standard that they deemed sufficiently protective.[11] As a compromise, the agricultural interests evaded the application of the Delaney Clause to pesticide residues, and the environmental groups obtained a highly protective new standard.[12]

Achieving such a compromise is generally regarded as a success, evidence of the effectiveness of the democratic process, evincing the virtues of moderation.[13] However, the FQPA compromise was not universally applauded. Some of the more adamant environmental groups were disappointed to lose the application of the Delaney Clause.[14] Conversely, an analyst for the conservative Competitive Enterprise Institute complained that the new law was unreasonably tough and would preclude the application of numerous pesticides.[15] Many believe these criticisms evidence the virtues of the FQPA, reasoning that opposition from both ideological extremes signals the quality of the legislation.

In this article, I take issue with such conventional wisdom that the FQPA represents a significant policy advance. While compromise obviously has some merit in disputes between competing public values, it is a poor policy for fundamentally science-based matters. One does not try to establish the origins of humanity by crafting some compromise between creationism and evolution. Likewise, if the goal is to maximize protection of public health, compromise between interest groups is unlikely to yield the best policy.

The central thesis of this article is that pesticide residues in food have been and will continue to be overregulated. The FQPA may well exacerbate the overregulation. While industry commonly laments compliance costs, the true tragedy of such overregulation is found in its effects on public health. As detailed below, patterns of pesticide regulation that persist in the FQPA will likely be hazardous. Rather than compromise, wise policy requires a fundamental rethinking of the need for regulation of pesticide residues.

Parts I and II of this article set forth the history of government regulation of pesticides in food, and the new provisions of the FQPA. Part III examines

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of cancer-causing agents").

10. *See id.* at 1644.

11. *See id.*

12. *See id.* (discussing the evolution of the compromise).

13. *See id.* at 1644 (reporting that "both sides won").

14. *See id.*

15. *See* Jonathan Tolman, *The Real Pests Aren't in the Food*, WALL ST. J., Sept. 18, 1996, at A18.

the implications of new legislative requirements and observes how they may perversely increase harm to overall public health.[16] Part IV addresses how such counterproductive effects might be evaded within the statute, but also notes the limitations of such measures. Finally, in Part V, I propose a wiser and more effective regulatory regime for pesticide residues on food.

I. THE DELANEY CLAUSE AND HISTORY OF PESTICIDE RESIDUE REGULATION

Pesticide regulation has been tangled in overlapping dualism. Under FIFRA, the Environmental Protection Agency ("EPA") decides what pesticides and uses are approved for registration in this country.[17] For example, the agency might register pesticide *X* for use on crop *Y*. The test for such registration is functionally a cost-benefit balancing analysis.

Under the FFDCA, EPA regulates the pesticide residues that are allowed on food products sold in interstate commerce.[18] Allowable levels are called tolerances.[19] The Act creates different standards for carcinogenic pesticide residue tolerances in raw foods and processed foods.[20] Tolerances in raw foods were based on a type of cost-benefit balancing,[21] but tolerance levels in processed foods could not consider benefits from the pesticide's use. Consequently, processed food tolerances seemed to fall under the Delaney Clause.

The Delaney Clause amended the FFDCA in 1958 to prohibit the use of

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16. This article assumes that the FQPA will be implemented as written. This may be a bit naive, inasmuch as political influences can cause standards to be based on factors other than those dictated by statute. *See, e.g.*, Sam Peltzman, *Toward a More General Theory of Regulation*, 19 J.L. & ECON. 211 (1976) (claiming that regulation is predictably influenced by extralegal political considerations). The assumption is nonetheless reasonable. Surely it is fair to critique the statutory language, which will inevitably influence implementation even if it is not rigorously obeyed. Moreover, empirical investigation of pesticide regulations demonstrates the presence of political influence but also indicates that basic legal commands are generally followed. *See* Maureen L. Cropper et al., *The Determinants of Pesticide Regulation: A Statistical Analysis of EPA Decision Making*, 100 J. POL. ECON. 175 (1992).

17. Pesticides must be registered under FIFRA before they can be sold or used in the United States. *See* 7 U.S.C. § 136a (1994). EPA may deny any registration, but commonly registers the product with some restrictions, such as labeling requirements. EPA bases its decision regarding registration on consideration of risks to health or the environment, "taking into account the economic, social, and environmental costs and benefits" of the pesticide's use. 7 U.S.C. § 136(bb). Also, EPA may suspend a previously issued registration if necessary to avert an imminent hazard. *See* 7 U.S.C. § 136d(c).

18. *See* 21 U.S.C. § 331 (1994).

19. *See* 21 U.S.C. § 346 (1994).

20. *See* 21 U.S.C. § 346a (1994).

21. The law provides that consideration of tolerances on raw foods shall include "the necessity for the production of an adequate, wholesome, and economical food supply[.]" 21 U.S.C. § 346a(b) (1994).

any food additives in processed food that were found to induce cancer in humans or animals.[22] The Clause states that no additive can be considered safe if "it is found to induce cancer when ingested by man or animal, or if it is found, after tests which are appropriate for the evaluation of the safety of food additives, to induce cancer in man or animal."[23]

The Food and Drug Administration ("FDA") was to implement the Delaney Clause with respect to classic food additives (such as preservatives or color additives) but EPA was charged with implementation for pesticide residues in processed foods.[24] The application of the Delaney Clause to pesticide residues was neither universal nor straightforward. The law effectively grandfathered the legality of any pesticides that had been approved for use at the time of the Clause's passage.[25] The Clause applied only to processed foods, as residues on raw foods were regulated under a cost-benefit balancing standard elsewhere in the Act.[26] The amendment also had a "flow-through" provision that limited Delaney's application. If pesticides on raw foods, approved under the balancing test, simply flowed through as the raw foods were constituents of processed foods, Delaney would not apply.[27] However, if the pesticide residue concentrated at all in the processed foods to a higher level, the clause did apply.[28]

The application of the Delaney Clause to concentration of pesticide residues in processed foods had a significant backward effect. EPA adopted a "coordination policy" to contribute some consistency to the wildly divergent standards within the FFDCA.[29] Under this policy, the Delaney Clause could

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22. For a discussion of the Delaney Clause's legislative history, see Frederick H. Degnan & W. Gary Flamm, *Living with and Reforming the Delaney Clause*, 50 FOOD & DRUG L.J. 235, 237-39 (1995).

23. 21 U.S.C. § 348(c)(3)(A) (1994).

24. EPA received these responsibilities through Reorganization Plan No. 3 of 1970, eff. Dec. 2, 1970. 35 C.F.R. 15623, 84 Stat. 2086 (1970). A pesticide residue in processed food is treated as a food additive subject to section 409. *See, e.g.*, United States v. Ewig Bros., 502 F.2d 715 (7th Cir. 1974).

25. The provisions having this effect are discussed in Douglas T. Sheehy, *A* De Minimis *Exception to the Delaney Clause: A Reassessment of* Les v. Reilly, 50 FOOD & DRUG L.J. 257, 261 (1995).

26. *See* 21 U.S.C. § 346a (1994).

27. *See* 21 U.S.C. § 342(a) (1994).

28. The application of the flow-through provision was elaborated in regulations found at 40 C.F.R. § 180.1(f) (1993).

29. The nature of the coordination policy was explained in an EPA publication:

> [I]t has been EPA's policy to revoke or refuse to issue section 408 raw food tolerances or FIFRA registrations for pesticide uses on foods that might become processed foods in cases where a section 409 tolerance cannot be allowed. Largely because it is often difficult at the time a pesticide is applied to predict whether the crop will be eaten raw or processed, EPA refuses otherwise acceptable raw food tolerances if for some reason a food additive regulation cannot be issued for residues of the pesticide in processed food.

Victor J. Kimm, *The Delaney Clause Dilemma*, 19 EPA J. 39, 40 (1993).

direct standards for residues on raw foods. If a pesticide residue concentrated such that the agency would prohibit any section 409 tolerance under the Clause, the agency would also deny any section 408 tolerance on raw foods, regardless of any cost-benefit balancing.[30] Hence, the possible concentration of pesticide residues on concentrated foods effectively banned the pesticide's application to all foods.

Perhaps the Delaney Clause was a reasonable response to scientific understanding of the time, but it became a dubious regulatory approach.[31] The problems of the Delaney Clause were two-fold. First, science discovered potential carcinogenic effects of a large number of substances, at least where laboratory animals were exposed to the Maximum Tolerated Dose (MTD), an extremely high level of exposure.[32] Second, scientific knowledge advanced to where we could detect extremely minute amounts of a substance. Rather than preventing significant exposures to a few very hazardous substances, the Delaney Clause would preclude minuscule exposures to a broad range of chemicals, even if that exposure produced little or no actual harm.[33]

Recognizing these shortcomings, EPA sought to evade the application of the Delaney Clause to pesticide residues.[34] EPA responded to a National Academy of Sciences report, discussed below, which concluded that rigid application of Delaney could cause more harm than it prevented. In 1988,

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EPA never formally adopted this policy as a rule. The policy was described in connection with other rules. The agency expressed concern that the legality of a pesticide application in the field should not turn on whether the food was ultimately processed, as this would create too much uncertainty. *See* 55 Fed. Reg. 17,560, 17,562 (1990).

30. The existence of this concentration effect is based on scientific data from processing studies. *See* Carol S. Curme, *Regulation of Pesticide Residues in Foods: Proposed Solutions to Current Inadequacies Under FFDCA and FIFRA*, 49 FOOD & DRUG L.J. 609, 641 (1994).

31. *See Hearings Before the Subcomm. on Health and Environment of the House Commerce Comm. on the Food Quality Protection Act of 1995*, 104th Cong. 34-35 (1995) [hereinafter *1995 Hearings*]. In those hearings, Carl Winter, Director of the Foodsafe Program of the University of California, testifies:

> Such an approach may have been prudent in 1958 when our detection capabilities and understanding of the mechanisms of cancer were less developed. In 1995, however, our continued enforcement of the Delaney Clause represents a scientific embarrassment which perpetuates misinformation and increases consumer anxiety rather than providing meaningful public health protection.

Id.

32. *See infra* Part III.A. for discussion of the use of the MTD.

33. The developing shortcomings of the Delaney Clause are well summarized in Richard A. Merrill, *FDA's Implementation of the Delaney Clause: Repudiation of Congressional Choice or Reasoned Adaptation to Scientific Progress?*, 5 YALE J. ON REG. 1, 13-21 (1988).

34. FDA, which was responsible for implementing the Delaney Clause with respect to most food additives, likewise sought to evade the absolute strictures of its provisions. *See* Merrill, *supra* note 33, at 21-33.

EPA changed its historic interpretation of Delaney and issued a policy statement that it would permit carcinogenic residues as long as the consequent risk of cancer was no more than de minimis.[35] The agency defined the de minimis standard as a one in one million (1.0×10^{-6}) risk of cancer over a lifetime.[36]

EPA's efforts to evade the rigid application of the Delaney Clause through a de minimis policy were promptly challenged in court. In *Les v. Reilly*, the Ninth Circuit held that EPA's policy was illegal and set it aside.[37] The court acknowledged the policy shortcomings of the original Delaney approach but stressed that "[r]evising the existing statutory scheme" was not EPA's or the court's responsibility; any change must come from Congress.[38] The parties to the action eventually settled, with EPA agreeing to reconsider processed food tolerances for approximately sixty substances.[39] Given the failure of the administrative corrigent, attention turned to legislation.

The decision in *Les v. Reilly* was immediately threatening to growers. Under the strict application of the Delaney Clause, EPA was forced to prohibit a number of substances. Potato growers, for example, would lose the two fungicides necessary to control "an epidemic of the potato blight that caused the Irish potato famine of the last century."[40] Similar threats were presented to growers of other products, including rice, peanuts, and oranges.[41] These prospective losses no doubt concentrated the attention of the food industry, making it more amenable to the compromises found in the FQPA.

In addition to the Ninth Circuit decision mandating the application of the Delaney Clause, the FQPA provisions were provoked in large part by two reports of the National Research Council of the National Academy of

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35. *See* Regulation of Pesticides in Food: Addressing the Delaney Paradox Policy Statement, 53 Fed. Reg. 41,104 (1988). The FDA had earlier sought to apply a similar de minimis criterion under the Delaney Clause. *See supra* notes 25-28 and accompanying text.

36. *See* 53 Fed. Reg. at 41, 107.

37. 968 F.2d 985 (9th Cir. 1992). An earlier opinion on FDA regulation under the Delaney Clause had reached a similar conclusion, that the Delaney Clause must be literally read. *See* Public Citizen v. Young, 831 F.2d 1108 (D.C. Cir. 1987).

38. 968 F.2d at 990.

39. *See Delaney Clause Suit Sets EPA Timetable*, Food Drug Cosm. L. Rep. (CCH) ¶ 40,684, at 41,050-51 (Feb. 10, 1995).

40. *See 1995 Hearings*, *supra* note 31, at 37 (statement of Leonard P. Giannessi of the National Center for Food and Agricultural Policy).

41. *See 1995 Hearings*, *supra* note 31, at 38 (reporting that "[r]ice growers stand to lose the only fungicide that is effective in controlling rice blast;" that peanut growers "would lose . . . about $18 million of peanuts every year due to competition with weeds due to the use of less effective chemical herbicides;" and that Florida citrus growers "would lose $14 million in oranges every year to a disease that could not be controlled without a Delaney targeted fungicide").

Sciences ("NAS"). The first of these was issued in 1987 and focused on the practical effects of the Delaney Clause and inconsistent standards under the FFDCA.[42] The central theme of the report revolved around the fact that a cost-benefit test was applied to the tolerance for residues on raw agricultural commodities, while the Delaney Clause would bar any tolerance for residues that concentrated in processed food. The NAS reported that it was "unable to identify any sound scientific or policy reason for regulating pesticides present in or on raw commodities differently than those present on processed foods."[43]

The problem with the inconsistent regulatory scheme, however, was not limited to irrationality. The NAS observed that inconsistent standards could increase the risk from pesticides. The NAS reported:

> [s]uppose a registered pesticide *X* with known oncogenic effects and an existing substitute *Y* which is a weaker oncogen are under review. Both agents produce roughly equal benefits for comparable uses. *X* does not concentrate in any processed apple products, but *Y* concentrates marginally. The EPA could be forced by the Delaney Clause to deny a section 409 tolerance for *Y* and also would be compelled to cancel its section 408 tolerance and registration. Pesticide *X* would claim a larger share of the market. Human cancer risk would rise, not fall.[44]

The NAS also observed that the law would prohibit EPA from registering a new, safer pesticide if that new pesticide had any carcinogenic effect and concentration in processed foods.[45] The report characterized these perverse effects from inconsistent standards as the Delaney Paradox.

The Delaney Paradox report was not wholly abstract and theoretical. The NAS computed the effects of a consistent de minimis risk standard for both sections 408 and 409. The report found that such a consistent standard would reduce the overall risk from exposures to many specific pesticides more than the present system, including the Delaney Clause.[46]

The second salient NAS report on pesticides examined the consequences of pesticide residues on infants and children.[47] This report concluded that

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42. *See* COMM. ON SCIENTIFIC AND REGULATORY ISSUES UNDERLYING PESTICIDE USE PATTERNS AND AGRICULTURAL INNOVATION, NATIONAL RESEARCH COUNCIL, REGULATING PESTICIDES IN FOOD: THE DELANEY PARADOX (1987) [hereinafter REGULATING PESTICIDES IN FOOD].

43. *Id.* at 40.

44. *Id.* at 41-42.

45. *See id.* at 42.

46. *See id.* at 121.

47. COMM. ON PESTICIDES IN THE DIETS OF INFANTS AND CHILDREN, NATIONAL RESEARCH

prevailing methods of scientific risk analysis might systematically underestimate the risks of pesticides to infants and children, thereby causing insufficient regulation of pesticide residues on foods. There were two reasons for such incorrect estimates. First, there was relatively little data on food consumption patterns of infants and children. As a result, there was a risk that infants and children received higher exposures than anticipated.[48] Second, infants and children could have particular sensitivity to exposures to pesticide residues. The report outlined a number of reasons to believe that this was the case.[49] This suggested applying an additional margin of safety to protect infants and children from exposure to risk from pesticide residues on food.

The two NAS reports appeared to run in different directions. The first report implied that the apparent harshness of the Delaney Clause was counterproductive, counseling for the sort of de minimis standard that EPA futilely sought to implement administratively. This apparently called for a weakening of the standard. The second report suggested that prevailing standards were insufficient for the protection of infants and children, calling for a strengthening of standards. Rather than fostering a contradiction, however, the two reports in effect provided a recipe for compromise.[50]

II. THE NEW DECISION STANDARD—"REASONABLE CERTAINTY" OF SAFETY WITH QUALIFICATIONS

Environmental laws aimed at protecting human health employ a wide range of decision standards. Some are risk-based, such as those requiring

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COUNCIL, PESTICIDES IN THE DIETS OF INFANTS AND CHILDREN (1993) [hereinafter PESTICIDES IN THE DIETS OF INFANTS AND CHILDREN].

48. Children "consume notably more of certain foods relative to their body weight than do adults" such that "their ingestion of pesticide residues on these foods may be proportionately higher than that of adults." *Id.* at 323. Therefore "risk assessment methods that have traditionally been used for adults may require modification when applied to infants and children." *Id.* at 323. *See also* Robin M. Whyatt & William J. Nicholson, *Conducting Risk Assessments for Preschoolers' Dietary Exposure to Pesticides*, *in* PESTICIDE RESIDUES AND FOOD SAFETY: A HARVEST OF VIEWPOINTS 235, 235 (B.G. Tweedy et al. eds., 1991) (noting that "[y]oung children generally receive greater exposure (in mg/kg bw) than adults to pesticide residues in food due to higher caloric requirements and food intake rates").

49. The report was actually rather cautious in this claim. While noting that infants and children might be especially susceptible to pesticide risk, the report conceded that "empirical evidence to support this is mixed." PESTICIDES IN THE DIETS OF INFANTS AND CHILDREN, supra note 47, at 359. *Id.* at 359. While claiming that for some pesticides, infants and children are at "greater risk than adults," the report also acknowledged that for other pesticides, infants and children might "exhibit less risk." *Id.*

50. *See* Charles Benbrook, *Analysis of the Basic Food, Drug and Cosmetic Act Provisions of H.R. 1627*, attachment to Benbrook letter, *supra* note 5 (saying the FQPA essentially "implement[s] the major recommandations of the two NRC/NAS reports").

standards to ensure a "margin of safety" for public health.[51] Some laws are feasibility-based, mandating that pollution be kept as low as technologically and economically feasible.[52] Still others are based on cost-benefit analysis, proscribing "unreasonable risks."[53]

Rather than embrace any of these extant standards, the FQPA adopted the following decision rule: there must be reasonable certainty of no harm from pesticide residues on food. While not commonly employed in environmental regulation, the decision rule has been used for noncarcinogenic threshold risks from food additives. This standard could well become "the health standard adopted over the next decade or so in other federal environmental and public health law."[54] The Act's new standard is not inflexible: the FQPA also contained some qualifications that theoretically could either increase or decrease permissable tolerances. This section reviews the nature of the new FQPA decision standard.

A. Defining the New Decision Standard

The FQPA directs the Administrator to allow a certain pesticide residue[55] tolerance on foods upon a determination that the level is safe.[56] "Safe" is defined as "a reasonable certainty that no harm will result" from exposure.[57] EPA was to apply this standard not only to new pesticides but also to already registered pesticides, reviewing them and setting new tolerances over the next several years.[58]

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51. *See, e.g.*, Clean Air Act standards for criteria pollutants calling for an "adequate margin of safety," 42 U.S.C. § 7409(b)(1) (1994).

52. *See, e.g.*, Clean Air Act standards for hazardous air pollutants relying on available technology, 42 U.S.C. § 7412(d) (1994); Clean Water Act standards for water toxics based on technological feasibility, 33 U.S.C. § 1311(b)(2)(A) (1994).

53. *See, e.g.*, FIFRA standards for pesticides applying to substances that cause "unreasonable adverse effects," 7 U.S.C. § 136a(c)(5)(D) (1994).

54. Benbrook letter, *supra* note 5. Benbrook predicts this outcome for U.S. law and also notes that other developed nations "are bound to eventually adopt the same or a similar standard for pesticide residue exposures." *Id.*

55. A "pesticide residue" is newly defined as either a pesticide chemical itself or a substance that results from the metabolism or degradation of a pesticide chemical. *See* Food Quality Protection Act of 1996, Pub. L. No. 104-170, § 402(a)(2)(A), 110 Stat. 1489, 1513. The term does not include residues attributable to natural causes or residues that the Administrator considers more appropriately regulated under a different section of the Act. *See* § 402(a)(3)(A) (codified as amended at 21 U.S.C. § 321(q)(3)(A) (1997)).

56. For a good summary of the tolerance-setting process, see WILLIAM H. RODGERS, ENVIRONMENTAL LAW § 5.21(B) (1988). While this discussion predates the FQPA passage, the procedures were not materially changed by that law.

57. Section 405(b)(2)(A) (codified as amended at 21 U.S.C. § 346a(b)(2)(A) (1997)).

58. *See* Brian Broderick, *Worries About Food Safety Law's Effect on Quick Approvals Aired at Group Meeting*, Daily Env't Rep. (BNA), at d17 (Nov. 18, 1996) (discussing EPA's schedule for

The new standard departs not only from the clear but arbitrary standard of the Delaney Clause but also from the decision standard in most other environmental laws. Given the lack of precedent for the decision rule, the FQPA text leaves considerable ambiguity. Specifically, how much certainty is required for reasonable certainty?

The House Report on the FQPA eliminates much of the textual ambiguity. It indicates that tolerance for harmful pesticides with discernible threshold effect should be a level at which 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 public health."[59] In general, reasonable certainty is the application of a hundred-fold safety factor to the "no observable effect" level.[60] The Committee emphasized the importance of the hundred-fold safety standard when data must be extrapolated from animal bioassays.[61]

For the large number of pesticides regulated for cancer risk with no definable threshold exposure,[62] the no observable effect level is not helpful. For these substances a reasonable certainty of no harm exists when "any increase in lifetime risk, based on quantitative risk assessment using conservative assumptions, will be no greater than 'negligible.'"[63] Negligible in turn is defined by the Committee's understanding of prevailing EPA practice of considering a of one-in-one-million lifetime risk to be negligible.[64] While the one-in-one-million level is often considered a "consensus benchmark" for federal regulation,[65] the level is extremely low and quite cautious. One author observed that if the one-in-one-million standard were universally applied "cooks could not cook (benzopyrene and other indoor carcinogens), roads could not be paved (hot asphalt and products of incomplete combustion), dentists could not X-ray (X-rays), anesthesiologists could not anesthetise (halothane), and stone masons (thorium), plumbers (lead, fumes), painters (solvents, epoxides), carpenters

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reassessing all existing tolerances by August 2006).

59. H.R. REP. NO. 104-669, pt. 2, at 41 (1996).

60. *Id.*

61. *See id.*

62. *See* Tina E. Levine, *Assessment and Communication of Risks from Pesticide Residues in Food*, 47 FOOD & DRUG L.J. 207, 210 (reporting that in the U.S. "all carcinogenic pesticides are treated as nonthreshold").

63. H.R. REP. NO. 104-669, pt. 2, at 41 (1996).

64. *See id.*

65. *See, e.g.*, David A. Wirth & Ellen K. Silbergeld, *Risky Reform*, 95 COLUM. L. REV. 1857, 1864 (1995).

(wood dusts) and farmers (UV from sunlight) could not work."[66] In practice, the one-in-one-million standard is not a trigger for regulation in other areas.[67]

While Congress did not incorporate the one-in-one-million standard into the text of the FQPA, the Report warns against any administrative effort to apply a different standard for reasonable certainty of no harm. It provides that if an Administrator seeks to change the definition of reasonable certainty, "the new interpretation should be adopted by regulation and should be at least equally protective of public health."[68] Moreover, the Administrator is to "bear the burden to demonstrate that the revised interpretation is equally protective of the public."[69] The legislative history thus makes quite clear that reasonable certainty of no harm means a lifetime risk no greater than one in one million, probably based on a conservative quantitative risk assessment.

The FQPA creates an exception to its general "reasonable certainty" standard for certain "eligible pesticide chemical residues."[70] In order to be considered an eligible chemical residue for nonthreshold substances, the Administrator must first determine that "the lifetime risk . . . is appropriately assessed by quantitative risk assessment."[71] Once this relatively easy threshold is crossed, the Administrator may permit a tolerance higher than the one-in-one-million test if either of the following two conditions exist: (i) the pesticide protects consumers from adverse effects that would create a higher risk than that from exposure to the residue; or (ii) use of the pesticide is necessary to "avoid a significant disruption in domestic production of an adequate, wholesome, and economical food supply."[72] Even if one of these two conditions is met, the law still imposes quantitative limits on the agency's discretion. The "yearly risk" can be no more than "ten times" the reasonable certainty standard (apparently one in one hundred thousand).

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66. Paul Milvy, *A General Guideline for Management of Risk from Carcinogens*, 6 RISK ANALYSIS 69, 70 (1986).

67. *See* Dennis J. Paustenbach, *A Survey of Health Risk Assessment*, *in* THE RISK ASSESSMENT OF ENVIRONMENTAL HAZARDS 27, 94 (Dennis J. Paustenbach ed., 1989) (citing C.C. Travis et al., *Cancer Risk Management*, 21 ENVTL. SCI & TECH. 415 (1987)) (noting that for risks to the entire population, a risk level below one in one million never triggered action; however, a risk level above three in ten thousand always triggered action"). Thus, in some contexts, agencies have accepted residual risks of more than one in one million. *See id; see also* Frank B. Cross, *Beyond Benzene: Establishing Principles for a Significance Threshold on Regulatable Risks of Cancer*, 35 EMORY L.J. 1 (1986) (reviewing varying standards for de minimis risk among federal agencies).

68. H.R. REP. NO. 104-669, pt. 2, at 41 (1996).

69. *Id.*

70. Food Quality and Protection Act of 1996, Pub. L. No. 104-170, § 405 (b)(2)(B), 110 Stat. 1489.

71. Section 405(b)(2)(B)(i)(I) (codified as amended at 21 U.S.C. § 346a(b)(2)(B)(i)(I) (1997)).

72. Section 405(b)(2)(B)(iii) (codified as amended at 21 U.S.C. § 346a(b)(2)(B)(iii) (1997)). This provision roughly traces the traditional balancing standard for tolerances in raw commodities but adds the requirement of a "significant disruption." *See id.*

Also, the lifetime risk can be no greater than twice that standard (apparently one in five hundred thousand).[73]

The House Report again adds some clarity to the new provisions on eligible residues. The first condition is designed to deal with situations where "eating food treated with the pesticide chemical is safer for consumers than eating the same food that is not treated with the pesticide."[74] An example might be a fungicide that controlled food contamination from the known carcinogenic fungus of aflatoxin.[75]

The Report also establishes some criteria for the second condition, which allows higher tolerances to protect against a significant disruption of food supply. In applying this condition, the Administrator "is expected to take into account the availability and effectiveness of alternative pest control methods, the impact of loss of the pesticide on crops, the impact of the national availability and cost of food combined with the dietary impact of such loss, and the impact on the ability of consumers to access a nutritious food supply."[76] An FDA decision rendered in the 1980s to allow higher aflatoxin exposure on corn and thus to "avoid widespread shortages of animal feed" is presented as a "representative" example of the sort of significant disruption envisioned by the second condition.[77] These health considerations are permitted only for evaluating existing tolerances, however, and are not for use in establishing new tolerances.[78]

Both the exception conditions for eligible pesticide residues permit the Administrator to establish higher tolerances than otherwise permitted. The practical effect of the conditions is uncertain for reasons discussed below. Significantly, even when an exceptional condition applies, there is a strict tolerance ceiling that the Administrator may not evade.[79] With respect to this ceiling, there is little room for consideration of pesticide benefits, even health benefits.

B. Exposure of Infants and Children

The FQPA added a provision to existing law in an attempt to ensure

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73. *See* § 405(b)(2)(B)(iv) (codified as amended at 21 U.S.C. § 346a(b)(2)(B)(iv) (1997)).

74. H.R. REP. NO. 104-669, pt. 2, at 42.

75. *See id.*

76. *Id.*

77. *See id.* at 42-43.

78. *See* Office of Pesticide Programs, Envt'l Protection Agency, *Consideration of Pesticide Benefits Under FQPA* 1, 1 (last modified Oct. 9, 1996) <http://www.epa.gov/opppspsl/fqpa/benefits.htm>.

79. *See id.*

consideration of the unique exposure conditions and susceptibilities of infants and children. This subsection directs the Administrator to consider the risk of pesticide residues in light of particular consumption patterns and the special susceptibility of infants and children, as well as unique cumulative effects of common toxicity mechanisms on infants and children.[80] Once these factors have been considered, the Administrator must ensure to a reasonable certainty that no harm will occur to infants and children and publish a specific determination of this effect.[81] Moreover, when information on effects is incomplete for pesticides with threshold effects, the Administrator must apply an additional factor of ten to the (otherwise hundredfold) margin of safety for infants and children.[82] The legislative history adds little to the understanding of this new provision, save for a reference to the NAS report on pesticide exposures of infants and children.[83] In practice, EPA will explicitly determine that a tolerance is safe for children, consider the need for an additional safety factor of ten in order to account for uncertainty in the data, and consider any special exposures or sensitivities children may have to the pesticide in question.[84]

In limited practice under the new law, the concern for children has not had a material impact.[85] EPA does not consider itself bound by its initial tolerance setting,[86] however, and the special consideration could potentially cause a significant reduction in tolerances.

C. Cumulative Effects of Exposures and Common Toxicity Mechanisms

Another new provision found in the FQPA requires consideration of

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80. Food Quality Protection Act of 1996, Pub. L. No. 104-170, § 408(b)(2)(C), 110 Stat. 1489, 1514. Within the special susceptibility criterion, the statutory text specifically references "neurological differences between infants and children and adults, and effects of in utero exposure to pesticide chemicals." Section 408(b)(2)(C)(i)(II) (codified as amended at 21 U.S.C. § 346a(b)(2)(C)(i)(II) (1997)).

81. *See* § 408(b)(2)(C)(ii) (codified as amended at 21 U.S.C. § 346a(b)(2)(C)(ii) (1997)).

82. *See id.*

83. *See* H.R. REP. NO. 104-669, pt. 2, at 43.

84. PR Notice No. 97-I, *supra* note 6.

85. *See, e.g.*, 62 Fed. Reg. 1284, 1287 (1997) (to be codified at 40 C.F.R. pt. 180) (finding no need for special consideration of infants and children given myclobutanil exposure levels); 62 Fed. Reg. 1288, 1291-92 (1997) (tolerance for zinc phosphide requires no special consideration because no evidence indicates special sensitivity); 62 Fed. Reg. 5333, 5335-36 (to be codified at 40 C.F.R. pt. 180) (1997) (tolerance for glufosinate ammonium requires no special consideration for infants and children); 62 Fed. Reg. 4911, 4914 (to be codified at 40 C.F.R. pt. 180) (1997) (tolerance for carboxin requires no special consideration).

86. *See* 62 Fed. Reg. 5370, 5371 (1997) (to be codified at 40 C.F.R. pt. 180) (declaring that "these early tolerance and exemption decisions will be made on a case-by-case basis and will not bind EPA as it proceeds with further rulemaking and policy development").

various factors in "establishing, modifying, leaving in effect, or revoking a tolerance or exemption for a pesticide chemical residue."[87] These factors include "available information concerning the cumulative effects of such residues and other substances that have a common mechanism of toxicity."[88] A related factor is the information concerning "aggregate exposure levels of consumers ... to the pesticide chemical residue and to other related substances, including dietary exposure under the tolerance and all other tolerances in effect for the pesticide chemical residue, and exposure from other non-occupational sources."[89]

The Act provides that EPA must aggregate total exposures from water, residential, and lawn care uses with dietary exposures in setting a standard. This provision seeks to prevent a large cumulation of small risks. The Act also calls for cumulation of exposures from these pathways for different pesticides that have a common mechanism of toxicity. Whole groups of pesticides could therefore be grouped within a single tolerance.

The House Report adds nothing to the understanding of this new provision, although the purpose of the provision seems clear. Environmental groups observed that the seemingly protective one-in-one-million risk standard was not so protective when consumers were exposed to that standard in food residues, with additional exposure from other sources. Suppose that the one-in-one-million standard were applied to ten different crop uses of pesticide *X*. A consumer who ate the maximum tolerated amount of residue on each crop would therefore have a consequent risk of ten in one million (one in one hundred thousand). The consumer might also be exposed to pesticides in the water supply and other sources that could increase the risk further. The consideration of "cumulative effects" and "aggregate exposure" is intended to avoid increasing the risk level through multiple exposure sources.

The application of the cumulative risk standard raises a host of significant implementation questions. EPA could either require data that demonstrate a common mode of action, use Structure Activity Relationships[90] to group

87. Section 408(b)(2)(D) (codified as amended at 21 U.S.C. § 346a(b)(2)(D) (1997)).

88. Section 408(b)(2)(D)(v) (codified as amended at 21 U.S.C. § 346a(b)(2)(D)(v) (1997)). This Section also requires consideration of other important factors. For example, the Administrator is to consider the quality of the available scientific information on health effects from pesticide exposure. *See* § 408(b)(2)(D)(i)-(iii) (codified as amended at 21 U.S.C. § 346a(b)(2)(D)(i)-(iii) (1997)). The Administrator also is to consider the existence of population groups with unusually high exposures or susceptibility to pesticide residues. *See* § 408(b)(2)(D)(iv), (vii) (codified as amended at 21 U.S.C. § 346a(b)(2)(D)(iv) and (vii) (1997)).

89. Section 408(b)(2)(D)(vi) (codified as amended at 21 U.S.C. § 346a(b)(2)(D)(vi) (1997)).

90. Structure Activity Relationships (SARs) compare the chemical structure of substances and presume that relatively similar structures produce similar effects.

chemicals, or group all chemicals with the same or similar endpoints (e.g., carcinogenicity at a particular body site, such as thyroid tumors in experimental animals). The first approach would be the most precise and restrictive, but EPA currently does not collect data for this determination and doing so would be costly and time-consuming.[91] The third approach would be most precautionary, but would result in many false positives.[92] EPA's present intention is to combine the second and third approaches, grouping chemicals that cause the same effect through the same pathway, as determined through structural configurations.[93]

In December, 1996, EPA's Office of Prevention, Pesticides & Toxic Substances ("OPPTS") adopted an "interim decision logic" for the application of the FQPA pending final and formal risk assessment policies.[94] The interim logic creates a screening model that assigns exposure levels to different pathways and creates an overall "risk cup" of total risk exposure.[95] Presumably, once the cup is full, meaning that exposures equal but do not exceed the statutory threshold risk, the agency will not approve any more uses of a pesticide. The logic also considers cumulation through common mechanisms of toxicity. Many different pesticides are within the general group known as organophosphates. To the extent that such pesticides have the same mechanism of toxicity, the agency may group them for purposes of tolerance setting, and may not permit overall exposures higher than the tolerance level.

While the interim decision logic did not expressly apply to carcinogenic pesticides, EPA has applied a similar procedure for these substances. The cumulative effects standard was applied in the context of a petition for an emergency time-limited tolerance for the pesticide lactofen.[96] EPA grouped lactofen with four other related pesticides, in the category diphenyl ethers. Given the absence of scientific information, EPA initially observed that it

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91. *See* Office of Pesticide Programs, Envt'l Protection Agency, *FQPA—Food Safety Advisory Committee Risk Issues Subgroup Aggregate Exposure and Common Mode of Action* 2 (last modified Oct. 9, 1996) <http.//www.epa.gov/opppsI/fqpa/riskiss.htm>.

92. *See id.*

93. *See id.* The adoption of this approach is not yet official. The agency has elsewhere stated that if "a pesticide shares a common toxicological endpoint and structural similarity with other substances, EPA will assume that a common mechanism of toxicity may exist." PR Notice 97-1, *supra* note 6. The latter procedure appears similar to the third approach. Moreover, Don Barolo, the director of the OPP, has said that EPA would "apply conservative assumptions to protect safety" in this determination. *See* Brian Broderick, *Determining Risks under FQPA Considered by EPA Advisory Group*, Daily Env't Rep., at d6 (Oct. 24, 1996).

94. *See EPA Managers Approve Food Safety Law 'Interim Decision Logic'*, INSIDE EPA'S RISK POL'Y REP., December 20, 1996, at 7, 7.

95. *See id.*

96. *See* 61 Fed. Reg. 65,395 (1996).

was "premature to add the risk from these chemicals," but then deemed it "prudent" to cumulate the risk under FQPA.[97] The carcinogenic risk from lactofen was estimated to be only 1.54×10^{-8}, but other diphenyl ethers had a risk as high as 1×10^{-5} for diclofop methyl. Because the risk for these other diphenyl ethers is so high, the FQPA might appear to preclude any new tolerances for diphenyl ethers. However, EPA observed that a lactofen's contribution to aggregate cancer risk is insignificant compared to the other diphenyl ethers.[98] EPA also observed that risk estimate for diclofop methyl and other ethers were based on exposures higher than those in the new standard. Once anticipated residues and new cancer potency levels were revised, "the projected risks will be much lower than 1×10^{-6} for all of these chemicals."[99] EPA finally noted that it, along with industry, was "developing a methodology for determining whether or not multiple exposures will occur and with what frequency for these and other chemicals."[100] If such exposures were infrequent, EPA suggested that it would be inappropriate to cumulate the risks.[101]

Lactofen provides an interesting example of the cumulative risk provision. EPA permitted the tolerance in large part because the substance "contributes insignificant chronic toxicity and carcinogenic risks as compared to the other diphenyl ethers."[102] It is not plain that this is a relevant fact under the FQPA because the law's required cumulation was arguably aimed at such small additional incremental risks. Perhaps EPA recognized this fact and independently justified the tolerance on grounds that diphenyl ethers presented a cumulative risk of less than 1×10^{-6}. However, this independent escape may be unavailable for more significant groups of pesticides such as organophosphates.

Significantly, the cumulative effects of common toxicity mechanisms permit some limited consideration of pesticide benefits. When the cumulative residues of a group of pesticides threaten to exceed the standard, EPA may use a benefits assessment to determine which applications within the risk cup are the most beneficial. Then, if a new and valuable use comes along, EPA may use benefits considerations to cancel existing uses in order to "make room" for a new application.[103] This consideration is quite limited, however,

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97. *See id.* at 65,399.
98. *See id.*
99. *Id.* at 65,400.
100. *Id.*
101. *See id.*
102. *Id.* at 65,398.
103. *See* Office of Pesticide Programs, *supra* note 78, at 2. EPA's authority in this matter is somewhat limited by external realities. Pesticide manufacturers may withdraw or voluntarily cancel

in that it applies only within a given risk cup.

D. The Exemption Process

Federal law has long provided for emergency exemptions to pesticide residue regulations. The FQPA retains a strict exemption process for pesticide tolerances. The Administrator can establish a new exemption or leave an existing exemption in place only after determining that "the exemption is safe" under the reasonable certainty of no harm test.[104] The exemption process also incorporates the cumulative effects test; thus, a determination to a reasonable certainty that no harm "will result from aggregate exposure to the pesticide chemical residue" is required.[105] This aggregate exposure must include exposures even beyond those in the diet.[106] Exemptions are also prohibited unless there is either a practical method for measuring residues or a reason why such a method is not needed.[107] Prior to the FQPA, EPA was not required to establish an exposure tolerance in connection with an emergency exemption, but the new law directs EPA to provide for a time-limited tolerance.[108]

The specific language of the exemption provision makes it appear quite rigorous and difficult to satisfy. Industry representatives have cautioned that the new exemption process threatens a "train wreck" and "crop disasters."[109] EPA's Associate Assistant Administrator for OPPTS has declared that such exemptions are "harder to achieve" under the new statute than under preceding law.[110] Nevertheless, EPA has begun issuing and denying emergency exemptions under the new authority[111] even though final regulations for such exemptions are at least months away. Producers tend to seek exemptions for temporary applications of pesticides on crops for which

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uses in order to make more room for their other products. *See id.* The application with the most benefits is not necessarily the most profitable for the manufacturer. In practical effect, the manufacturer can ultimately control which application is cancelled.

104. *See* Food Quality and Protection Act of 1996, Pub. L. No. 104-170, § 408(c)(2)(A), 110 Stat. 1489, 1514.

105. *See* 408(c)(2)(A)(ii) (codified as amended at 21 U.S.C. § 346a(c)(2)(A)(ii) (1997)).

106. *See* H.R. REP. NO. 104-669, pt. 2, at 45.

107. *See* § 408(c)(3) (codified as amended at 21 U.S.C. § 346a(c)(3) (1997)).

108. *See* § 408(l)(6) (codified as amended at 21 U.S.C. § 346a(1)(6) (1997)).

109. *See* Broderick, *supra* note 58 (quoting Rick Holt of DuPont Agricultural Products).

110. *See* Brian Broderick, *EPA Announces new Section 18 Approvals, Holds Meeting on New Law*, Daily Env't Rep., at D5 (Nov. 22, 1996) (quoting Jim Aidala).

111. *See, e.g.*, 61 Fed. Reg. 65,395 (1996)(lactofen); 61 Fed. Reg. 63,721 (1996) (triadimefon); 61 Fed. Reg. 58,135 (1996) (propiconazole). Exemptions were denied for requested uses of lactofen and esfenvalerate based on dietary risk concerns. Office of Pesticide Programs, Envt'l Protection Agency, Emergency Exemptions and the FQPA (last modified Sept. 25, 1996) <http://www.epa.gov/apppspsl/fqpa/r.

the product is not yet registered. The exemptions are important in this context but are seldom used to increase a tolerance level that has already been set.

III. CONSEQUENCES OF THE NEW DECISION RULE

The FQPA was intended to lend coherence to the law regulating pesticide residues in food, avoiding the Delaney Paradox, while still providing additional protections, particularly to infants and children. This section examines the likely consequences of the law. The short- and long-term effects of the legislation seem apparent. After the FQPA's passage, EPA withdrew a number of revocations and permitted continued applications[112] of substances whose tolerances it had previously revoked after a federal court ordered EPA to take the Delaney Clause literally.[113]

Over the long term, however, the FQPA is likely to reduce overall pesticide usage. The former director of the NAS agricultural board, Charles Benbrook, has estimated that "about two-thirds of the existing tolerances on the books will be affected, and about one-half of these will be affected significantly, *i.e.*, lowered more than 10-fold."[114] Thus the law is expected to cause a considerable reduction in pesticide usage, but the health consequences of this reduction are less clear.

Before projecting health consequences of the FQPA, some basic understanding of the problem is required. Therefore, the initial subsection below examines the nature of the public health risk posed by pesticide residues. Then, in discussing the full implications of the statute, this section presents some new and continued health paradoxes persisting under the FQPA. These paradoxes prove that while the Delaney Clause was theoretically indefensible, the compromises of the FQPA offer little or no improvement.

A. Public Health Risks of Pesticide Residues

The central motivation behind the FQPA is its concern over the public health risks due to exposure to pesticide residues. Any investigation of the merits of that legislation must account for the nature of those risks.

Often assumptions are made without evidence. Some believe that the use of various pesticides is routine or even universal within agriculture. Many

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112. *See id.*

113. The effect of *Les v. Reilly* on proposed revocations is discussed in the notice at 61 Fed. Reg. 50,684 (1996) (to be codified at 40 C.F.R. pt. 185) (discussing consent decree arising out of litigation and consequent revocation of seventeen tolerances).

114. Benbrook, *supra* note 5.

people fear that material residues of pesticides remain on produce that is not labeled organic. In addition, there is a widespread concern that pesticides may cause cancer or other ailments.[115]

In 1987, the NAS Delaney Paradox, in response to an EPA request for risk assessment, report provoked considerable concern over the effects of pesticide applications on public health.[116] The Academy used the then prevailing EPA tolerances for residues on foods and the conventional linear risk assessment models developed by the agency to provide an estimate of cancer risk.[117] This produced a conservative estimate of what one might call "legally-allowable risk." Estimating an individual's risk from seventy years of exposure to the maximum allowable exposure of twenty-eight carcinogenic pesticides yielded a total risk of about six in one thousand.[118] This level is extremely high for environmental risks of cancer and could imply that exposure to pesticide residues caused tens of thousands of cancer cases annually.

By design, the NAS study was a measure of maximum legally allowable risk, not actual measured risk,[119] but this distinction was readily overlooked.[120] Many Americans believe that industry will pollute in an amount up to and perhaps exceeding that allowed by law. While this assumption may have some merit in circumstances involving air and water pollution, it is extremely inaccurate in the case of pesticides. Scrutiny of the

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115. *See, e.g.*, Carl K. Winter, *Pesticide Tolerances and Their Relevance as Safety Standards*, 15 REG. TOXICOLOGY & PHARMACOLOGY 137, 137 (1992) (reporting that "80% of American Shoppers consider pesticide residues to be a major concern").

116. *See* REGULATING PESTICIDES IN FOOD, *supra* note 42.

117. *See* Archibald & Winter, *supra* note 7, at 26-27 (summarizing the NAS study).

118. *See id.* at 27.

119. *See id.* (referring to the NAS estimate as one of "legally allowable risk"). This is not a measure of actual risk, as the NAS explains:

> The EPA traditionally has estimated dietary exposure conservatively by incorporating worst-case assumptions. Pesticide residues are assumed to be present in foods at the published tolerance level. The agency also generally assumes that 100 percent of the acreage of a crop that could be treated with a pesticide will be treated. Estimating exposure in this way nearly always produces an overestimate of actual dietary exposure. . . .

REGULATING PESTICIDES IN FOOD, *supra* note 42, at 59. The agency itself refers to its calculation as the Theoretical Maximum Residue Contribution ("TMRC") and "has acknowledged for a long time the shortcomings of this method." *Id.* at 60. *See also* Winter, *supra* note 115, at 141 (noting that the TMRC calculation assumes that "100% of the crops that may be legally treated with a pesticide are in fact treated and that residues are always present at the tolerance levels").

120. *See* Winter, *supra* note 115, at 149 (observing that "this mathematical construct has often been improperly substated as an indicator of *actual* pesticide exposure"); Leonard P. Gianessi, *Use of Pesticides in the United States*, *in* PESTICIDE RESIDUES AND FOOD SAFETY, *supra* note 48, at 24, 25 (noting that NAS used "totally unrealistic assumption" of maximum allowable risk). The presumption that producers "blindly . . . use prophylactic spraying of all recommended pesticides every year" is a "simplistic straw man." *Id.* at 27.

facts relating to exposure indicates that health concerns about pesticide residues are substantially overblown.

In some contexts, industry resists pollution control measures because such technologies can have considerable cost. For pesticides, however, it is the chemicals themselves that cost money, so agricultural producers have a financial incentive to use as little as possible. The NAS reports that farmers spent about five billion dollars on pesticides in 1984.[121] In this context, producers have a financial incentive to minimize their pesticide usage independent of government regulation.

The use of "legally allowable risk" in risk estimation inevitably leads to "a substantial upward bias in risk estimates."[122] Data are available to demonstrate the magnitude of the upward bias. For example, twelve different pesticides may be used in growing tomatoes. Data from California reveal that the most commonly used of these pesticides is applied to only 26.42% of the tomato crop in the state.[123] The pesticide of median use was applied to only about four percent of the crop.[124] Fifty-four insecticides were approved for use on tomatoes, yet no California grower used more than five insecticides at a time, and most growers used either one or zero insecticides.[125]

Obviously, pesticides are employed much less than legally allowed, and in the case of tomatoes, the upward bias from this factor alone is about twenty-five fold. Tomatoes are not unique; similar results were found for other crops, including apples, lettuce, and oranges.[126] Although authorized by the government, many pesticides often are not applied.[127]

Furthermore, even when pesticides are applied in the field, they do not necessarily remain present on the harvested crop. Residues in the field may volatilize into the air, be removed by water, degrade from sunlight and other

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121. *See* REGULATING PESTICIDES IN FOOD, *supra* note 42, at 49. While this represented only 4% of all production costs for all of agriculture, *id.*, the percentage could be much higher for individual farms that use the chemicals. As discussed below, many producers require little or no pesticide use.

122. Archibald & Winter, *supra* note 7, at 35.

123. *Id.* at 37 tbl. 1-9.

124. *See id.*

125. *See* Winter, *supra* note 115, at 143-44 (citing C.F. CHAISSON ET AL., PESTICIDES IN OUR FOOD: FACTS, ISSUES, DEBATES AND PERCEPTIONS (1987)); *see also* Gianessi, *supra* note 120, at 25 (noting that "only about one-half of the nation's wheat acreage is treated with any pesticides at all—usually just a single herbicide").

126. *See* Gianessi, *supra* note 120, at 25. The other crops have at least one pesticide that is used more broadly on virtually all planted acres. However, a considerable number of permitted pesticides are used on less than one percent of the acreage. *See id.*

127. The FQPA recognizes this fact and allows tolerances to be adjusted according to the percent of food actually treated. Section 408(b)(2)(F) (codified as amended at 21 U.S.C. § 346a(b)(2)(F) (1997)). This adjustment may be used, however, only when reliable data are available for the specific application, exposure data are not underestimated for any significant subpopulation, and exposure is not higher in a particular area. *See id.*

factors, or dissipate as the product grows.[128] Given enough time, residues will inevitably approach zero.[129] In some cases, pesticides are applied to the soil and are not even absorbed by the plants grown.[130] Thus, pesticides are not so ineluctably persistent as may be presumed.

Evidence demonstrates that the pesticide residues resulting even from treated acreage tend to be much lower than the tolerances. Again according to California data,[131] some crops with permitted pesticides such as bananas and beans had zero residue on all tested produce.[132] For many other crops, only a small percentage of the tested produce had any positive residues.[133] For these crops on which residues were found, within a majority of samples (celery, peaches, strawberries, sweet potatoes), the levels of those residues were typically less than 10% of the maximum allowable residue.[134]

Such low residue levels are not unique to California. An FDA study found a very low percent of samples had positive residue detections.[135] Other studies confirm these results.[136] A 1988 joint sampling by EPA and the National Food Processors Association found that 81% of sampled products had no detectable residues.[137] Moreover, sampling procedures may be skewed to overstate the presence of residues, so true public exposure levels are lower than the low estimates yielded by residue analysis.[138] Even

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128. *See* PESTICIDES IN THE DIETS OF INFANTS AND CHILDREN, *supra* note 47, at 206 (discussing these and other mechanisms through which residues dissipate).

129. *Id.* at 207. *See* COMM. ON COMPARATIVE TOXICITY OF NATURALLY OCCURRING CARCINOGENS, NATIONAL RESEARCH COUNCIL, CARCINOGENS AND ANTICARCINOGENS IN THE HUMAN DIET 246 (1996) [hereinafter CARCINOGENS AND ANTICARCINOGENS IN THE HUMAN DIET] (noting that pesticide residues depend on various factors, including "the time between pesticide application, harvest, and sampling; and the degree of postharvest processing"). Moreover, washing, blanching, and canning all reduce pesticide levels. *See id.*

130. *See* PESTICIDES IN THE DIETS OF INFANTS AND CHILDREN, *supra* note 47, at 227.

131. California has by far the most extensive monitoring program for food pesticide residues. *See id.* at 220.

132. *See* Archibald & Winter, *supra* note 7, at 42.

133. *See id.* The percentages of positive residues for various common crops were as follows: apples (10.48%); broccoli (4.62%); grapes (20.62%); onions (11.88%); potatoes (9.17%); and spinach (24.03%). *See id.*

134. *See id.*

135. *See* PESTICIDES IN THE DIETS OF INFANTS AND CHILDREN, *supra* note 47, at 244-45. The median number of positives found for several dozen pesticides was less than five percent. *Id.* When detected the residues were typically at levels well below the EPA tolerance. *See id.* at 256.

136. *See* Winter, *supra* note 115, at 143 (reporting that "residue monitoring data for the past several years have consistently shown that residue levels rarely approach or exceed tolerance levels and that residues have not been detected in the majority of samples analyzed").

137. *See* PESTICIDES IN THE DIETS OF INFANTS AND CHILDREN, *supra* note 47, at 221. More recent sampling have found even lower levels of residues. *See id.* at 222 (saying a National Food Professors Association study from 1988-93 found residues on 97.5% of all samples to be below limits of quantification).

138. *See id.* at 226 (noting that "[s]ampling may be biased to seek positive results when

controlled field studies may overstate actual residues, as demonstrated by the following table, which displays tolerances (in ppm), anticipated residues based on field studies, and the maximum actual observed residue levels for a major pesticide (captan) in the state of California.[139]

TABLE 1
Summary of Captan Residue Data

Commodity	Tolerance	Field Study	Maximum Observed
Almonds	2	0.14	<0.01
Apricots	50	4.98	5.0
Cherries	100	18.59	20.0
Nectarines	50	2.17	<0.01
Peaches	50	6.59	10.0

The highest residue levels actually observed are often much less than those estimated by controlled field studies; they are always much less than the allowed tolerance.[140] Another study suggests that "pesticide use restrictions on fruit and vegetable production and processing would have extremely small or even infinitesimal effects on pesticide residues in food."[141]

Legally allowable exposure is an unreliable proxy for actual exposure.[142] Furthermore, the overstatement of exposure applies to the most hazardous substances. The NAS theorized that the major contributor to cancer risk from pesticide applications on tomatoes was chlordimeform, but the California

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application of the pesticides is known" and may not occur when it is known that pesticides were not applied).

139. The chart is taken from Winter, *supra* note 115, at 139 (citing C.F. CHAISSON ET AL., PESTICIDES IN OUR FOOD: FACTS, ISSUES, DEBATES AND PERCEPTIONS (1987)).

140. Notwithstanding the extremely small risk, EPA has banned the use of captan on forty-four fruits and vegetables. Cropper et al., *supra* note 16, at 190 n.10.

141. C. Robert Taylor, Economic Impacts and Environmental and Food Safety Tradeoffs of Pesticide Use Reduction on Fruits and Vegetables, at 19 (1995) (unpublished research paper, Auburn Department of Agricultural Economics and Rural Sociology).

142. *See* Archibald & Winter, *supra* note 7, at 46 (reporting that "[o]ur analysis clearly shows that tolerances do not equal exposure and demonstrates that the use of tolerance values to calculate risk is not appropriate"). *See also 1995 Hearings*, *supra* note 31, at 35 (statement of Carl Winter):

> Previous risk assessment efforts have commonly focused on identifying the maximum legal exposures which assume that all food items are treated with all possible pesticides, that the residues are always present at the maximum allowable levels, and that the residues in the fields are the same as those on our plates.
>
> This approach ignores substantial evidence demonstrating that the actual use of pesticides in food crops is much less than 100 percent, that the average residues are present at small fractions of the allowable levels, and that things you and I may do in our own kitchens such as washing, peeling, and cooking foods may serve to decrease residues dramatically.

Id.

laboratory detected no residues of this pesticide on any tomato samples.[143]

Yet even these low numbers from the California laboratory may overstate exposure: residues when eaten can be much lower than those measured at harvest due to further degradation and kitchen preparation methods.[144] Actual residues may exceed the allowable tolerance, but this is relatively uncommon.[145] For example, merely washing produce can reduce residues by 97%.[146] The dissipation of residues is well demonstrated empirically: a study on tomatoes found field residues of a pesticide at 17.4% of the tolerance, but the concentration declined to less than 0.2% of the tolerance by the time the produce reached the grocery store.[147] Carl Winter of the University of California at Davis estimates that "more realistic measures of exposure are commonly thousands to hundreds of thousands of times lower than those obtained by estimating the maximum legal exposures."[148] Another study of eight carcinogenic pesticides "found that actual human exposures in food had been overestimated by factors of 99,000 to 463,000."[149] When the NAS estimates were recalculated for specific pesticides based upon actual dietary exposures, the true risks "were from 4600 to nearly 100,000 times lower."[150]

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143. *See id.* at 44.

144. *See id.* at 35 (statement of Carl Winter). Residues are measured on unwashed fresh produce, with the skin or peel left intact. *See* Philip H. Abelson, *Pesticides and Food*, 259 SCIENCE 1235 (1993); *see also* Charles M. Benbrook, *What We Know, Don't Know, and Need to Know about Pesticide Residues in Food, in* PESTICIDE RESIDUES AND FOOD SAFETY, *supra* note 48, at 140, 144. Benbrook observes: "[S]ubsequent handling, washing, processing, and cooking of the crop typically reduces the level of residues in the food as ultimately consumed by the public". *Id.* Benbrook goes on to observe that these factors may dissipate all of the residues on the food actually consumed. *See id.*

145. *See* REGULATING PESTICIDES IN FOOD, *supra* note 42, at 61 (GAO and FDA have found residues exceeding tolerances in 3-4% of samples). Most of this 3-4% violation rate is not attributable to high exposures on permitted crops but is due to the identification of some residue, often minuscule, on crops for which no tolerance has been established. *See* Archibald & Winter, *supra* note 7, at 36; *see also* Charles L. Trichilo & Richard D. Schmitt, *The Role of the Environmental Protection Agency in Assuring a Safe Food Supply, in* PESTICIDE RESIDUES AND FOOD SAFETY, *supra* note 48, at 286, 288 (most violations were not quantitative exceedances of tolerances but presence of some residues on commodities for which tolerances were not established). It is also salient that "consumer exposure to violative residues would usually be of short duration, while the risks of cancer and most other toxic effects typically require continuous exposure to a chemical for periods of weeks to years." *Id.* at 46.

Other research has found a violation rate of only one percent. *See* Abelson, *supra* note 144; *see also 1995 Hearings, supra* note 31, at 171 (statement of Professor C. Robert Taylor) (reviewing studies finding violation rate of 1.1% to 1.84%). Even when violations occur, they generally present no public health risk. *See* Winter, *supra* note 115, at 145-47 (noting that tolerances are often set below levels of safety or negligible risk).

146. *See* Henry B. Chin, *The Effect of Processing on Residues in Foods, in* PESTICIDE RESIDUES AND FOOD SAFETY, *supra* note 48, at 177 (reporting study of pesticide residues on tomatoes).

147. Gary L. Eilrich, *Tracking the Fate of Residues from the Farm Gate to the Table, in* PESTICIDE RESIDUES AND FOOD SAFETY, *supra* note 48, at 209.

148. *1995 Hearings, supra* note 31, at 35.

149. HARVARD CENTER FOR RISK ANALYSIS, RISK IN PERSPECTIVE, Mar. 1995, at 1.

150. Archibald & Winter, *supra* note 7, at 39.

Adopting the lower end of this overstated risk level as typical would still reduce the overall risk from pesticide residues from 6 in 1000 to about 2 in 1,000,000. Taking the upper end would reduce the risk to 1 in 10,000,000. An FDA analysis of actual consumption, the Total Diet Study, has found that "dietary intakes of pesticides are usually less than 1% of the Acceptable Daily Intakes (ADIs) established by the United Nations' Food and Agriculture Organization and the World Health Organization."[151] The Green Mountain Institute for Environmental Democracy, which studies comparative risks, took note of the low residue levels and reported that pesticides on food present "very little if any risks to the general population."[152]

These extremely low risk figures assume that all of the consumed pesticides are absorbed and have a consequent effect on health. Yet recent research suggests that even some consumed pesticides might have no effect. The pesticides might be bound in such a way that they are not biologically available and are harmlessly excreted.[153]

Absent bioavailability, even the above risk figures might be overstated in actual fact. Thus, scientists distinguish between the absorbed dose ingested and the internal, or effective dose, which is "the amount of a risk agent reaching a tissue or an organ where it inflicts damage."[154] For at least one pesticide, atrazine, residue-based exposure assessments overestimate actual risk because its "metabolites have been shown to pass through animals rapidly and essentially unchanged."[155]

A final reason why the NAS overstated the public health risk of pesticides lies in its risk assessment methodology. The Academy used EPA procedures which embrace a "conservative policy in estimating risk" that "probably overstate[s] true oncogenic risk."[156] In effect, "all components of the risk analysis are taken at their most conservative value."[157] Dated assumptions about dietary patterns further exaggerate the estimated risk.[158] While this

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151. Pasquale Lombardo & Norma J. Yess, *The Food and Drug Administration Program on Pesticide Residues in Food, in* PESTICIDE RESIDUES AND FOOD SAFETY, *supra* note 48, at 162, 166.

152. Green Mountain Institute for Environmental Democracy, *Technical Reviews,* SYNERGY, Nov./Dec. 1996, at 13.

153. *See* Sam Kacew et al., *Bioavailability of Bound Pesticide Residues and Potential Toxicologic Consequences—An Update*, 211 PROC. SOC'Y EXPERIMENTAL BIOLOGY & MED. 62 (1996).

154. JOHN J. COHRSSEN & VINCENT T. COVELLO, RISK ANALYSIS: A GUIDE TO PRINCIPLES AND METHODS FOR ANALYZING HEALTH AND ENVIRONMENTAL RISKS 75 (1989).

155. Larry G. Ballantine & Bruce J. Simoneaux, *Pesticide Metabolites in Food, in* PESTICIDE RESIDUES AND FOOD SAFETY, *supra* note 48, at 96, 104.

156. REGULATING PESTICIDES IN FOOD, *supra* note 42, at 50.

157. Archibald & Winter, *supra* note 7, at 34-35.

158. *See id.* at 36-38.

approach is not uncommon in the context of government regulation of environmental carcinogens, the practice overstates the actual public health threat, often considerably.[159] The risk estimates obtained from conservative assumptions are the "*highest* probability of increased incidence of cancer . . . Actual incidence of cancer should be lower than the calculated estimate, and may even be zero."[160]

B. Persisting Paradoxes of Pesticide Policy

The FQPA was aimed at a central policy paradox identified by the NAS. The law successfully eliminated a set of inconsistent standards that produced perverse regulations contrary to the overall public health interest. However, the new law falls short of resolving some of the most serious paradoxes of pesticide regulation, which will continue to yield counterproductive effects. This section examines major paradoxes associated with the new FQPA regime. The paradoxes arise either from the inability to acknowledge the full benefits of pesticide usage or from continued inconsistency in regulatory standards.

1. The Farmworker Paradox

The "reasonable certainty of no harm" standard applies to food pesticide residues, but does not consider risks to farmworkers. These risks remain regulated under a looser "unreasonable risk" standard in FIFRA. The differential decision standards parallel the source of the Delaney Paradox and create similar perversities. Under the FQPA and FIFRA, regulation of pesticides will transfer risk from consumers to farmworkers and will probably increase overall pesticide danger in the process.

The risk transference to workers is common throughout environmental law. The choice to regulate a particular environmental problem may not eliminate a risk but instead transfer that risk to another group of people. There is an innate tendency of regulation to shift risks from more influential groups to those with less political sway.[161] Quite often, reducing public risk

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159. For a review of conservative assumptions in risk assessment, *see infra* notes 278-80 and accompanying text.

160. J. Robert Tomerlin & Reto Engler, *Estimation of Dietary Exposure to Pesticides Using the Dietary Risk Evaluation System*, *in* PESTICIDE RESIDUES AND FOOD SAFETY, *supra* note 48, at 192, 197.

161. *See* Frank B. Cross, *The Subtle Vices of Environmental Values*, DUKE L. & POL'Y F. (forthcoming).

often means creating occupational risk.[162] Thus, regulating pesticide residues on food can increase pesticide risks to farmworkers, a particularly disempowered group.[163] The differential standards in the law make such a transfer legally acceptable, even if greater overall risk results. The history of pesticide regulation, notably the developments surrounding regulation of DDT and ethylene dibromicide demonstrates the reality of this concern.[164]

The ban on DDT transferred material risks to innocent farmworkers. DDT was of particular concern to environmentalists, inspired by Rachel Carson, in part because it was environmentally long-lived.[165] DDT was banned, and its applications were replaced by a series of pesticides from a group known as organophosphates.[166] Unfortunately, the organophosphates were much more acutely toxic, so the use of these substances "caused incidents of serious poisoning among unsuspecting workers and farmers who had been accustomed to handling the relatively nontoxic DDT."[167] The failure to consider the risk of replacement to farmworkers is estimated to have "cost several hundred lives."[168] The President of the NAS announced that the "predicted death or blinding by parathion of dozens of Americans last summer must rest on the consciences of every car owner whose bumper sticker urged a total ban on DDT."[169] The undue public and regulatory cathexis on a tiny risk of pesticide residues[170] resulted in a transfer of greater

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162. *See* Chris Whipple, *Nonpessimistic Risk Assessment and de Minimis Risk as Risk Management Tools*, *in* THE RISK ASSESSMENT OF ENVIRONMENTAL HAZARDS, *supra* note 67, at 1105, 1109.

163. *See* George M. Gray & John D. Graham, *Regulating Pesticies*, *in* RISK VERSUS RISK: TRADEOFFS IN PROTECTING HEALTH AND THE ENVIRONMENT 173, 189 (John D. Graham & Jonathan Baert Weiner eds., 1995) (observing that "the beneficiaries of reduced residue and persistence—consumers and wildlife—may be enjoying the benefits of a risk transfer to farm workers" who "may be particularly vulnerable when they are migrant, low-income, minority workers who lack a political voice or the English skills to read labels").

164. *See* Donald T. Hornstein, *Paradigms, Process, and Politics: Risk and Regulatory Design*, *in* WORST THINGS FIRST? THE DEBATE OVER RISK-BASED NATIONAL ENVIRONMENTAL PRIORITIES 147, 160 n.7 (Adam M. Finkel & Dominic Golding eds., 1994)(observing that until recently "EPA's risk assessments of pesticides focused predominantly on carcinogenicity among consumers due to residues and all but ignored the workplace exposure to pesticides among the nation's two million hired farmworkers).

165. *See* Frank B. Cross, *Paradoxical Perils of the Precautionary Principle*, 53 WASH. & LEE L. REV. 851, 870 (1996).

166. *See id.*

167. Gray & Graham, *supra* note 163, at 174 (citing M.A. OTTOBONI, THE DOSE MAKES THE POISON: A PLAIN-LANGUAGE GUIDE TO TOXICOLOGY (2d ed. 1991)).

168. AARON WILDAVSKY, BUT IS IT TRUE? 80 (1995).

169. RICHARD L. STROUP & JOHN C. GOODMAN, NAT'L CTR. FOR POL'Y ANALYSIS, MAKING THE WORLD LESS SAFE: THE UNHEALTHY TREND IN HEALTH, SAFETY, AND ENVIRONMENTAL REGULATION 4 (1989) (quoting Dr. Philip handler).

170. While DDT was prohibited in part out of concerns of carcinogenicity, the best research suggests that this concern was unwarranted. *See* research cited in Cross, *supra* note 165, at 890 n.203.

risks to a less politically-prominent group, migrant farmworkers.

Ethylene dibromide ("EDB") cancellation likewise transferred and increased risks from pesticides. EDB was a fumigant employed to control extremely hazardous molds, such as aflatoxin.[171] The need for treatment was such that producers employed a substitute fumigant, but this alternative presented greater risks to workers.[172]

Traditionally, government has blithely ignored the consequences of risk transference to farmworkers. In practice, "risks to applicators and consumers are predicated on the assumption that no other active ingredient will be substituted for one banned in a particular use."[173] Of course, assuming does not make it so. In reality, "such substitutions are the rule rather than the exception[.]"[174]

Nothing in the FQPA eliminates the risk from substitutes, and the projected added regulation could readily exacerbate the risk. Even when the risks from substitutes to farmworkers are acknowledged in regulation, risks will shift and increase. Given the legal language and political realities, "the EPA tolerates higher risks for exposures to pesticides incurred by workers who manufacture, distribute, or apply pesticides than they do for the general population."[175]

Of course, an across-the-board reduction in all pesticide usage would have some health benefit for farmworkers as well. But the effect of FQPA will not be a complete reduction, but will involve a shift in applications and categories of pesticides. As experience with DDT and EDB show, in the past a shift in pesticide categories has been toward greater risks for applicators of pesticides, even while risks to consumers have been reduced. Therefore, overall reduction in the total quantity of pesticides used could increase the cumulative risk from pesticides.

For example, the overall risk would increase if the law caused a shift to so-called natural pesticides, which are used even by organic farmers. Some natural pesticides are notoriously unsafe. George Gray of the Harvard Center for Risk Analysis reports that sulfur, a widely used pesticide on organic farms, caused more occupational illnesses in California during the period

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171. *See* Gray & Graham, *supra* note 163, at 186. The authors proceed to observe that consumers may be enjoying "the benefits of a risk transfer to farm workers." *Id.* at 189.

172. *See* STEPHEN BREYER, BREAKING THE VICIOUS CIRCLE: TOWARD EFFECTIVE RISK REGULATION 17 (1993) (alternative to EDB was "more dangerous fumigants"); William R. Havender, *EDB and the Marigold Option*, REGULATION, Jan.-Feb. 1984, at 13, 16 (describing risks to farmworkers from substitute product).

173. Cropper et al., *supra* note 16, at 194.

174. *Id.*

175. JOSEPH V. RODRICKS, CALCULATED RISKS 213 (1992).

1984-90 than any man-made pesticide.[176] Nicotine is a natural substance, once used as a pesticide, but abandoned as unsafe.[177] Arsenic is likewise "natural," but relatively hazardous.

2. The Cumulative Effects Paradox

The FQPA's cumulation of effects from pesticides with common toxicity mechanisms actually tends to increase risks. The increase again results from the existence of different legal standards for substances that operate through a common mechanism and those that do not. By holding the former category of pesticides to a functionally higher standard, risk will be transferred to the latter group, and will probably increase overall.

Consider the following hypothetical. Suppose that three pesticides (A_1, A_2, and A_3) share a common mechanism of action requiring cumulation of effects under the FQPA. Prevailing risk assessment places the carcinogenic hazard from each at 0.4 in one million. Cumulating the risks results in a total hazard of 1.2 in one million, exceeding the statutory negligible one-in-one million risk standard. This means that one application cannot be approved. Suppose that an unrelated pesticide *B* can substitute for any of the uses of the *A* group and cause an assessed risk of 0.8 in one million for the typical application. Under the FQPA, the agency could not register all three *A* uses but could register two *A* uses and one *B* use, even though this will result in an increase of overall risk from 1.2 to 1.6 in one million. The latter combination is statutorily preferred even though it is more harmful. EPA would therefore compel an increased risk.

While the example is theoretical, it is also highly plausible. Pesticides come in broad groups of similar substances, such as organophosphates. The common mechanism standard must inevitably drive some uses of these groups off the market and replace those uses with pesticides. Conceivably, the substitute could be safer than the group, but there is no reason to expect this result. If the substitute is *sui generis* and shares no common mechanism with other substances, EPA could approve a tolerance of up to about one in one million, as allowed under the Act.

In practice, the common mechanism provision will not decrease pesticide use or decrease risk and it is even likely to increase overall risk from applications. The provision will have the effect of driving manufacturer research and development into unrelated product lines, which will not be

176. *See 1995 Hearings, supra* note 31, at 44-45.

177. W.R. Furtick, *Uncontrolled Pests or Adequate Food?, in* PESTICIDES AND HUMAN WELFARE 3, 12 (D.L. Gunn & J.G.R. Stevens eds., 1976).

combined under the common mechanism provision. Once the "risk cup" is full for a certain category of pesticides, manufacturers will tend to shift to other categories, regardless of relative efficacy or health consequences.

Practically, the government will not dictate which pesticides are used; industry has some control over this choice. Suppose that the "risk cup" is full so that EPA will not register new uses of a given pesticide or category of pesticides. Industry can begin a new use of the pesticide or a similar pesticide by eliminating one of the pre-existing uses. The "manufacturer of the pesticide may voluntarily cancel other existing uses of the pesticide to clear room for the proposed tolerance under the . . . ceiling."[178] Hence, private industry has some control over which uses of pesticides will be allowed.

The possibility of voluntary cancellations adds economic efficiency to the process and prevents valuable applications from being driven off the market by less valuable usages. Voluntary cancellations do not, however, address problems created by the cumulative effects paradox. Voluntary cancellations are driven by the profit maximization of the pesticide manufacturer, not the social welfare of users, consumers or society as a whole. Suppose that the failure to register pesticide A_3 results in use of pesticide B and thereby increases overall risk from the substances. If A_3 and B are produced by the same company, there may be no incentive for voluntary cancellation, as the company simply profits from greater sales of B. Even if the pesticides have different manufacturers, there will be no voluntary cancellation unless A_3 happens to be a more profitable application than the usages already registered. One cannot rely on the good will of manufacturers to choose the mix of pesticides that provides the highest health protection or benefit to agricultural producers.[179]

3. *The Benefits Paradox*

Pesticides, which cost money, are only used by farmers for some sufficient reason. Typically the chemicals are applied to grow more produce or harvest a given amount less expensively. Debates over pesticide regulation often feature drawn-out debates over whether the health risks of pesticides should be balanced against the economic benefits of usage. The economic benefits of pesticide applications can be considerable. Direct benefits to

178. Winter, *supra* note 115, at 141.

179. This article contends that pesticide use has considerable positive public health externalities and presents relatively low risk. This fortuity is the consequence of the market's invisible hand and certain scientific facts, however, not the social conscience of pesticide manufacturers. I certainly do not propose that the good will of these manufacturers will itself guarantee public safety.

farmers are estimated to be $3 to $5 for each $1 invested in the use of pesticides.[180] However, pesticide critics commonly contend that no amount of money can justify risks to life.[181] This environmentalist position against balancing lives and money does not confront the true paradox because pesticide use often has *health* benefits. A reduction in pesticides to reduce health risks may cause net health harm to the public through the loss of these benefits. This section explores the very real health harms that will result from reduced pesticide usage.

The practical benefits of pesticides should be apparent; agricultural producers would not spend money on the substances if they were not beneficial. Yet some maintain that pesticides are unnecessary, as if producers were voluntarily throwing their money away. Some also maintain that pesticides will become unproductive as pests develop resistance.[182] Not only are such assertions contradicted by yield data,[183] but the position is also illogical: producers will not spend on useless substances.[184]

The nature of pest resistance is often overstated. Although "some pest species have developed a vexing resistance to man's chemicals, most have not; the resistant portion may be far less than one percent."[185] Moreover, resistance typically does not render a chemical useless in all applications; resistance may be localized and controlled by modified pesticide application techniques.[186] Additionally, pests that develop resistance typically contain other deficiencies in fitness or reproductive behavior that render them amenable to other control methods, such as a "less potent but more specific pesticide than the one to which resistance has developed."[187] Agricultural

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180. *See* David Pimentel et al., *Environmental and Economic Effects of Reducing Pesticide Use*, 41 BIOSCIENCE, 402, 402 (1991) (citations omitted)).

181. This argument was made by a number of environmental groups testifying on an earlier version of the FQPA. *See generally 1995 Hearings*, *supra* note 31.

182. *See also id.* at 81-82 (statement of Erik Olson of Natural Resources Defense Council) (suggesting that pesticide benefits were limited by development of resistant pests).

183. *See infra* note 194 and accompanying text.

184. Nor will manufacturers produce pesticides that lack a market due to resistance. *See* George P. Georghiou, *The Magnitude of the Resistance Problem*, *in* COMMITTEE ON STRATEGIES FOR THE MANAGEMENT OF PESTICIDE RESISTANT PEST POPULATIONS, NATIONAL RESEARCH COUNCIL, PESTICIDE RESISTANCE: STRATEGIES AND TACTICS FOR MANAGEMENT 14, 35 (1986) [hereinafter PESTICIDE RESISTANCE] (slowing in development and introduction of new pesticides to anticipated resistance problems).

185. GREGG EASTERBROOK, A MOMENT ON THE EARTH 79 (1995). This small number does not demean the prospect of resistance, which is a problem for certain pests of certain crops throughout the world. *See* Georghiou, *supra* note 184, at 14-44 (discussing development of pesticide resistance). The relative infrequency of serious pesticide resistance does demonstrate that the chemicals have continuing value to agriculture.

186. LEONARD T. FLYNN, PESTICIDES: HELPFUL OR HARMFUL? 28-29 (1989).

187. *Id.* at 29.

researchers have devoted considerable energy toward effectively managing the development of resistance and perpetuating the effectiveness of pesticides.[188] Furthermore, the presence of resistance also increases the need for registration of new pesticide products.[189]

The case for the practical benefits of pesticides does not rest merely upon theoretical models. Empirical experience with regulation demonstrates the effect. When EPA canceled one pesticide under the Delaney Clause, mint yields promptly declined by 13%.[190] Another cancellation resulted in a loss of 50 million pounds from North Carolina's apple crop.[191] In many past cases, losses from pesticide cancellation were small because substitute pesticides were available.[192] However, the FQPA is not expected merely to shift pesticide uses, but to reduce overall use considerably.

Even when alternative pesticides were available, producers suffered economic losses or added costs.[193] As a general rule "crop yields from organic farms are typically half or less those of high-yield mainstream farmers."[194] The most comprehensive study available concluded that a 50% reduction in pesticide use would increase the per unit production costs of fruits and vegetables from 20 to 40%.[195] Research suggests that each 1% reduction in crop yield "results in a corresponding 4.5% increase in the farm

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188. *See, e.g.*, Executive Summary, *in* PESTICIDE RESISTANCE, *supra* note 184, at 1, 1-2 (describing effectiveness of methods of pesticide resistance management); *see also* P.K. Leonard, *IRAC Fruit Crops Working Group Spider Mite Resistance Management Strategy*, *in* RESISTANCE '91: ACHIEVEMENTS AND DEVELOPMENTS IN COMBATING PESTICIDE RESISTANCE 41 (Ian Denholm et al. eds., 1992) [hereinafter RESISTANCE '91]; Louise R. Cooke, *Resistance to Phenylamide Fungicides: Strategies and Their Evaluation*, *in* RESISTANCE '91, *supra*, at 100; Bernard C. Smale, *The International Organization for Resistant Pest Management (IRPM): A Fresh Collaborative Approach*, *in* RESISTANCE '91, supra, at 112.

189. *See* Bruce D. Hammock & David M. Soderlund, *Chemical Strategies for Resistance Management*, *in* PESTICIDE RESISTANCE, *supra* note 184, at 111, 113:

> The effective management of pesticide resistance, however, involves not only the judicious use of existing compounds but also the discovery and development of new chemical control agents. No management strategy can prolong the useful life of pesticides indefinitely. New chemical tools will be needed, particularly those that exploit new biochemical targets. Thus, rather than removing us from a "pesticide treadmill," IPM and resistance management will only slow the treadmill. . . .

190. *See 1995 Hearings*, *supra* note 31, at 38-39 (statement of Leonard Gianessi of National Center for Food and Agricultural Policy).

191. *See id.*

192. *See* Cropper et al., *supra* note 16, at 182 (reporting that in "35 percent of all cases, losses [from pesticide cancellation] are negligible because of the availability of substitute pesticides").

193. *See* 1995 *Hearings*, *supra* note 31, at 38 (suggesting that cancellations from the application of the Delaney clause would increase costs by $40 million for cotton growers, $22 million for grape growers, and $5 million for citrus growers).

194. Dennis Avery, *Saving the Planet with Pesticides*, *in* THE TRUE STATE OF THE PLANET 49, 70 (Ron Bailey ed., 1995) (citing P.J. Michaels & D.E. Stooksbury, *Global Warming: A Reducing Threat?* 73 BULL. AM. METEOROLOGICAL SOC'Y 1563 (1992)).

195. *See 1995 Hearings*, *supra* note 31, at 173 (statement of Professor C. Robert Taylor).

price of goods."[196] Not all of this increase will be passed on to consumers, but a significant portion eventually will have to be paid by purchasers of fruits and vegetables.[197] "[E]conomic studies indicate that restricting pesticides to reduce carcinogenic residues would likely raise the prices of foods significantly."[198]

The simplest economic graph informs us that an increase in a product's price will result in some reduction in its consumption. The relationship between these variables is expressed as price elasticity. For domestic use, a 10% increase in the price of fruits and vegetables would cause a reduction of consumption between 2.2 and 6.1%, depending on the particular product category.[199] A 50% reduction in pesticide use would promptly reduce domestic consumption of fruits and vegetables by 4 to 7%.[200] The consumption drop-off would be even greater if added restrictions were placed on imports produced with pesticides.[201] Much of this reduced consumption would be felt by the poor.[202]

Reduced consumption of fruits and vegetables is not a good thing for the overall public health. Consumption of such produce "is associated with a lowered risk of degenerative diseases such as cancer, cardiovascular disease, cataracts, and brain and immune dysfunction."[203] As it happens, pesticides are essential for those fruits and vegetables that are most beneficial to health.[204] In general, "increasing consumption of fruits and vegetables can

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196. Pimentel et al., *supra* note 180, at 406.

197. *See 1995 Hearings, supra* note 31, at 174-75 (statement of Professor C. Robert Taylor) (observing that in the intermediate term about half of the increase will be passed on, while in the long run, nearly all will be passed forward to consumers); Cropper et al., *supra* note 16, at 181 (suggesting that yield losses or cost increases will lead to consumer price increases).

198. Gray & Graham, *supra* note 163, at 190.

199. *See 1995 Hearings, supra* note 31, at 174 (statement of Professor C. Robert Taylor).

200. *See id.* at 175.

201. *See id.*

202. *See* HARVARD CENTER FOR RISK ANALYSIS, *supra* note 149, at 2 (observing that for "poor families and households on fixed incomes, higher food prices increase the risk of malnutrition and its associated illnesses").

203. Bruce N. Ames & Lois Swirsky Gold, *The Causes and Prevention of Cancer, in* THE TRUE STATE OF THE PLANET, *supra* note 194, at 144, 150.

204. *See* REGULATING PESTICIDES IN FOOD, *supra* note 42, at 49 (observing that "[v]irtually all perishable fresh fruits and vegetables . . . depend heavily on pesticides. Some are treated a dozen or more times each year with six or more different active ingredients"); CARCINOGENS AND ANTICARCINOGENS IN THE HUMAN DIET, *supra* note 129, at 246 (pesticides most common in fresh fruits and vegetables).

A public interest organization called the Environmental Working Group has recently counseled against eating such products as strawberries, bell peppers, spinach, cherries, peaches, cantaloupe, celery, and other vegetables, due to pesticide residues. *See* ENVIRONMENTAL WORKING GROUP, A SHOPPER'S GUIDE TO PESTICIDES IN PRODUCE 3. Yet consumers who follow the publication's advice will probably harm their health, losing substantial health benefits in exchange for minuscule risks posed by the pesticides. *See* Cross, *supra* note 165, at 887-89.

help reduce the risk of some cancers, with benefits far outweighing possible cancer risks from the pesticides."[205]

The health of consumers will suffer if fruits and vegetables become unavailable. While produce will probably still be available, reductions in pesticides may increase the costs of fruits and vegetables markedly. Philip Abelson, the president of the American Academy for the Advancement of Science, warns:

> The public has become increasingly aware that a diet that includes four or five fruits or vegetables per day substantially reduces the incidence of many types of cancers. At present, supplies of these foods are abundant and relatively inexpensive. But continuation of trends in the cancellation (banning) of fungicides could lead to food scarcities.[206]

Abelson states that if synthetic fungicides were wholly unavailable, experts predict apple production would drop 40%, and losses for grapes, peaches, and strawberries would be 33%, 49%, and 38%, respectively.[207]

Given the extremely low risk from pesticide residues, a reduction in the amount of health-giving products will surely cause greater health harms. Scientists from the National Cancer Institute warn that if pesticides were outlawed, "prices would rise and demand could fall for foods that have been shown to prevent cancer, such as broccoli and carrots."[208] Even if availabily and cost were unaffected, pesticide reductions could still have further health disadvantages. For example, "fruits that are inadequately protected against pests have been shown to have lower nutritional value, including less Vitamin C in apples, than fruits protected with pesticides."[209]

The adverse effects of pesticide regulation on fruit and vegetable availability will be felt especially by poor individuals.[210] Organic foods grown without pesticides are distinctly more expensive. Such foods will be

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205. 135 CONG. REC. 12, 821 (1989) (remarks of Rep. Hamilton).

206. Philip H. Abelson, *Adequate Supplies of Fruits and Vegetables*, 266 SCIENCE 1303 (1994).

207. *See id.*

208. *See* Sheehy, *supra* note 25, at 275 n.191 (quoting Keith Schneider, *Cancer Controversy: An Appeals Court Ruling Would Ban 35 Pesticides*, N.Y. TIMES, July 12, 1992, at D2). *See also* Gray & Graham, *supra* note 163, at 179 (observing that "[i]f substitute pesticides are less effective or more expensive than the banned products, crop yields may be constrained and the prices of critical foods, especially fruits and vegetables, may rise significantly").

209. *1995 Hearings, supra* note 31, at 44 (statement of George Gray, Deputy Director of the Harvard Center for Risk Analysis).

210. *See* Gray & Graham, *supra* note 163, at 190 (observing that if "the prices of fruits and vegetables rose, the effect would be very regressive, with the largest impact on the poorest segment of the population").

consumed less, and consumers will lose the considerable health benefits offered generally by fruits and vegetables. Ames claims that "[m]aking these foods more expensive by reducing synthetic pesticide use is likely to increase cancer."[211] The consequences of pesticide regulation are not merely counterproductive in terms of public health; they are distributionally perverse. The health burdens of pesticide reductions will be borne centrally by the disadvantaged. "Higher food prices and less nutritional food selection at the market would disproportionately affect low-income consumers, who may spend as much as sixty percent of their income on food."[212]

The health risks of the FQPA should not be exaggerated. Some evidence suggests that pesticide use could be reduced significantly without serious adverse consequences to agriculture. Indeed, one survey contends that total U.S. pesticide usage might be reduced as much as 35 to 50%, at a cost of about $1 billion.[213] Of course, $1 billion is not a trivial increase in cost, and not everyone agrees with the conclusions of this study.[214] Moreover, the feasible reduction in use proposed by these authors comes not from regulatory action, but rather from improved application and monitoring equipment.[215]

The problem with the FQPA and other government efforts to reduce pesticide usage is the crudeness of the regulatory tool. The mandatory reductions in pesticide use will not parallel the safe and effective reductions possible. Even if overall use could be reduced by one-half, this is not necessarily the same half to be reduced through regulation. The authors who proposed the feasibility of the dramatic usage restriction favored improved efficiency in pesticide applications, not bans on such applications.

No uniform national regulations can rationally reduce the risks from pesticides without compromising the health benefits of pesticides. The NAS discussed some of the factors associated with the wise usage of pesticides:

> In any growing season, economic factors can alter which pesticides are used on a given crop in a given area. The price of the crop might be up or down, affecting how much growers are willing to spend for a certain amount of pest control. Weather and soil conditions can

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211. *See* Ames & Gold, *supra* note 203, at 159. *See also* Julie Corliss, *The Delaney Clause: Too Much of a Good Thing?*, 85 J. NAT'L CANCER INST. 600, 601 (1993) (citing Professor Manfred Kroger to the effect that "the benefits of pesticides, which help ensure widely available and affordable fruits and vegetables, justify their use").

212. Curme, *supra* note 30, at 643.

213. *See* Pimental et al., *supra* note 180.

214. *See* Pimental et al., *supra* note 180, at 404.

215. *See* Pimental et al., *supra* note 180, at 404

> preclude or command certain treatments. The presence or absence of a given pest affects pesticide use. The emergence of pest resistance to previously applied pesticides can lead to rapid shifts in pesticide use patterns.[216]

Pesticide applications vary wildly, depending upon "unusual weather and pest problems."[217] Setting a given tolerance for specific crop applications cannot account for these fluctuating needs. Consequently, a regulatory regime for pesticide residues cannot produce the desirable reduction in use of the product. Agricultural producers have an incentive to respond to dynamic conditions and reduce unnecessary use of inputs such as pesticides when possible,[218] so that an information program or incentives for more efficient application will be safer and more effective than regulation.

Many advocates of stricter pesticide regulation embrace Integrated Pest Management ("IPM").[219] IPM is a rather amorphous combination of biological controls intended to control pests in the absence of synthetic pesticides. It may include the introduction of natural predators, such as a beetle that eats the target insect. IPM may also include the introduction of parasites, diseases, or naturally resistant characteristics in plants. The following section will address the possibility that some of these natural pest controls may prove more hazardous to human health than synthetic ones. Regardless of that risk, the potential value of IPM does not delegitimate the use of pesticides.

The effectiveness of IPM is far from perfect, a fact that should be obvious from prevailing use of synthetic pesticides. IPM is already statutorily encouraged,[220] and if it had the utopian effectiveness promised by advocates at reasonable cost, agricultural producers would be falling over themselves to adopt the practice.[221] While chemical manufacturers of pesticides may have

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216. REGULATING PESTICIDES IN FOOD, *supra* note 42, at 48-49.

217. *See* Gianessi, *supra* note 120, at 26. The author notes that in an ordinary year, about 1% of Illinois soybeans are treated with insecticides, but the drought of 1988 required that 40% of the crop be so treated. *Id.*

218. *See* J.L. Adams, *The Farmer's Stake in Food Safety*, *in* PESTICIDE RESIDUES AND FOOD SAFETY, *supra* note 48, at 47. Adams suggests that for economic reasons farmers have no interest in applying even "one unnecessary drop of agrichemical" to crops. *Id.* Adams further observes that agrichemical use is actually declining. *See id.*

219. *See, e.g., Hearings on Food Safety Issues, Before House Subcomm. on Dep't Operations, Research and Foreign Agriculture*, 102d Cong., 2d Sess. 805 (1992) (statement of Mike Wallace, co-chairman of National Coalition on Integrated Pest Management) (reporting that expanded IPM could reduce pesticide use by half).

220. *See* 7 U.S.C. § 136w-3 (1994).

221. *See* Allen L. Jennings, *Some Economic and Social Aspects of Pesticide Use*, in PESTICIDE RESIDUES AND FOOD SAFETY, *supra* note 48, at 32, 36 (observing that farmers use pesticides because they offer some economic or social advantage over the alternatives). In trying to discover why

an incentive to undermine IPM, that incentive does not extend to the producers who actually decide what pest method to apply. The continued use of synthetic pesticides is testimony to their efficiency and effectiveness compared to IPM.[222]

IPM has had some successes and is currently employed,[223] but the practice has too many failures to eliminate the need for synthetic pesticides.[224] Very few pests are effectively managed by using IPM alone without pesticides.[225] EPA's ban on the controversial pesticide Alar undermined an IPM program for apples and incidentally caused a greater overall use of pesticides.[226] Moreover, IPM involves its own set of environmental risks, as the introduction of non-native species can disrupt ecosystems in unforeseen ways.[227]

Indeed, while IPM will often reduce total pesticide usage, many IPM

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bioherbicides have not caught on, a commentator noted that no one had yet produced "a commercial product effective against a major weed in one of the world's main crops." M.P. Greaves & M.D. MacQueen, *Bioherbicides: Their Role in Tomorrow's Agriculture*, *in* RESISTANCE '91, *supra* note 188, at 295, 299. Moreover, bioherbicides may be more difficult to apply effectively. *See id.* at 301.

222. *See* FLYNN, *supra* note 186, at 41 (observing "[m]odern agriculture does not neglect nonchemical methods when they are appropriate because today's farmers cannot afford to waste resources on unnecessary chemicals when less costly nonchemical methods will suffice"); Adams, *supra* note 218, at 48 (suggesting that "producers are always looking for ways to cut back if there's a way to get the same results with lower application rates").

223. *See* FLYNN, *supra* note 186, at 38-39 (describing how introduction of natural insects and diseases are used to control citrus fruit diseases, Japanese beetle grubs, and other threats to crops).

224. *See* Louis A. Falcon, *Development and Use of Microbial Insecticides*, *in* BIOLOGICAL CONTROL IN AGRICULTURAL IPM SYSTEMS 229, 236 (Marjorie A. Hoy & Donald C. Herzog eds., 1985) (declaring that relative ineffectiveness of microbial insecticides indicates that they may have "no future . . . as commercial products"); J.D. Podgwaite, *Strategies for Field Use of Baculoviruses*, *in* VIRAL INSECTICIDES FOR BIOLOGICAL CONTROL 775, 776 (Karl Maramorosch & K.E. Sherman eds., 1985) (reporting that inconsistent field results with biological control products prevent use as substitute for pesticides); J.P. Hudson, *Fruit Crops: A Rather Special Case*, *in* PESTICIDES AND HUMAN WELFARE, *supra* note 177, at 81, 89 (observing that IPM "has had rather little success up to now with the major pests of temperate fruits"); Jürgen Kranz, *Vegetables*, *in* PESTICIDES AND HUMAN WELFARE, *supra* note 177, at 93, 101 (reporting that there "is little scope for immediate biological control in short-lived vegetables").

225. *See* KEITH C. BARRONS, ARE PESTICIDES REALLY NECESSARY? 47 (1981); D.L. Gunn, *Alternatives to Chemical Pesticides*, *in* PESTICIDES AND HUMAN WELFARE, *supra* note 177, at 241, 249-50 (reporting how efforts to sterilize pest species have generally failed) and 251-52 (indicating use of hormones and pheromones to control pests has little promise).

226. This story is explained in Gianessi, *supra* note 120, at 29.

227. *See, e.g.*, Gunn, *supra* note 225, at 244 (pointing out that the danger of "introducing an exotic plant parasite is that it will not confine itself to the target species of plant but will attack crop species and become a pest itself"); *id.* at 245 (observing that biological microbes used to attack insect pests may also threaten human health). Mongooses were introduced to the island of Jamaica in order to control rat populations, but the rats managed to escape the mongooses, which then became a pest attacking poultry in the island. *See id.* at 247. Australia is releasing genetically engineered viruses in an attempt to control imported red foxes and rabbits. *See* Virginia Morell, *Australian Pest Control by Virus Causes Concern*, 261 SCIENCE 683, 683 (1993).

programs require the use of pesticides.[228] IPM usage is often only possible if farmers have "a wide variety of pesticides to choose from."[229] "[Very few] pests can be effectively managed by integrated genetic, biological and cultivation methods alone."[230] IPM may be far more effective thanks to "synergistic action" when combined with conventional pesticides.[231] In some circumstances, "IPM programs have resulted in increased pesticide use."[232] Those regulations "which eliminate appropriate uses of pesticides may be counterproductive to the implementation of IPM."[233] Moreover, the most effective biological IPM controls are likely to depend upon advances in biotechnology,[234] which itself is challenged by many environmentalists.[235]

To the extent that IPM is beneficial, as it may often be, pesticide regulation may undermine the practices. Moreover, pesticide regulation is not necessary to encourage effective IPM. When its implementation reduces pesticide usage, it will also mean "increase[d] profits for the producer[.]"[236] Encouraging IPM may require additional research and education, but additional pesticide regulation may only frustrate IPM's development.[237] The effectiveness of IPM systems will vary by weather, soil, geography, crop and other factors. A heavy-handed uniform federal regulatory system cannot accommodate such variations as well as the judgments of local producers.

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228. *See* Curme, *supra* note 30, at 625.

229. *See* Patrick W. Weddle, *Pesticide-Free Tree Fruit Crops: Can We Meet Consumer Demands?*, *in* PESTICIDE RESIDUES AND FOOD SAFETY, *supra* note 48, at 58, 62 (observing that IPM "can be hampered by excessive constraints to pesticide use").

230. *See* FLYNN, *supra* note 186, at 41; *see also* Gianessi, *supra* note 120, at 27 (reporting that "pesticides have an important role in most successful IPM programs"). When pesticide use is abandoned, "the IPM programs are completely disrupted[.]" *Id.*

231. *See* Greaves & MacQueen, *supra* note 221, at 302.

232. Gianessi, *supra* note 120, at 27-28 (referencing IPM program for peanuts in Alabama as example).

233. Weddle, *supra* note 229, at 63.

234. *See* Greaves & MacQueen, *supra* note 221, at 302-03 (absent biotechnological manipulation, the discernment of effective controls is "very hit-and-miss"). *See also* Marjorie A. Hoy, *Genetic Engineering of Predators and Parasitoids for Pesticide Resistance*, *in* RESISTANCE '91, *supra* note 188, at 307.

235. *See, e.g.*, David J. Earp, *The Regulation of Genetically Engineered Plants: Is Peter Rabbit Safe in Mr. McGregor's Transgenic Vegetable Patch?*, 24 ENVTL. L. 1633 (1994) (reviewing the controversy and calling for additional federal regulatory authority over agricultural biotechnology, including development of natural pesticides).

236. K. Jack Haugrud, *Agriculture*, *in* SUSTAINABLE ENVIRONMENTAL LAW § 8.5 (Celia Campbell-Mohn et al. eds., 1993).

237. *See* Weddle, *supra* note 229, at 65 (observing that most effective approach to IPM implementation would be providing farmers with additional information and experience on methods).

4. The Natural Risk Paradox

Critics of chemical pesticides typically have a Panglossian view of nature. They presume that "organic" foods, produced without manmade chemicals, are somehow safe to eat. Yet this presumption is dubious. Natural presticides "are apparently present in all plants and may make up 5-10% of a plant's dry weight."[238] Professor Bruce Ames of California estimates that "99.99 percent of the pesticides we eat are naturally present in plants to ward off insects and other predators."[239]

Naturalness does not render these substances benign, as many of these natural pesticides are apparently carcinogenic, at least as indicated by the sort of rat or other animal bioassays employed to test synthetic chemicals for carcinogenic potential.[240] Furthermore, the quantity of natural carcinogens is much greater, and natural substances on average appear to have a greater carcinogenic potency than synthetic carcinogens.[241] Indeed, "few synthetic chemicals equal the potency and human toxicity of naturally occurring products."[242] Ames suggests that "[t]here are more rodent carcinogens in a single cup of coffee than potentially carcinogenic [synthetic] pesticide residues in the average American diet in a year."[243]

Even those who acknowledge the presence of natural carcinogens may argue that we might as well reduce exposure to synthetic carcinogens. Yet this logic is inapplicable if regulation of synthetics merely increases exposure to natural pesticides, particularly when some forms of IPM may be far more hazardous than synthetic chemical pesticides.[244] When researchers bred a pest-free potato, it was "so full of natural pesticides that it was acutely poisonous to humans."[245] Other species seek to protect their seeds from pests by releasing hydrogen cyanide when damaged, but this mechanism is too toxic to rely upon.[246]

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238. Fred R. Shank et al., *Evolving Food Safety*, *in* PESTICIDE RESIDUES AND FOOD SAFETY, *supra* note 48, at 297, 299.

239. Ames & Gold, *supra* note 203, at 143; *see also* Bruce N. Ames et al., *Ranking Possible Carcinogenic Hazards*, 236 SCIENCE 271 (1987).

240. See Ames et al., *supra* note 239; *see also* Carl K. Winter, *Toxins of Plant Origin*, *in* CHEMICALS IN THE HUMAN FOOD CHAIN, *supra* note 7, at 221 (reviewing evidence of harmful effects of a variety of natural substances).

241. *See* CARCINOGENS AND ANTICARCINOGENS IN THE HUMAN DIET, *supra* note 129, at 291.

242. MELVIN A. BENARDE, OUR PRECARIOUS HABITAT 71 (1989).

243. Ames & Gold, *supra* note 203, at 159.

244. *See* Avery, *supra* note 194, at 69.

245. Cross, *supra* note 165, at 873. Similar efforts with celery caused a substantial increase in a naturally occurring carcinogen. *See id.*

246. *See* John A. Pickett, *Potential of Novel Chemical Approaches for Overcoming Insecticide Resistance*, *in* RESISTANCE '91, *supra* note 188, at 354, 357.

In addition to natural pesticides engineered by humans, nature itself may produce higher levels of hazardous substances. When fungicides are not applied, "plants in self-defense create phytoalexins, some of which are toxic to humans and induce carcinomas in rodents."[247] Now-Justice Breyer, previously a judge on the U.S. Court of Appeals for the First Circuit, observed that regulators must consider behavior such as "when a farmer, deprived of his small-cancer-risk artificial pesticide, grows a new, hardier crop variety that contains more 'natural pesticides' which may be equally or more carcinogenic[.]"[248] On balance, "there would be adverse indirect food safety consequences of severely restricting or banning pesticide use in terms of increased contamination by fungal products and by phytoalexins created in self-defense by plants."[249] The levels of natural carcinogens in food "may increase dramatically in plants damaged by insects or fungi[,]"[250] and the resultant natural carcinogens may prove much more hazardous than the synthetic carcinogens regulated. Dr. Bruce Ames has flatly declared that "pesticides lower the cancer rate."[251]

Moreover, pesticides are often applied in order to combat natural carcinogens directly, particularly mycotoxins. Mycotoxins are "highly poisonous compounds of small molecular weight produced by molds or fungi."[252] One well-known example of a mycotoxin is the poisonous mushroom. Mycotoxins are replete throughout the food supply, found in dozens of foods.[253] One such mycotoxin is aflatoxin, a notoriously carcinogenic substance contaminating grains. Pesticides are often applied in order to combat human exposure to mycotoxins.

Past regulation of pesticides has increased the overall risk from exposure to mycotoxins. For example, the notorious pesticide Alar was used to control fruit drop of apples. When treated apples remain on the trees, the fruit is less susceptible to hazardous molds.[254] The fungicide EDB, cancelled due to a largely unwarranted cancer scare,[255] was at the time "the safest known way to

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247. Abelson, *supra* note 206.

248. BREYER, *supra* note 172, at 23.

249. Taylor, *supra* note 141, at 18.

250. Shank et al., *supra* note 238, at 299-300.

251. Jane E. Brody, *Strong Views on Origins of Cancer*, N.Y. TIMES, July 5, 1994, at C1 (quoting Ames).

252. Dennis P.H. Hsieh & Stefan H.O. Gruenwedel, *Microbial Toxins*, *in* CHEMICALS IN THE HUMAN FOOD CHAIN, *supra* note 7, at 239, 243.

253. *See id.* at 244 tbl. 5-4 (noting in chart the presence of mycotoxins in products from wheat, corn, nuts, apples, beans, and other foods).

254. Bruce N. Ames & Lois Swirsky Gold, *Environmental Pollution and Cancer: Some Misconceptions*, *in* RATIONAL READINGS ON ENVIRONMENTAL CONCERNS 165 (Jay H. Lehr ed. 1992).

255. *See* Havender, *supra* note 172 (discussing how EDB ban was based on overblown fears of consumer carcinogenicity).

combat molds, which produce some of the most potent carcinogens in all of nature."[256] Interestingly, organic apple juice was condemned by a California health department because of high mycotoxin concentrations.[257] Meanwhile, EDB, as noted above,[258] was replaced by substitute pesticides more hazardous to farmworkers. In this case, regulation may have sidestepped the natural risk paradox, but only at the cost of causing the farmworker paradox.

EPA can set standards for natural carcinogens, but such rules are rare and established under a looser standard than that of the FQPA. The contrast is apparent from the EDB ban. "Aflatoxin B_1 is about 1,000 times more potent than EDB, yet it is allowed in foods at levels as high as 20 ppb—nearly ten times higher than the average level of EDB found pre-ban in grain-based food products."[259] This result occurred notwithstanding the fact that EDB was the safest known way to combat molds, some of which contain the natural toxin aflatoxin.[260]

5. The Hormetic Paradox

Federal public health regulation is based on the premise that if exposure to a high level is bad, lower exposures are always safer. Thus, when high exposure levels in scientific testing demonstrate a statistically significant increase in cancer, the government typically assumes that the relationship between exposure and hazard is linear. Consequentially, lower exposure levels always mean less risk, and zero exposure is the safest situation of all.

While these presumptions are well established politically, their scientific basis is less certain. A concept known as hormesis[261] occurs when "a substance presenting a mortality risk at high levels of exposure actually protects against death or disease at low levels of exposure."[262] Lest this seem paradoxical or implausible, remember that vaccination often involves

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256. STROUP & GOODMAN, *supra* note 169, at 4 (1989); *see also* John D. Graham & Jonathan Baert Weiner, *Confronting Risk Tradeoffs*, *in* RISK VERSUS RISK, *supra* note 163, at 13, 13-14 (observing that the EDB ban "may have left on grains and nuts a fungus that promotes aflatoxins more carcinogenic than the fungicide").

257. *See* Weddle, *supra* note 229, at 66.

258. *See supra* notes 171-72 and accompanying text.

259. AMERICAN COUNCIL ON SCIENCE AND HEALTH, DOES NATURE KNOW BEST?: NATURAL CARCINOGENS AND ANTICARCINOGENS IN AMERICA'S FOOD" 31 (1996).

260. Frank B. Cross, *The Public Role in Risk Control*, 24 ENVTL. L. 887, 943 (1994) (reporting safety and effectiveness of EDB); BREYER, *supra* note 172, at 17 (observing that EDB ban could lead farmers "to fumigate their crops less well, leaving more mold residues, which bring with them an increased cancer risk from aflatoxin").

261. Hormesis is subsumed in the study of the biological effects of low level exposure, sometimes called BELLE.

262. Cross, *supra* note 165, at 896.

exposure to a very low level of the disease to be prevented.[263] Exposure to a low level toxicant may provoke the body to increase its production of protective substances.[264] Indeed, the exposure might stimulate DNA repair that exceeded the DNA damage caused by the low exposure.

While hormesis has been generally ignored by federal regulators,[265] a significant number of studies now demonstrate hormetic effects for carcinogens and other hazards.[266] A large study conducted by EPA scientists on numerous chemicals found that exposure to very low doses of chemical carcinogens actually decreased the amount of DNA damage found in test animals, or even improved the animals' health in other ways.[267] Even a leading FDA scientist has declared that information "is accumulating rapidly indicat[ing] that hormesis is operating in a number of areas of toxicology."[268] If hormesis is true for pesticides, efforts to reduce the already low exposure levels may do more harm than good. Hormetic effects have indeed been found for some pesticide exposures.[269] Indeed, in "many chronic studies on

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263. Recognition of hormesis goes back some time. Hippocrates observed that a substance (hellebore) caused cholera-like symptoms at high doses but successfully treated cholera when given in low doses. *See* Harold Boxenbaum et al., *Hormesis, Gompertz Functions, and Risk Assessment*, 19 DRUG METABOLISM REV. 195, 200 (1988).

264. *See* Donald E. Stevenson et al., *Challenges To Low-Dose Linearity In Carcinogenesis From Interactons Among Mechanistic Components As Exemplified By The Concept Of "Invaders" and "Defenders,"* BELLE NEWSL. (U. Mass. Sch. of Pub. Health, Amherst, Ma.), Nov. 1994, at 2-3 (discussing response); R.W. Hart & L.T. Frame, *Toxicological Defense Mechanisms and How They May Affect the Nature of Dose-Response Relationships*, BELLE NEWSL. (U. Mass. Sch. of Pub. Health, Amherst, Ma.), June 1996, at 1 (noting that risk assessment models generally "do not take into account the diverse toxicological defense mechanisms that *enhance* survival in the face of minor environmental adversity"). The nature of this response curve is shown in Werner K. Lutz, *Dose-Response Relationships in Chemical Carcinogenesis: From DNA Adducts to Tumor Incidence*, 283 ADVANCES IN EXPERIMENTAL MED. & BIOLOGY 151 (1991).

265. *See* Sidney Green & Michael L. Dourson, *How Regulatory Agencies View BELLE*, BELLE NEWSL. (U. Mass. Sch. of Pub. Health, Amherst, Ma.), May 1995, at 6 (reporting that "[a]lthough the concept of hormesis has been discussed in the scientific community for a number of years, it was accorded only superficial recognition until recently"). Even today, Green reports, a deficiency of research resources caused FDA to ignore hormesis in its internal planning report. *See id.* at 7. EPA has considered hormesis only for nutrients. *See id.* (statement of Michael L. Dourson, Chief of Systemic Toxicants Assessment Branch of U.S. EPA).

266. *See* J. Michael Davis & David L. Svendsgaard, *U-Shaped Dose-Response Curves: Their Occurrence and Implications for Risk Assessment*, 30 J. TOXICOLOGY & ENVTL. HEALTH 71 (1990) (summarizing twenty-nine studies demonstrating such effects).

267. *See* Kirk T. Kitchin & Janice L. Brown, *Dose-Response Relationship for Rat Liver DNA Damage Caused by 49 Rodent Carcinogens*, 88 TOXICOLOGY 31 (1994).

268. Green & Dourson, *supra* note 265, at 6.

269. There has been limited testing for hormetic effects, but one study of female rats showed that ingestion of low levels of DDT throughout their lives gave the rats a much longer reproductive life span. *See* Alice Ottoboni, *Effect of DDT on the Reproductive Life-Span in the Female Rat*, 22 TOXICOLOGY & APPLIED PHARMACOLOGY 497 (1972). Other research reveals that animals exposed to low levels of dioxin in laboratory tests actually lived longer than control animals. Boxenbaum et al., *supra* note 263, at 208. Hormetic effects have been observed for quite a variety of chemicals, including

pesticides, animals receiving small intakes have been healthier than the control animals."[270]

The notion of hormesis remains scientifically controversial and has not been conclusively "proved." But neither has the linear low dose extrapolation model common to regulation been so proved.[271] If hormesis applies, it will not apply in the same manner to all substances. The evidence for some hormetic effect is certainly reasonably strong in the context of regulatory science. The FQPA's apparent devotion to ever lower pesticide exposures could therefore have the paradoxical effect of lowering public health.

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The above discussion focuses upon the specific paradoxes attendant to the FQPA standards. More general effects might well result from features of the law that are common to environmental regulation. For example, increased time, resources, and attention devoted to the relatively minimal health effects of pesticide residues could be drawn from greater environmental threats.[272] The added costs borne by producers and consumers will themselves cause health harms and additional deaths, as these revenues become unavailable for other health-protective expenditures.[273]

Reduced pesticide usage will also have adverse consequences for the natural environment. While pesticides can themselves harm the environment (*e.g.*, by killing birds or nontarget insects), the natural benefits of pesticide

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some pesticides. *See e.g.*, Edward J. Calabrese et al., *The Occurrence of Chemically Induced Hormesis*, 52 HEALTH PHYSICS 531 (1987).

270. Boxenbaum et al., *supra* note 263, at 209 (quoting D.E. Stevenson).

271. There is a tendency to subject hormesis to a higher standard of proof than required of the conventional linearity model. Such differential standards of proof will be as paradoxical in effect as different legal standards. *See The Second Annual BELLE Conference: A Review*, BELLE NEWSL. (U. Mass. Sch. of Pub. Health, Amherst, Ma.), Aug. 1993, at 3 (discussing comments of John Graham of Harvard School of Public Health to the effect that we must be even-handed in our standards of proof for the non-threshold model as for hormesis); *see also* Cross, *supra* note 165, at 897 (noting that existence of hormesis is not beyond dispute but that double standard in evaluating hormesis will cause more harm to public health). For a variety of reasons, hormesis has been downplayed. *See* Edward J. Calabrese, *Expanding the RfD Concept to Incorporate and Optimize Beneficial Effects While Preventing Toxic Responses from Non-Essential Toxicants*, BELLE NEWSL., October 1995, at 7-8 (noting that disincentives to investigate hormetic effects include ideological bias, the need for larger sample sizes to detect the relatively small effects, toxicological traditions, and biases in federal grants).

272. *See, e.g.*, Tammy O. Tengs et al., *Five Hundred Life-Saving Interventions and Their Cost-Effectiveness*, 15 RISK ANALYSIS 369 (1995) (more efficient allocation of federal regulatory resources could save tens of thousands of lives); Cross, *supra* note 165, at 908-14 (discussing examples of misallocation, including how undue attention to pesticide residues on food draws regulatory attention from much greater hazards of microbial food contamination).

273. For an extensive review of the effects of reduced income on health harms, see Frank B. Cross, *When Environmental Regulations Kill: The Role of Health/Health Analysis*, 22 ECOLOGY L.Q. 729 (1995); Susan L. Ettner, *New Evidence on the Relationship Between Income and Health*, 15 J. HEALTH ECON. 49 (1996).

applications are too often ignored. A reduction in pesticide usage probably would result in increasing the acreage under cultivation. Much of this new acreage would be on "marginal land and would increase erosion and sedimentation, increase use of fertilizer and energy, and reduce wildlife habitat."[274] Many individuals do not realize that "traditional and organic farmers suffer the highest rates of soil erosion per ton of food output."[275]

IV. ESCAPING PERVERSE POLICIES UNDER FQPA

The only express statutory provision enabling escape from regulatory actions that paradoxically increase risk is the exemption provision. Unfortunately, this provision is closely bounded and does not expressly permit consideration of adverse consequences as a basis for granting exemptions. Relying on exemptions would strain the statute beyond reasonable limits and surely fail a test of judicial review. Some of the statutory perversities may be avoided through certain modified risk assessment procedures. Such modifications are not necessarily dishonest intellectually; risk assessment includes a considerable amount of "trans-science," policy judgments not grounded in scientific evidence.[276]

Congress plainly stated that the FQPA "does not preclude EPA from changing its risk assessment methodology as the science of risk assessment evolves."[277] Adjusting risk assessment methodologies could mitigate some of the paradoxical consequences of the law. Quantitative assessments of risks to humans from pesticides are highly uncertain and depend upon a range of assumptions. The presence of such debatable assumptions leaves considerable discretion in the risk assessor.[278]

Given risk assessment discretion, government could modify its assessment practices in order to reach more desirable results. Government could employ more realistic, rather than conservative, assumptions in the assessment. Such modifications are generally scientifically defensible. Current approaches to risk assessment commonly use "conservative" assumptions in order to ensure that the true risk could not be plausibly

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274. *See 1995 Hearings, supra* note 31, at 177 (statement of Professor C. Robert Taylor).

275. Avery, *supra* note 194, at 74.

276. *See* Wendy E. Wagner, *The Science Charade in Toxic Risk Regulation*, 95 COLUM. L. REV. 1613, 1619-22 (addressing policy considerations of trans-science) & 1720-23 (1995) (listing National Research Council's trans-scientific junctures in carcinogen risk assessment).

277. H.R. REP. NO. 104-669, pt. 2, at 41 (1996).

278. *See generally* COMM. ON RISK ASSESSMENT OF HAZARDOUS AIR POLLUTANTS, NATIONAL RESEARCH COUNCIL, SCIENCE AND JUDGMENT IN RISK ASSESSMENT (1994); COHRSSEN & COVELLO, *supra* note 154; Mark Eliot Shere, *The Myth of Meaningful Environmental Risk Assessment*, 19 HARV. ENVTL. L. REV. 409 (1995).

understated by the assessment.[279] Within the context of food safety decision making, regulators use conservative default assumptions designed to produce the largest plausible estimated risk.[280] Yet a conservative approach to addressing one risk may simply transfer and increase risks elsewhere, so that apparent conservatism may in reality increase overall risk.[281] Given the paradoxes possible from overregulation of pesticide residues on food, it makes more sense to obtain the most accurate assessment possible, without seeking conservatism.

The risk assessment process involves several distinct steps. First is *hazard identification*, a process through which a substance is identified as hazardous, such as by finding it to be a carcinogen. The hazard identification process is not highly conservative[282] and often involves animal bioassays. In animal bioassays, laboratory animals are dosed with a substance and then followed for tumorigenicity. If the exposed animals have a statistically significant greater number of tumors than control animals, this might be used as the basis for finding the substance carcinogenic. The evidence is seldom unambiguous, and EPA has used a scale of probable carcinogenicity for various substances.[283] This identification has a bias for positive findings, though, and "uncertainties regarding the biological meaning of certain types of tumors are usually resolved by assuming the worst plausible interpretation."[284]

Under the traditional Delaney Clause, a finding that a substance induced

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279. *See, e.g.*, BREYER, *supra* note 172, at 47 (suggesting that prevailing risk assessment practices often overstate risks by "factors of a thousand or even a million or more"); Philip H. Abelson, *Exaggerated Risks of Chemicals*, 48 J. CLINICAL EPIDEMIOLOGY 173, 175 (1995) (concluding that risk assessment overstates the true risk to human health by a factor ranging from one hundred to infinity); Wagner, *supra* note 276, at 1629 n.55 (observing "agencies typically err on the side of more stringent standards in order to be conservative"); L. Daniel Maxim, *Problems Associated with the Use of Conservative Assumptions in Exposure and Risk Analysis*, *in* THE RISK ASSESSMENT OF ENVIRONMENTAL HAZARDS, *supra* note 67, at 526 (surveying literature on use of conservative assumptions).

280. *See* Joseph Rodricks & Michael R. Taylor, *Application of Risk Assessment to Food Safety Decision Making*, *in* READINGS IN RISK 143, 150-51 (Theodore S. Glickman & Michael Gough eds., 1990) (listing conservative default assumptions and noting that they will overstate risk by a "substantial" amount "in many cases").

281. *See generally* Cross, *supra* note 165.

282. The requirement of statistical significance combats conservatism somewhat. However, conservatism is introduced through EPA's methods of counting tumors, by including benign tumors and pre-tumor neoplasms and estimation methods. Compared to the identified number, the "most likely number of excess tumors is certainly lower, and may be zero[.]" Shere, *supra* note 278, at 435-36.

283. *See id.* at 431-32 (explaining EPA scale). EPA's proposed changes in its Guidelines for Carcinogen Risk Assessment would move away from this scale to a more simplified classification scheme. 61 Fed. Reg. 17,960, 17,961 (1996).

284. *See* Rodricks & Taylor, *supra* note 280, at 150.

cancer in laboratory animals was sufficient to invoke a prohibition. Under the new "reasonable certainty" de minimis risk standard for residues, EPA must obtain a quantitative estimate of cancer risk to humans. Seldom is actual evidence of human risk available. Epidemiological studies have a variety of methodological difficulties[285] and, more profoundly, can detect only very large increases in cancer rates.[286] A one in one million risk to be regulated by the FQPA is not conceivably detectable epidemiologically. Quantitative risk assessment, therefore, typically requires a *dose-response assessment* extrapolation of the animal bioassay results and relies upon highly uncertain assumptions to enable the extrapolation.

To obtain statistically significant evidence of carcinogenicity requires a significant excess of tumors, which in turn often requires that very high doses be given to laboratory animals.[287] Commonly, researchers use several dose levels. These include a level called LD_{50}, which corresponds to the level at which fifty percent of the study animals will die from immediate acutely toxic effects, or one called MTD (maximum tolerated dose), which is the highest dose a species can tolerate without significant noncarcinogenic effect.[288] Such high level exposures may be required for a study's consideration by government.[289] Yet MTD exposures are uncertain guides to inherent carcinogenicity of a substance because they may cause qualitative changes in the subject animal or overwhelm self-defense mechanisms that are effective at lower doses.[290]

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285. Epidemiological studies conducted in the real world lack laboratory conditions. Hence it is difficult to distinguish exposed populations and control for the interfering effects of other variables, among other factors. *See generally* Michael Dore, *A Proposed Standard for Evaluating the Use of Epidemiological Evidence in Toxic Tort and Other Personal Injury Cases*, 28 HOW. L.J. 667 (1985); *see also* T.W. Fuhremann, *Food Safety Assessment for Various Classes of Carcinogens*, *in* PESTICIDE RESIDUES AND FOOD SAFETY, *supra* note 48, at 221, 222 (observing that "reliable human epidemiology data is seldom available and that direct human testing is not possible").

286. *See* FRANK B. CROSS, ENVIRONMENTALLY INDUCED CANCER AND THE LAW 47 (1989) (noting that epidemiology can detect only "gross increases" such as a 30% increase in cancer rates); Dale Hattis & David Kennedy, *Assessing Risks from Health Hazards: An Imperfect Science*, *in* READINGS IN RISK, *supra* note 280, at 156, 158 (noting that "the rates of specific illnesses from a given hazard often must be increased several times above average before one can conclude that they aren't simply random fluctuations").

287. *See* COHRSSEN & COVELLO, *supra* note 154, at 42 (noting that doses "in bioassays are necessarily relatively high so as to increase the sensitivity of the experiments").

288. *See id.* at 39-42.

289. *See, e.g.*, 62 Fed. Reg. 5333, 5334 (1997)(rejecting consideration of negative study on glufosinate ammonium because a "high enough dose was not tested").

290. *See* COHRSSEN & COVELLO, *supra* note 154, at 53; Abelson, *supra* note 279, at 176. Of the substances found carcinogenic in animal tests, only one-third showed carcinogenicity at doses less than the MTD. *See id.*; *see also* Maria E. Matteo, *How Many Mice Must Die?*, 7 TEMPLE ENVTL. L. & TECH. J. 103, 109 (1988) (describing use of MTD and how doses lower than MTD but higher than actual human exposure levels often yield negative doses).

Once it is established that a certain high dose of a substance produces statistically significant increases in tumors in certain laboratory animals (generally rats or mice), the government must take those results and quantify the increase expected for a lower exposure level in humans. When experimental data yield conflicting results, the agency choses the set "which will yield the highest estimate of low dose risk."[291] This requires that assumptions be made about two major factors—the translation of the animal species to humans and the translation from a higher dose to a lower dose. Both are quite debatable.

Translating results among species is complicated and requires some simplifying assumptions. It is not even clear that such translations are scientifically appropriate.[292] The "rodent studies now used to predict human risk were never intended for that purpose."[293] Yet prevailing patterns of conservatism require that the translation be made. In making the cross-species analysis, a major problem derives from the disparate size of humans and test animals. Relative doses require some size scaling, which might be done by body weight, surface area, or by some other method. The choice between these two methods is highly salient. Extrapolations based on surface area rather than body weight may result in a tenfold increase in estimated risk.[294]

Other interspecies differences also require uncertain extrapolation.[295] Some interspecies differences call the entire process into question. For example, one study found an elevated cancer rate from exposure to unleaded

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291. *See* Rodricks & Taylor, *supra* note 280, at 150.

292. *See, e.g.*, Aaron Wildavsky, *Regulation of Carcinogens: Are Animal Tests a Sound Foundation?*, INDEP. REV. 29 (questioning reliance on such tests); Lester B. Lave et al., *Information Value of the Rodent Bioassay*, 336 NATURE 631, 633 (1988) (suggesting that rodent bioassays "give limited and uncertain information on carcinogenicity"); Abelson, *supra* note 279, at 176 (observing that humans and rodents may "differ significantly in their modes of biochemical and physiological disposition of chemicals"); COHRSSEN & COVELLO, *supra* note 154, at 42 (saying that "long-term animal bioassays may not be completely reliable predictors of carcinogenicity in humans" and that at least some earlier studies provide only "limited or inadequate evidence of carcinogenicity"). Many substances are carcinogenic in rats but not mice, and vice versa. *See id.* The extrapolation is even more complicated by a tendency to rely upon unusually sensitive test species in bioassays. *See* OFFICE OF MANAGEMENT AND BUDGET, REGULATORY PROGRAM OF THE UNITED STATES GOVERNMENT 16-17 (1991); *see also* Abelson, *supra* note 279, at 175 (questioning use of "inbred, obese rodents" as a proxy for effects in humans).

293. Dennis J. Paustenbach, *Health Risk Assessments: Opportunities and Pitfalls*, 14 COLUM. J. ENVTL. L. 379, 397 (1989).

294. *See* Arlene Yang, *Standards and Uncertainty in Risk Assessment*, 3 N.Y.U. ENVTL. L.J. 523, 529 (1995).

295. *See* COHRSSEN & COVELLO, *supra* note 154, at 81 (observing that such extrapolations are "highly uncertain because of differences in size, metabolism, anatomy, physiology, and population heterogeneity").

gasoline in male rats, but not female rats or mice. Further research disclosed that the increase was due to the buildup in kidneys of a protein that is only found in male rats, not in humans.[296] Yet another translation problem arises because the routes of administration of the substance in test animals are often different from the route of human exposure.[297]

With respect to the translation from a higher dose to a lower dose, government agencies typically use some form of a linear model.[298] This model assumes that tumors are proportionately lower with lower exposures, but that there is no safe exposure level above zero.[299] This model does not consider the possibility of a threshold safe level or a hormetic effect at low levels. Yet "there is no consistent scientific rationale for assuming a linear relation between dose and response."[300] The extrapolation is grounded in malleable political policies rather than science.

Many linear models do not account for the biological mechanisms of carcinogenesis.[301] Accounting for mechanisms can significantly change the extrapolation. For example, carcinogens may be distinguished between those that directly cause gene mutation or alternation, called genotoxic or initiators, and those that may contribute to cell proliferation only after such damage has already occurred, called epigenetic or nongenotoxic or promoters.[302] The case for linearity is stronger for the first set of substances, while epigenetic effects are more likely to have a threshold.[303] Agencies have historically assumed that all carcinogens were genotoxic,[304] though this presumption may be changing. Even the mutagenic effects of genotoxic substances may sometimes be attributed to the very high MTD level used in animal

296. *See* Wildavsky, *supra* note 292, at 35; CENTER FOR RISK ANALYSIS, HARVARD SCHOOL OF PUBLIC HEALTH, A HISTORICAL PERSPECTIVE ON RISK ASSESSMENT IN THE FEDERAL GOVERNMENT 43 (1994). A more common problem arises when substances cause cancer in the zymbal gland of the rat, because humans have no such gland. OFFICE OF MANAGEMENT AND BUDGET, *supra* note 292, at 19.

297. CROSS, *supra* note 286, at 43.

298. *See* Wildavsky, *supra* note 292, at 37 (noting that "[t]he regulatory response is that the dose-response relationship is linear"). Federal agencies are currently easing away from an automatic presumption of linearity, but such models remain the default choice, used in most instances.

299. *See* Rodricks & Taylor, *supra* note 280, at 150 (reporting that agencies "select mathematical models for high-to-low dose extrapolations that yield the highest prediction of risk at low doses").

300. BREYER, *supra* note 172, at 44.

301. *See* Wildavsky, *supra* note 292, at 36-39 (noting the lack of biological explanation for extrapolation models).

302. *See* discussion in Wildavsky, *supra* note 292, at 40-41; Matteo, *supra* note 290, at 107.

303. Wildavsky, *supra* note 292, at 41 (suggesting threshold for nongenotoxic substances); CENTER FOR RISK ANALYSIS, *supra* note 296, at 43 (same).

304. *See* Rodricks & Taylor, *supra* note 280, at 150 (noting that [a]gencies assume all carcinogens act by the same mechanism (genotoxicity), which is the mechanism that predicts the greatest risk at low dose").

bioassays.[305]

Some substances may be carcinogenic only because they produce certain metabolic changes in the body. These metabolic changes may be dose-dependent, which would mean that there was a safe threshold below which the substance was not carcinogenic.[306] Regulatory policy, though, "assumes all [carcinogens] to be of the riskiest kind," which "seems odd, given all we have read about the important mechanistic distinctions among toxicants of this class, and the possibility of sublinear or even thresholds in the dose-response curves of some of them."[307] EPA's proposed changes in its Guidelines for Carcinogen Risk Assessment admit the relevance of metabolic and pharmacokinetic data, but still have a default presumption of linearity.[308]

The risk assessment of a substance culminates with some measure of abstract carcinogenic potency. To ascertain actual human risk, EPA also requires some measure of human exposure to the substance. These exposure assessments are frequently as conservative as the potency assessments. Exposure assessments typically employ unrealistic assumptions and may presume that an individual remains exposed consistently to the maximum possible level throughout an entire seventy-year lifetime.[309]

Exposure assessments for pesticides commonly are based on the "maximum amount of pesticide which could be ingested."[310] For example, regulators may "[a]ssume a person consumes food at a very high rate (90th percentile of consumers of that food) and that every mouthful for a whole lifetime contains a pesticide residue at the maximum allowed concentration."[311] When EPA checked its theoretical risk assessment for the pesticide captan against real world data, the agency discovered that the former assessment overestimated exposure by a hundredfold.[312] Other

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305. Bruce N. Ames & Lois Swirsky Gold, *Too Many Rodent Carcinogens: Mitogenesis Increases Mutagenesis*, 249 SCIENCE 970, 870 (1990).

306. *See* CENTER FOR RISK ANALYSIS, *supra* note 296, at 42 (noting that consideration of pharmacokinetic information on substances can suggest propriety of non-linear extrapolation of risk).

307. RODRICKS, *supra* note 175, at 189. See *id.* at 145-57 for a discussion of such mechanistic differences.

308. *See* 61 Fed. Reg. at 17,960, 17,968.

309. OFFICE OF MANAGEMENT AND BUDGET, *supra* note 292, at 22-23; *see also* BREYER, *supra* note 172, at 46 (describing unrealistic assumptions in exposure assessment for groundwater exposure); A HISTORICAL PERSPECTIVE ON RISK ASSESSMENT IN THE FEDERAL GOVERNMENT, *supra* note 296, at 38 (finding that government exposure scenarios are "hypothetical and arbitrary"). When setting a tolerance for carboxin under the FQPA, EPA considered the unrealistic Theoretical Maximum Residue Contribution which assumes that all foods have the maximum concentration. *See* 62 Fed. Reg. 4911, 4913 (1997). However, the agency went on to express a willingness to consider more accurate measures of actual exposure if available. *See id.*

310. RODGERS, *supra* note 56, at § 5.21(C).

311. RODRICKS, *supra* note 175, at 190.

312. OFFICE OF MANAGEMENT AND BUDGET, *supra* note 292, at 23.

assumptions similarly tend to overstate actual exposure to pesticide residues.[313] As discussed above, typical actual exposures are far below permissible tolerances.

Changes in risk assessment extrapolation judgments could produce a substantial effect on estimated risks and corresponding levels of "reasonable certainty that no harm will result" (one-in-one-million risk of cancer). Changing conservative assumptions to most-likely-estimate assumptions will produce risk assessments that allow greater pesticide usage. Indeed, changing the assumptions can potentially alter risk estimates by a factor of 100,000 or more.[314]

While the variations in quantitative risk assessment estimates are often overstated as a practical matter, much lower alterations, say a factor of two, would have a very practical significance on pesticide residue tolerance levels. Such a modification could help avert the overregulation of pesticides based on fears of food residues, thereby ameliorating the paradoxical health consequences.

The risk assessment approaches discussed above might mitigate the perverse paradoxes of pesticide regulation, but these approaches are highly imperfect, indirect solutions to the problems. Some paradoxes, such as the hormetic paradox, could be cured largely through improved risk assessment methodologies. Others, such as the benefits and farmworker paradoxes might be ameliorated—less overall regulation would permit more benefits and require less risk-shifting to workers. But such amelioration would be only coincidental and fortuitous.[315] Because risk assessment fixes do not confront the tradeoffs of the paradoxes themselves, changes in assessment represent a partial solution and may perversely prevent society from confronting the reality of such tradeoffs.

V. SENSIBLE AND SAFE PESTICIDE REGULATION

The need for government regulation of pesticide residues is not terribly

313. *See* Tomerlin & Engler, *supra* note 160, at 198. For example, the assessments assume "that all foods presumed to contain residues of the pesticide are eaten at one sitting" and that pesticide residues are uniformly distributed. *See id.*

314. *See* Yang, *supra* note 294, at 553.

315. Optimal results might not depend on happenstance, as regulators might adapt risk assessment assumptions to reach the desired result in each case. This effect is by no means unknown in environmental regulation. *See* Wagner, *supra* note 276, at 1645 n.107 (observing that regulators use risk assessment to legitimate their prior choices). Such an approach suffers from dishonesty and inconsistency, however, which can make regulatory decisions vulnerable on appeal to courts. Moreover, the science can be stretched only so far, and even premeditated, strategic use of risk assessment cannot guarantee a justification for optimal decisions.

strong. The common case for environmental regulation rests on the concept of externalities. For example, air pollution is nearly free to the polluter but imposes potentially significant costs on others, who have very little opportunity to control the pollution through the free market.[316] While pesticides also have negative externalities, agricultural producers themselves bear much of the cost of pesticide applications, giving them ample incentive to reduce usage when unnecessary.[317] Moreover, when discussing residues in food, consumers who prefer to avoid the risk can do so by purchasing organic produce at higher cost.[318] Those who prefer lower cost fruits and vegetables may accept a risk from pesticide residues. Theory suggests that negative externalities are therefore slight, and evidence indicates that this is indeed the case. Considerable experience demonstrates the market incentives for reducing any pesticide risk.[319] Because pesticides "account for 0.01% of the total carcinogens" in food, the case for regulation is not strong.[320]

There remains, however, some case for regulating pesticide residues.[321] Extremely low prevailing risk levels may be in part due to the existence of regulation. A useful pesticide might prove highly hazardous to consumers, even at low residue levels. Such consumers cannot be expected to take the time to conduct research necessary to protect themselves from the hazard.

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316. While Coase taught us that those who breathe air pollution theoretically might combine and contract with polluters to reduce emissions, the transaction costs of such action make it highly unrealistic.

317. *See, e.g.*, G. Schuhmann, *The Economic Impact of Pesticides on Advanced Countries*, *in* PESTICIDES AND HUMAN WELFARE, *supra* note 177, at 55, 61 (observing that "[s]o far as the farmer is concerned his input of pesticides must be justified by the increased value of his crop"). This internalization of costs of pesticide applications is not perfect. Absent common law liability, producers do not bear the external costs of disease from residue on foods. However, because the amount of residue roughly tracks the amount of pesticide applied, producers have an indirect incentive to control residues.

318. William Rodgers argues that consumers have no meaningful way to escape pesticides on food, but does not mention why the presence of certifiably organic produce does not provide such choice. RODGERS, *supra* note 56, at § 5.21(A).

319. *See* Perry J. Gehring, *Risk Management in the Absence of Credible Risk Assessment*, *in* PESTICIDE RESIDUES AND FOOD SAFETY, *supra* note 48, at 267, 274:

> Newer pesticides are being developed with use rates a thousand times less, and these pesticides are more selective to the target and have less capability to move to unwanted sites. Users of pesticides are being trained and licensed. Delivery systems are being developed that reduce exposure to the users, reduce the risk of environmental contamination, and minimize the chance of over-application. Industry, government and academia are cooperating to elucidate more efficient, targeted pesticide application that maximizes the desired effect while minimizing potential adverse risks to health and the environment.

Id.

320. Green Mountain Institute, *supra* note 152, at 14.

321. The Green Mountain Institute for Environmental Democracy concluded that "low levels of concern" for pesticides in food were warranted, given the large exposed population and the possibility of tolerance violations. *See* Green Mountain Institute, *supra* note 152, at 13.

Requiring each individual to self-inform would be less efficient than the use of government regulation. While some pesticide residue regulation can be justified, the relative weakness of the justification means that regulation might readily become counterproductive; government should therefore proceed carefully.

Sane regulation of pesticides, in order to avoid perverse paradoxes, must incorporate two important principles. First, standards must be consistent throughout the various risks presented by the chemicals.[322] Otherwise, there will be a systematic tendency to shift risks from areas of tougher standards to areas where standards are more lax. Differential standards readily permit not only a transfer of risk but an increase in overall risk. The risk increase and transfer attributable to differential standards also will tend to injure already disadvantaged groups, such as farm-workers and the poor.

Second, standards must consider the benefits of pesticide applications. Pesticide use produces positive externalities to human health. Environmentalists have long resisted such benefit considerations, because benefits were expressed as economic gains to producers or manufacturers, and were used to justify risks to health.[323] But the critical benefits of pesticides are not the financial ones but those related to human health.[324] Pesticides provide considerable health benefits by combatting or indirectly reducing exposure to natural toxins and by assisting the production of low cost fruits and vegetables. The case for excluding consideration of such health benefits is hard to imagine. When the statutory goal is public health protection, the law should not direct actions that undermine health.

Of course, the popular perception of pesticides as chemical invaders of our health lies at the root of paradoxical regulatory pursuits.[325] Given the public fears and predilections, it is not surprising that Congress compromised on a stringent regulatory approach for pesticide residues, notwithstanding the paradoxical health consequences of that approach. But we need not be prisoners of public misperceptions.[326] Administrative tinkering with assessment methodologies can alleviate the paradoxical risks created by the

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322. *See* Curme, *supra* note 30, at 626 (discussing how inconsistent standards result in higher risks of cancer).

323. In practice, EPA's cost/benefit balancing has focused on the calculation of monetary losses to growers and not the costs or foregone benefits to consumers. *See* Cropper et al., *supra* note 16, at 194.

324. For a discussion of this distinction, see RODGERS, *supra* note 56, at § 5.22(B)(3).

325. *See* Furtick, *supra* note 177, at 11 (observing that much of the concern over pesticide exposures "is derived from people who have unfounded fear about all synthetic chemicals who seek to consume or come into contact with only 'natural' substances").

326. *See* Cross, *supra* note 260, generally and at 955-58 (recommending measures to focus risk regulation on scientific estimates rather than public misperceptions).

FQPA, but cannot do so in a principled manner and cannot eliminate those risks. Government needs to recognize a new paradigm for pesticide regulation that incorporates the considerable benefits attendant to pesticides and recognizes the risks associated with reckless regulation. The FQPA made some advances (adding consistency for raw and produced foods, directing more attention toward past registered pesticides rather than dwelling on new products), but the Act failed to address more serious paradoxes and its enhancement of regulatory stringency could greatly exacerbate those paradoxes.