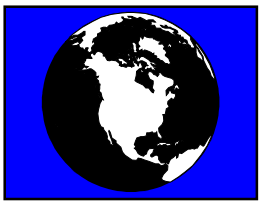


Center for International Trade and Agriculture (CITA)

CITA WORKING PAPER #5-2016

AGRICULTURE, FOOD, AND HUMAN HEALTH: A SURVEY OF ISSUES, CONCERNS, AND IMPLICATIONS

Editor's Note. The University of Kansas School of Law initiated the operations of the Center for International Trade and Agriculture ("CITA") in early 2010 with an inaugural symposium held in Lawrence, Kansas. Shortly thereafter another element of the CITA's expanding operations was put in place: the CITA Working Papers Series. The overall aim of the CITA Working Papers Series is to provide a forum in which interesting information and insights on a broad range of issues that lie at the intersection of three subjects – international trade, agriculture, and law – can be expressed and discussed in a manner that will benefit legal practitioners, policy-makers, academics, and those members of the general public who are interested in a thoughtful exchange of views on these issues. Although the CITA went into "dormant" status in 2014, occasional contributions to the CITA Working Paper Series are still invited.

For more information about the CITA Working Papers Series, including a set of "Guidelines for Contributions", see the CITA website – http://law.ku.edu/cita#. As explained there, contributions to the CITA Working Papers Series are intended to be just that: "working" papers. A contribution to the Series need not provide a completely polished "last word" on the author's views but can be a work in progress intended to solicit comments and responses from readers.

The first contribution to the CITA Working Papers Series – CITA Working Paper #1-2010 – was focused on international legal regimes to balance the protection of prairies and grasslands with their agricultural use. CITA Working Paper #2-2011, written by Dr. Surendra Bhandari, provided a country study on Nepal in the context of international trade and agriculture. Working Paper #3-2011, written by Ms. Jomana Qaddour, provided a country study on Syria in the context of international trade and agriculture. Working Paper #4-2011, written by Ms. Heba Hazzaa, offered insights into the challenges facing Egypt's economic sector – with special attention to international trade and agriculture – in the face of both internal and external changes. Now CITA is pleased to post CITA Working Paper #5-2016, examining the impacts that industrial agriculture has on human health. Comments on the paper are welcome, particularly in the form of either (i) direct communications with the author (caleb.hall.ch@gmail.com) or (ii) submission of a contribution to the CITA Working Papers Series on this or a related topic.

* * * * * * *

Agriculture, Food, and Human Health: A Survey of Issues, Concerns, and Implications

Caleb Hall *

O u t l i n e

---

* Caleb Hall, an attorney with background in environmental science and legal experience in environmental law and policy, earned his B.S. and J.D. degrees from the University of Kansas and is now a legislative analyst for the Missouri House of Representatives.

* * * * * * *

What direct implications does industrial agriculture have on human health? For instance, what human-health effects flow from the use of agricultural chemicals in the production of grains, which account for roughly two-thirds of human caloric intake? How does the rising reliance on antibiotics, genetic engineering, and other new innovations affect human health? As a principal contributing researcher for a larger study on global agriculture,[1] I examine in this article these and other human-health concerns associated with modern agriculture.

Specifically, I explore below five public health issues and concerns: (1) agricultural chemicals, (2) genetic engineering and genetic modification, (3) food-borne illness, (4) confined animal feeding operations ("CAFOs"), and (5) obesity. Naturally, some of these issues interact with the others, and most of them have causes and implications that lie outside the parameters of agriculture. Still, my aim is to draw from the relevant policy, technical, and professional literature in order to offer a brief survey of pertinent issues and concerns that can, or should, have important implications in shaping new national and global agricultural policies.

As an introductory note, it is worth emphasizing that the need for such new national and global agricultural policies is especially great now, in the aftermath of the so-called "Green Revolution" that resulted in such an enormous intensification of agricultural production. Although the plant-breeding innovations of the Green Revolution brought a dramatic increase in yields that could be coaxed from plants (particularly in such staple grains as wheat, rice, soybeans, and corn), some of its environmental implications – emerging, for instance, from its heavy use of newly-developed fossil-carbon-based fertilizers – have prompted a broad critical

[1] For a published manifestation of this study of global agriculture, see generally John W. Head, INTERNATIONAL LAW AND AGRO-ECOLOGICAL HUSBANDRY: BUILDING LEGAL FOUNDATIONS FOR A NEW AGRICULTURE (forthcoming 2016).

assessment of the Green Revolution.[2] While my aim in this article is not to explore the Green Revolution and its long-term implications in general, it is important to bear in mind that the cluster of specific human-health issues that I examine below fits within a broader landscape of concerns over agricultural policy for the world's current circumstances.

I. Agricultural Chemicals

A. An Array of Fertilizers and Pesticides

Aside from its emphasis on plant-breeding and its development of hybrid crops, some of the Green Revolution's biggest innovations involved agricultural chemicals such as pesticides and fertilizers,[3] so that today's agriculture – especially in the most highly developed agricultural systems – includes a wide variety of such substances. See Box #1 for a summary of the principal categories of agricultural fertilizers and pesticides.

Box #1. Categories of Principal Agricultural Fertilizers and Pesticides

This summary catalogue of key agricultural fertilizers and pesticides follows the relevant categories used by the FAO. Some representative commercial brand names have been indicated parenthetically.

Fertilizers

There are four main types of fertilizers: nitrogen (N), phosphate (P_20_5), potash (K_20) and complex (NP, PK, NK and NPK).

[See Food & Agriculture Organization, *FAOSTAT*, United Nations (2013), http://faostat.fao.org/site/575/default.aspx#ancor.] *Box #1 continues on next page*

[2] Some of the criticisms emerged over 40 years ago. See generally Harry Cleaver, *The Contradictions of the Green Revolution*, 62 THE AMERICAN ECONOMIC REVIEW, issue 1/2 (Mar. 1972), http://www.jstor.org/discover/10.2307/1821541?uid=3739672&uid=2&uid=4&uid=3739256&sid=2110490907646. The criticisms have intensified, however, in recent years. For one set of criticisms indicting the Green Revolution for its contributions to biodiversity loss, soil erosion and degradation, water scarcity, and greenhouse gas emissions, see *Sustainable Agriculture – An Overview*, on the website of the University of Arkansas National Agricultural Law Center, at http://www.nationalaglawcenter.org/assets/overviews/sustainableag.html. For another explanation of several political, socioeconomic, and nutritional criticisms leveled at the Green Revolution, see Daniel Pepper, *The Toxic Consequences of the Green Revolution*, available on the website for U.S. News & World Report at http://www.usnews.com/news/world/articles/2008/07/07/the-toxic-consequences-of-the-green-revolution.(July 7, 2008). It is worth noting that the term "Green Revolution" is not used consistently in the literature. In a narrow connotation, it encompasses just the plant-breeding and associated advances emerging from the work of a cadre of researchers (most prominently Norman Borlaug) with financial support from the Rockefeller Foundation and the US Government. In a wider connotation, the Green Revolution encompasses also a range of other developments and innovations that capitalized on advances in fossil-carbon fertilizers and other exogenous inputs.

[3] *See* Cleaver, *supra* note 2, at 177-86.

Pesticides

There are six key types of pesticides: (1) insecticides, (2) mineral oils, (3) herbicides, (4) fungicides and bactericides, (5) plant growth regulators, and (6) rodenticides.

Within the *insecticide* group there are chlorinated hydrocarbons (e.g., DDT, mirex, aldrin) used for arthropod pests generally, organo-phosphates (e.g., Dursban, Nuvan Top, Vapona, Kontrol) used for arthropods generally as well as in shampoos and pet care products, carbamates (e.g., Temik, Award, Logic, Larvin) used for arthropods generally but especially for Hymenoptera, and pyrethroids (e.g., Biomist, Scourge, Anvil) used widely in home arthropod control.

There are also certain insecticides used to treat seeds such as organo-phosphates, carbamates, and pyrethroids.

Within the *mineral oils* group, various products (e.g., Saf-T Side, Orchex, Trilogy) are used for arthropod pests generally, especially in organic farming or applications.

Within the *herbicide* group there are triazines (e.g., Miracle Gro, Scotts Bonus S Weed and Feed, Monsanto Lariat) used to kill broadleaf plants, phenoxy hormone products (e.g., Weed-B-Gone, Weedmaster) used to kill broadleaf plants, amides (e.g., Frontier, Outlook, Stampede) used to kill broadleaf plants and yellow nutsedge, carbamates (e.g., Chlorpropham, Sulfallate, Phenmedipham) used as photosynthesis inhibitors, dinitroanilines (e.g., Prowl H_2O) used to kill grasses and small seed broadleaf plants, urea derivatives used primarily in pre-emergence application against broadleaf plants, bipiridyls (e.g., paraquat, diquat) used as an enzyme inhibitor, and uracil (e.g., bromacil, isocil) used as a photosynthetic inhibitor. Another prominent herbicide is glyphosate, marketed beginning in the 1970s as "Roundup". Glyphosate is used for comprehensive elimination of plants – with the exception of some grain crops (such as "Roundup-Ready" corn) that have been developed to survive exposure to glyphosate.

Within the *fungicide and bactericide* group there are inorganics (including sulfur, sodium azide, potassium azide, marketed under various brand names), dithiocarbamates (e.g., Polyram, Manzate, Zineb), benzimidazoles (e.g., Spectrum), triazoles and diazoles (e.g., Stratego YLD), and diazines and morpholines (marketed under various brand names), each product being used to kill fungi and bacteria on different crop species. There are also those fungicides used to treat seeds such as dithiocarbamates, benzimidazoles, and triazoles diazoles.

Pesticides in the *plant growth regulators* group (e.g., Stinger, Paramount, Clarity) are used to make weeds grow uncontrollably till they die.

Within the *rodenticide* group there are anti-coagulants (e.g., Contrac, Terad$_3$ Blox, Rentokil), cyanide, hypercalcaemics (e.g., Agrid$_3$, CyKill), and narcotics, all of which are used against a variety of mammalian pests.

[Food & Agriculture Organization, *FAOSTAT*, United Nations (2013), http://faostat.fao.org/site/424/default.aspx#ancor.]

The introduction of fertilizers and pesticides of various types has undoubtedly aided in exponentially increasing crop yields, especially in the period from the 1940s to the 1970s and

extending to today. However, they have also attracted criticism for some environmental catastrophes, such as DDT's near extermination of the bald eagle. It seems that their public image has not fully recovered even today, several decades later, as there are now still numerous environmental-protection advocacy groups – including, for instance, Food & Water Watch and the Environmental Working Group – warning consumers about residual chemicals on their produce, meat, and cosmetics.[4] It seems as if having "cide" at the end of the names of these chemical groups (herbi*cide*, insecti*cide*, etc.), referring to "death" or "killing", gives them an unfair starting place to try to assuage consumer fears. However, maybe those concerns are not so irrational.

B. Concerns

The occupational hazards related to agricultural chemicals are clearly substantiated. The 20,000 pesticides currently marketed in the USA cause 10,000 to 20,000 farm worker poisonings each year because of their high degree of contact with crops.[5] Farmers could then potentially benefit physically by reducing pesticide use, and, harmoniously, this could also align with their economic interests as well. A recent study in France demonstrated that famers were able to reduce their costs, using fewer quantities of agricultural chemicals per hectare in a more labor intensive system, by as much as 25% in 80% of the studied cases.[6] Those results are also consistent with observations of Dutch sugar beet farmers.[7] As Frederick Kirschenmann has expressed it, ecological approaches to pest control and agriculture in general – as opposed to biotechnology approaches – have "produced positive effects. Agriculture that is based on complexities cannot be readily managed in large-scale monocultures. Highly intensive methods may support more people on the land with smaller-scale, highly productive farms."[8]

That both financial and health interests urge against prolific use of pesticides and herbicides makes it cruelly ironic that worker poisonings are still occurring. This continues despite the safeguards and education that the USA has implemented – and indeed this makes it all the more understandable that the results can be worse without those precautions. For example, Mexican farm workers have been known to unknowingly and ignorantly expose themselves to sprayings, not wear the recommended protective equipment, and be unable to read English warning labels.[9] If workers are being harmed by these products, then it stands to reason that it

---

[4] See, e.g., *(Triclosan (Endocrine Disruptor)*, Food & Water Watch, http://www.foodandwaterwatch.org/water/triclosan/; *Environmental Working Group*, http://www.ewg.org.

[5] *Pesticide Illness & Injury Surveillance*, Center for Disease Control and Prevention (Sept. 11, 2013), http://www.cdc.gov/niosh/topics/pesticides/.

[6] Jean-Philippe Boussemart et al., *Could Society's Willingness to Reduce Pesticide Use be Aligned with Farmers' Economic Self-Interest?*, 70 ECOLOGICAL ECONOMICS, issue 10, pp. 1797-804 (Aug. 2011), http://www.sciencedirect.com/science/article/pii/S0921800911001947.

[7] *Id.*

[8] Frederick L. Kirschenmann, CULTIVATING AN ECOLOGICAL CONSCIENCE: ESSAYS FROM A FARMER PHILOSOPHER 175 (2010).

[9] Angus Wright, THE DEATH OF RAMON GONZALEZ: THE MODERN AGRICULTURAL DILEMMA 10-50 (1990).

might possibly have health impacts to consumers via eating the sprayed food itself or by water contamination.

Although many of these chemicals are carcinogenic, as long as the pesticide residues in the food are found to have "negligible risk" or be below tolerable levels then these foods will remain on the shelves.[10] This regulatory approach appears to be a cost-benefit risk assessment rooted in the reactive tendencies of American law as opposed to the precautionary principle, which drives European lawmaking.[11] As such, the effectiveness of the US regulatory model is

---

[10] William Eubanks, *A Rotten System: Subsidizing Environmental Degradation and Poor Public Health with Our Nation's Tax Dollars*, 28 STANFORD ENVIRONMENTAL LAW JOURNAL, pp. 213-310 (May 2009), http://pubcit.typepad.com/files/eubanks-1.pdf ; Valerie Watnick, *Our Toxics Regulatory System and Why Risk Assessment Does Not Work: Endocrine Disrupting Chemicals as a Case in Point*, 4 UTAH LAW REVIEW, issue 4, pp. 1305-33 (Dec. 2004), http://papers.ssrn.com/sol3/papers.cfm?abstract_id=2284981.

[11] The US model is described as reactive because regulatory action is begrudged whenever possible, preferring to accept costs or let markets solve problems. The reactive model weighs economic costs more than possible harm, and only when pressure becomes too much, or if acting becomes advantageous, will government react. This is then often associated with political conservatives which is understandable that the reactive model originates in English common law. *See* Noga Morag-Levine, *Is Precautionary Regulation a Civil Law Instrument? Lessons from the History of the Alkali Act*, 23 JOURNAL OF ENVIRONMENTAL LAW, issue 1, pp. 1-43 (Jan. 2011), http://digitalcommons.law.msu.edu/cgi/viewcontent.cgi?article=1415&context=facpubs. The Common law tradition is itself rather conservative in that it is rather resistant to change, preferring to rely on precedent.

On the other hand, the precautionary principle is more often associated with protective politics and a desire to have paternalistic governing agencies act to prevent problems. It can be as lax as simply putting more emphasis on risk, or as strong as "calling for absolute proof of safety before allowing new technologies to be adopted." Kenneth Foster, Paolo Vecchla, & Michael Repacholi, *Science and the Precautionary Principle,* 288 SCIENCE, issue 5468, pp. 979-81 (May 2000), http://people.reed.edu/~ahm/Courses/Reed-POL-372-2011-S3_IEP/Syllabus/EReadings/05.2/05.2.FosterVecchia2000-Risk.pdf . The principle's roots stretch into German civil law, and it is more likely to accept the measures that put more regulatory hurdles to approve consumer products for the sake of public health. Robert V. Percival, *Who's Afraid of the Precautionary Principle?*, 23 PACE ENVIRONMENTAL LAW REVIEW, issue 1, pp. 21-81 (Feb. 2006), http://digitalcommons.pace.edu/cgi/viewcontent.cgi?article=1073&context=pelr. This is because the principle views probable harm as more pressing than supposedly freer markets.

One example of the political dichotomy created by following either the reactive model or precautionary principle is the policy decisions regarding biotechnology. Consider the US Coordinated Framework on Biotechnology, which is meant to focus on "the needs of industry for 'sensible' regulation that would not stifle innovation than on the needs of the public for rigorous regulation to protect public safety." Rebecca Bratspies, *Some Thoughts on the American Approach to Regulating Genetically Modified Organisms*, 16 KANSAS JOURNAL OF LAW & PUBLIC POLICY, issue 3, pp. 393-423 (Oct. 2007), reprinted in Susan Schneider, FOOD, FARMING, AND SUSTAINABILITY: READINGS IN AGRICULTURAL LAW 541-550 (2011), http://papers.ssrn.com/sol3/papers.cfm?abstract_id=1017832. The inherent principles of this Coordinated Framework were that 1) biotechnology poses no unique risks, 2) the product, not the process, should be regulated, 3) existing laws, not new ones, should be used, 4) any regulatory gaps should be coordinated amongst agencies, and 5) that designating any lead agency was inappropriate. *Id.*

However, whereas the US accepts biotechnology products as substantially equivalent to conventional counterparts, Europe and others are initially skeptical. They were slower to adopt GM varieties, and even now that they are starting to accept them the European Union, South Korea, Japan, New Zealand, and Australia either have or are establishing mandatory labeling regimes for genetically engineered food. Tadlock Cowan, *Agricultural Biotechnology: Background and Recent Issues*, Congressional Research Reports Paper 27 (Feb. 2009), reprinted in Susan Schneider, FOOD, FARMING, AND SUSTAINABILITY: READINGS IN AGRICULTURAL LAW 611-616 (2010), http://digitalcommons.unl.edu/crsdocs/27/.

limited because it ignores the very nature of the chemicals the EPA and other governmental bodies are regulating.

Many agricultural chemicals are endocrine disruptors, or synthetic hormones – tricking an organism's body into accepting the hormonal signal and sabotaging normal functions.[12] For insects and other pests, exposure to such chemicals usually just means death, but for humans it can also induce decreased sperm counts, breast, testicular, and prostate cancer, and neurological disorders.[13] Atrazine, the most commonly used pesticide in the USA, is one such endocrine disruptor with observed effects on genitalia – albeit in frogs.[14]

The endocrine disruptors at issue here are also sometimes referred to as persistent organic pollutants (POPs) because of their long life cycles. Whereas non-persistent compounds break down into baser, and hopefully safer, parts, POPs remain in environments, sometimes indefinitely, or remain intact and spread by forces such as wind or water across the entire globe.[15] The notorious DDT, for example, has a habit of traveling pole-ward.[16] In the 1980s, tissue samples were taken from Inuit people in southern Baffin Island and northern Quebec as part of a study of the accumulation of POPs in human breast milk.[17] The women sampled had among the highest recorded levels of POPs in the world, including DDT and PCB.[18] It was hypothesized that they received these chemicals by consuming marine mammals that had absorbed the chemicals as they concentrated up the food chain.

In addition, consider that traces of DDT are still measurable in the USA decades after the country's banning of the POP. In the USA, DDT and its components are still detectible in California condor offspring,[19] as well as the entire Mississippi River basin.[20] Also, even though DDT's *use* is banned in the USA, it still continues to be *produced* in the USA and shipped to

---

For a survey of how the precautionary principle has been reflected in international instruments, see note PPNOTE6, in Chapter 7 of this book.

[12] Sheldon Krimsky, *Hormone Disruptors: A Clue to Understanding the Environmental Causes of Disease*, 43 ENVIRONMENT, issue 4, pp. 22-38 (June 2001), http://www.tufts.edu/~skrimsky/PDF/environ.PDF.

[13] *Id.*

[14] *See, e.g.,* University of California – Berkley, *Pesticide Atrazine Can Turn Male Frogs Into Females*, SCIENCE DAILY (Mar. 10, 2010), http://www.sciencedaily.com/releases/2010/03/100301151927.htm.

[15] Shannon O'Lear, ENVIRONMENTAL POLITICS: SCALE AND POWER 146 (2010).

[16] *Id.* at 150.

[17] Terry Fenge, NORTHERN LIGHTS AGAINST POPS: COMBATTING TOXIC THREATS IN THE ARTIC 193 (2003).

[18] *Id.*

[19] John Moir, *New Hurdle for California Condors May be DDT from Years Ago*, N.Y. TIMES, Nov. 15, 2010, at D3

[20] P. Santschi et al., *Historical Contamination of PAHs, PCBs, DDTs, and Heavy Metals in Mississippi River Delta, Galveston Bay Sediment Cores*, 52 MARINE ENVIRONMENTAL RESEARCH, issue 1, pp. 51-79 (July 2001), http://www.ncbi.nlm.nih.gov/pubmed/11488356.

other countries where the pesticide is used on crops, and then those crops are imported by the USA.[21]

Atrazine also shares the tenaciously existing characteristics of a POP. The Natural Resources Defense Council reports that:

> Approximately 75 percent of stream water and about 40 percent of all groundwater samples from agricultural areas tested in an extensive U.S. Geological Survey study contained atrazine . . . . Atrazine was found in 80 percent of drinking water samples taken in 153 public water systems. All twenty watersheds sampled in 2007 and 2008 had detectable levels of atrazine, and sixteen had average concentrations above the level that has been shown to harm plants and wildlife.[22]

However, Syngenta, Atrazine's manufacturer, offers several countervailing assertions, as follows:

> Levels of atrazine in U.S. waters are well within the federal lifetime drinking water standard of 3 parts-per-billion – a level containing a 1,000-fold safety buffer. In 2008, none of the 122 Community Water Systems monitored in 10 states exceeded the federal standards set for atrazine in drinking water or raw water.[23]

Also, because these endocrine disruptors mimic hormones, they can also be accumulated in human body fat, a process known as bioaccumulation, thereby creating a toxic load that is eventually health damaging and can be released through weight loss.[24] They can also be just as effective in small doses as opposed to larger ones unfortunately.[25] This blurs the bright line between the amount of chemical needed to kill an insect pest and a human being. The effects are not always incremental, but exponential as well.

Therefore, consumers are trapped in a decision-making bind: they are assuaged knowing that these chemicals are regulated and kept at safe levels, but their potential toxicity may not necessarily correlate with exposure amounts. Meanwhile they are constantly advised to reduce

---

[21] O'Lear, *supra* note 15, at 146.

[22] *Atrazine: Poisoning the Well*, Natural Resources Defense Council (Aug. 22, 2009), http://www.nrdc.org/health/atrazine.

[23] *It's Time to Rethink Water*, Syngenta (Feb. 1, 2010), http://www.atrazine.com/water.

[24] Monika Rönn et al., *Circulating Levels of Persistent Organic Pollutants Associate in Divergent Ways to Fat Mass Measured by DXA in Humans*, 85 CHEMOSPHERE, issue 3, pp. 335-43 (Oct. 2011), http://www.ncbi.nlm.nih.gov/pubmed/21767864; M. Cho et al., *Associations of Fat Mass and Lean Mass with Bone Mineral Density Differ by Levels of Persistent Organic Pollutants: National Health and Nutrition Examination Survey 1999 – 2004*, 82 CHEMOSPHERE, issue 9, pp. 1268-76 (Feb. 2011), http://www.ncbi.nlm.nih.gov/pubmed/21196025.

[25] Shelby Flint et al., *Bisphenol A Exposure, Effects, and Policy: A Wildlife Perspective*, 104 JOURNAL OF ENVIRONMENTAL MANAGEMENT, pp. 19-34 (Aug. 2012), http://www.consbio.umn.edu/download/Flint_et_al_2012_BPA.pdf.

exposure to agricultural chemicals while pesticides are still used by the tons. Even more concerning is that despite some concerned consumers' efforts to reduce exposure, pesticide and herbicide use will likely increase. Historically, there has been a tenfold increase in both toxicity and amount of insecticide used in the US from the early 1940s to the 1990s,[26] and newly adopted genetically modified (GM) crops have encouraged current increases in chemical usage – about 7% more pesticide use, or 404 million pounds, between 1996 and 2011.[27]

Ironically, this increase appears to be occurring without concurrently increasing productivity. Also between the early 1940s and the 1990s, crop losses due to insects rose seven to 13%, losses to plant pathogens grew from 10% to 12%, and crops lost to weeds only decreased 2% from 14% originally.[28] This lack of return on chemical investments seems paradoxical and Sisyphean, but understandable given insect and pathogenic potential to create chemical resistance. This is substantiated by 12% corn crop losses in the 1990s, when chemical inputs were common, but loses were only 3.5% in 1945, when insecticides had just begun to be routinely used in the US.[29]

As chemical inputs increased the need for them, crop losses increased as well. This not only implies the strong resilience of pest species, but ultimately throws question on the logic of modern agriculture that technological advancement is justified by increasing productivity and public health. On the contrary, we have increased yields but at the expense of vulnerability to pests, and increased public concerns over health.[30] Considering this, the question whether the public's fears are merely perceived or legitimate should also involve a related question: why do we continue a food production model with such negative feedbacks?

C. Critiques of the Concerns

Well, why do we do so? Possibly because agricultural chemical use has created more food and a higher standard of living than at any other point in history.[31] It cannot be denied that overall people are healthier now, and that the comforts of the developed world exist largely because we have maintained a food surplus allowing people to explore careers and lives outside

---

[26] Harvey Blatt, AMERICA'S FOOD: WHAT YOU DON'T KNOW ABOUT WHAT YOU EAT 40 (2008).

[27] Charles Benbrook, *Impacts of Genetically Engineered Crops on Pesticide Use in the U.S. – the First Sixteen Years*, 24 ENVIRONMENTAL SCIENCES EUROPE, pp. 24-36 (Sept. 2012), http://www.enveurope.com/content/24/1/24.

[28] Blatt, *supra* note 26.

[29] Montague Yudelman, Annu Ratta, & David Nygaard, *Pest Management and Food Production: Looking to the Future*, International Food Policy Research Institute (1998), http://www.ifpri.org/sites/default/files/pubs/2020/dp/dp25.pdf.

[30] *Id.*

[31] William Motes, *Modern Agriculture and its Benefits – Trends, Implications, and Outlook*, Global Harvest Initiative (2014), http://www.globalharvestinitiative.org/Documents/Motes%20-%20Modern%20Agriculture%20and%20Its%20Benefits.pdf.

food production.[32] And yet we have seen a massive increase in the organic food market, presumably motivated by health concerns over pesticides, but largely headed by upper middle class, leading to criticisms that fretting over agricultural chemicals appears to be largely the concerns of luxury.[33]

Admittedly, there is an elitist stance in the pesticide debate.[34] Those who argue against using more agricultural chemicals are often upper middle class individuals who are not involved in food production and can readily purchase organic food or other choices to suit their beliefs. They do not bear the risk of exposure that farm workers endure, and have the luxury of choice when it comes to food. Those privileged individuals can then admonish others who buy cheaper conventional food, saying that the latter are poor decision makers or not concerned with their health, while completely ignoring social context. Accordingly, at best, the American public's recent adoption of organic food preferences is naïve. At worst, it is insulting.

This is a valid critique of chemical concerns, and it follows the earliest traditions of environmental law that was largely written by upper middle class representatives and largely benefiting upper middle class. It is then ironic that those people proliferate public health fears and technophobia when they are the people least likely to be harmed by agriculture's externalities.

However, even if public health concerns are being raised by the unaffected or the hypocritical, such irony does not necessary negate the existence of the issue. Though the developed world has largely bettered its own environmental conditions, it has done so simply pushing hazardous practices and chemicals, such as DDT, to developing nations.[36] So yes, upper middle class individuals can be scolded for being concerned about something that largely does not affect them, but then how does one legitimately disregard concern over the disproportionately affected and economically disadvantaged?

One could start by cross examining some of the other claims made by environmentalists against agricultural chemicals. Regarding endocrine disruptors, environmentalists must account for the failed predication in *Our Stolen Future*, a 1996 book by Theo Colborn, Dianne Dumanoski, and John Peterson, that tragic reproductive consequences would be immediately apparent. The book has since been critiqued for using unsubstantiated assertions of declining sperm counts and other effects and for ignoring the potential harm natural hormones.[37] Also, a report published in *Science*, that reported the synergistic ability of pesticides, was retracted by the study authors because neither the authors nor others could replicate the results.[38] Those

---

[32] *Id.*

[33] Roger Cohen, Op-Ed., *The Organic Fable*, N. Y. TIMES, Sept. 6, 2012.

[34] *Id.*

[36] O'Lear, *supra* note 15.

[37] Christopher Foreman, THE PROMISE AND PERIL OF ENVIRONMENTAL JUSTICE 77-78 (1998).

[38] *Id.*

examples are admittedly, though, bad science, but they do not speak to the recent independent research and figures mentioned above.

To that end, let us consider cancer, one of the main concerns with pesticide use. Cancer death rates overall, despite accounting for over 20% of US deaths[39], are slowing – decreasing and plateauing at best.[40] This certainly does not reflect an imminent doom from cancer inducing agricultural chemicals.[41] In fact, in developing countries, where pesticide use is increasing, cancer incidence rates are still lower than in the USA.[42] This indicates that other factors, such as lifestyle and food choices regarding nutrition, still play a significant role in cancer development. In fact, only 2% of cancer is attributable to pollution, while most of the remaining cancer instances can be explained by higher smoking rates amongst the working class.[43]

Cancer can also appear to be happening more frequently because of rising populations that correlate with advanced technology, such as pesticides. This is a correlation-equals-causation type of confusion. As more people live even longer because of higher living standards and technology, more cancer cases are bound to appear – but because that is happening alongside advancing technology, people attribute cancer to that. Cancer rates have not significantly increased since the beginning of the 1990s so the higher number of cancer cases are incidental to current pesticide and technology, and probably merely reflect a now stable occurrence rate with a higher population.[44]

D. Conclusion on Agricultural Chemicals

There seems to be no epidemic of cancer because of continued pesticide use in the USA, but agricultural chemicals are by no means absolved. The American public may always be suspicious of agricultural chemicals, given our history with DDT and other pesticide scares

---

[39] *Deaths, percent of total deaths, and death rates for the 15 leading causes of death: United States and each State, 2013*, Center for Disease Control (2014), *available at* **http://www.cdc.gov/nchs/nvss/mortality/lcwk9.htm** (following "LCWK9_2013) (26 pages of tables)" hyperlink).

[40] Melonie Heron, *Death: Leading Causes for 2013*, 65 Nat'l Vital Stat. Rpt., Center for Disease Cont. (2016), *available at* http://www.cdc.gov/nchs/data/nvsr/nvsr65/nvsr65_02.pdf.

[41] *See Cancer Facts and Figures*, American Cancer Soc'y, http://www.cancer.org/research/cancerfactsstatistics/allcancerfactsfigures/index.

[42] *Comparing More and Less Developed Countries*, World Cancer Research Fund International, http://www.wcrf.org/int/cancer-facts-figures/comparing-more-less-developed-countries; Ahmedin Jemal et al., *Global Patterns of Cancer Incidence and Mortality Rates and Trends*, 19 CANCER EPIDEMIOLOGY, BIOMARKERS & PREVENTION, issue 8, pp. 1893-907 (2010), http://www.ncbi.nlm.nih.gov/pubmed/20647400.

[43] *See* President's Cancer Panel, *Reducting Environmental Cancer Risk* 2, Nat'l Cancer Inst. (2009), http://deainfo.nci.nih.gov/advisory/pcp/annualReports/pcp08-09rpt/PCP_Report_08-09_508.pdf (explaining that the most quoted estimate of avoidable cancer deaths due to environmental factors is around 2%, but is admittedly out of date and circumspect).

[44] *Id.*

leading to Rachel Carson's seminal work, *Silent Spring*, and the subsequent environmental movement. That is good, though, because pesticides are still an issue for the workers handling it. Environmental quality is often balanced against economic imperatives for more, cheaper food so workers often must resort to using agricultural chemicals, putting them at risk. For example, in 1995 the EPA granted farmers an exception to the 1992 Worker Protection Standard allowing them to continue irrigation, resulting in "minimal contact with pesticide-treated surfaces" for up to eight hours, to avoid economic loss.[45] Therefore the public should be ever vigilant for themselves and farmers. If regulatory standards and practices are keeping the US public safe then, following past experiences, it stands to reason that continued regulatory relaxation could endanger not just more farmers, but possible recreate public health problems of the past. After exploring agricultural chemicals as a public health issue,[46] along with counter arguments against such a diagnosis, let us now shift to those concerns related to GM food.

II. Genetically Modified Food

A. Introduction

Farmers have been artificially modifying the genetics of their plants for 10,000 years, but within the past thirty years a new technological method of plant propagation has emerged. Genetic engineering has introduced plants with phenotypic traits that would have been impossible through cross breeding, or at least have taken countless generations as opposed to one with GM technology, but we have only begun to have GM food since 1996.[47]

Crops have been genetically modified to produce their own pesticide, to be resistant to a pesticide, to produce more of a desired compound, not to produce something such as seeds, to be temperature resistant, and/or to increase shelf life. However, only crops that produce their own pesticide or are resistant to a pesticide are currently marketed on a commercial scale.[48] The type of GM crop that produces its own insecticide is often called a plantincoporatedprotectant.[49] Such a crop often contains a gene from soil bacterium Bacillus thuringiensis (Bt). Bt is toxic to particular insects and protects plants from being destroyed by these pests. Bt corn and cotton have been available since the early 1990s.[50]

---

[45] *Id*. at 81-83.

[46] The foregoing discussion concentrates mainly on the US experience with agricultural chemicals. For a reference to agricultural chemical use and production in other countries, see note VIETNAMNOTE in Chapter 3 of this book.

[47] Jorge Fernandez-Cornejo & Margriet Caswell, *The First Decade of Genetically Engineered Crops in the United States*, 11 ECONOMIC INFORMATION BULLETIN (Apr. 2006), http://www.ers.usda.gov/publications/eib-economic-information-bulletin/eib11.aspx#.VDwoghbjnbw.

[48] *See* Tadlock Cowan, *Agricultural Biotechnology: Background and Recent Issues*, Congressional Research Service Reports Paper 27 (Jan. 2010), *http://digitalcommons.unl.edu/cgi/viewcontent.cgi?article=1026&context=crsdocs.* This report is available also in Schneider, *supra* note 11.

[49] Schneider, *supra* note 11, at 537.

[50] *Id.* at 536.

Despite this difference in food production, GM plants have come across relatively little resistance (at least when compared to the coordinated backlash against pesticides), as they have been admitted into the USA, and then into the world diet over the past two decades. GM animals, by contrast, have yet to be accepted as food items; a GM salmon was not approved for consumption during the summer of 2011, but efforts to get it approved still continue.[51] Some observers hope that animals will be genetically modified to reach market weight faster, produce less methane in their excrement, and be resistant to neurological diseases.[52]

With a new way of producing food, there might be new health concerns, legitimate or otherwise. Some scientists, environmentalists, and consumers have concerns over possible environmental and Trojan gene hazards of genetic engineering,[53] but for now this article focuses on the possible health effects resulting directly from GM technology. Regarding that, the same aforementioned groups attribute genetic modification to increased allergenicity, exposure to novel and hazardous proteins, animal physical abnormalities that translate to poor human health, and in extreme cases cancer, organ atrophy, and immune disruption.[54]

On the other hand, scientists, business interests, and other GM advocates dismiss those arguments as being unscientific and irrational,[55] and/or argue instead that GM food could be the next step in the Green Revolution, now called Gene Revolution, to help Africa achieve food security.[56] Such a Revolution could possibly provide edible vaccines or more nutrient rich crops such as a golden rice variety with more vitamin A.[57]

An overview of GM issues should begin by looking at the history of GM technology for some context, examining each of the concerns the anti-GM coalition shares, and then secondly

---

[51] Andrew Pollack, *An Entrepreneur Bankrolls a Genetically Engineered Salmon*, N.Y. TIMES, May 21, 2012, at B1.

[52] Cowan, *supra* note 48.

[53] See, e.g., various entries on the website of The Nature Institute (http://natureinstitute.org), under its project titled "Unintended Effects of Genetic Manipulation".

[54] See, e.g., Jeffrey Smith, SEEDS OF DECEPTION 12 (2003); VG Pursel et al., *Genetic Engineering of Livestock*, 244 SCIENCE, issue 4910, pp. 1281-88 (1989) (discussing high incidences of pigs having gastric ulcers, arthritis, cardiomegaly, dermatitis, and renal disease after being exposed to bovine growth hormone of genetically modified origin at elevated levels), http://www.sciencemag.org/content/244/4910/1281.short; *GE Foods*, Center for Food Safety, http://www.centerforfoodsafety.org/issues/311/ge-foods (last visited Oct. 13, 2014); *Genetic Engineering*, Greenpeace (2014), http://www.greenpeace.org/international/en/campaigns/agriculture/problem/genetic-engineering/.

[55] *Commonly Asked Questions about the Food Safety of GMOs*, Monsanto (2014), http://www.monsanto.com/newsviews/Pages/food-safety.aspx.

[56] Felicia Wu & William Butz, THE FUTURE OF GENETICALLY MODIFIED CROPS, LESSONS FROM THE GREEN REVOLUTION (2004).

[57] *Id.*

look at the science behind the views related to GM food's health effects. I will be unable to examine health concerns over eating GM animals, though, as there have been no peer reviewed studies examining this and no GM animal has of yet been approved for consumption – so there has been no known public exposure.

B. A Genetically Modified History

The genetic modification of organisms began in 1973 in the U.S. with transgenic bacteria created for biomedical research – although it was not until 1996 that foods derived from genetically modified organisms were commercially available.[58] The next GM crops developed were the "Roundup Ready" soy and corn plants, genetically altered to resist pests, diseases, or herbicides used to destroy weeds. "Roundup Ready" refers to the crops being engineered to resist Monsanto's glyphosate herbicide, marketed as "Roundup."[59]

From 1996 to 2000, 75% of US agricultural biotechnology patents went to commercial interests.[60] In 2008 the US controlled half (sixty two and a half million hectares) of the GM market with an ever increasing share of markets in developing countries.[61] Meanwhile, Argentina had 16.8%, Brazil had 12.6%, Canada and India each had 6.1%, and China controlled 3% of all GM cropland.[62] There are currently at least 156 GM food varieties approved by the US Food and Drug Administration, encompassing nearly all of the US soy, corn, canola, and sugar beets.[63] Over 70 GM crop varieties have been approved by the Animal and Plant Health Inspection Service of the US Department of Agriculture (USDA).[64] "Ninety-one percent of all U.S. soybean, 88% of all upland cotton, and 85% of all corn acres were planted with GE seed varieties in 2009", according to one source.[65] The table in Box #2 shows the growth of GM crop production over recent years, and the graph in Box #3 presents a visual representation of the largest GM crops by market share.

---

[58] Fernandez-Corejo, *supra* note 47.

[59] Schneider, *supra* note 11.

[60] Fernandez-Cornejo, *supra* note 47.

[61] Margaret Grossman, *Protecting Health, Environment and Agriculture: Authorisation of Genetically Modified Crops and Food in the United States and the European Union*, 14 DEAKIN LAW REVIEW, issue 2, pp. 257-304 (2009), http://search.informit.com.au/documentSummary;dn=952952716396616;res=IELHSS.

[62] Cowan, *supra* note 48.

[63] *Biotechnology Consultations on Food from GE Plant Varieties*, U.S. Food and Drug Administration (July 21, 2014), http://www.accessdata.fda.gov/scripts/fcn/fcnNavigation.cfm?rpt=bioListing&displayAll=false&page=1.

[64] Cowan, *supra* note 48.

[65] *Id.*

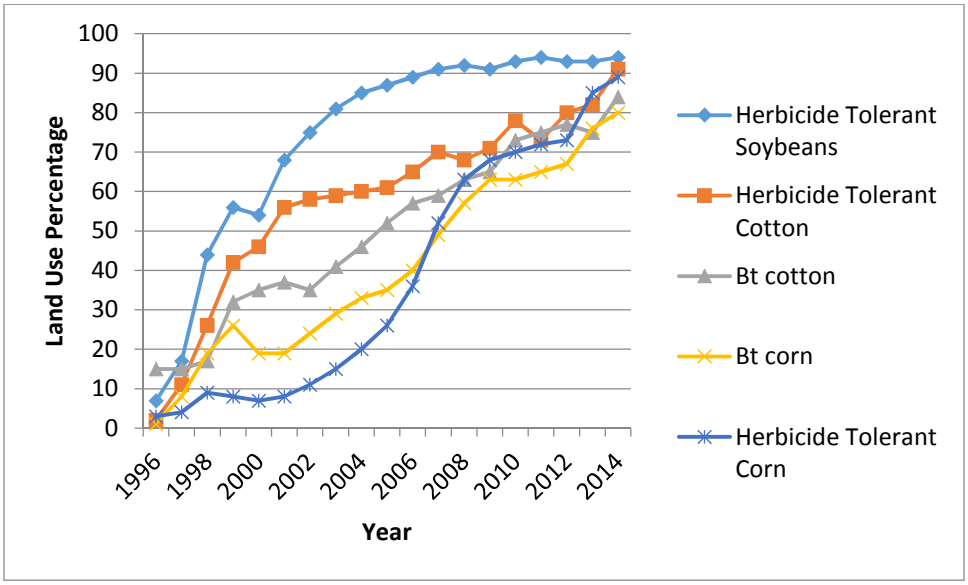
Box #2. Growth of GM Crop Production in the USA (table) [66]

US GM Crops - as a percentage of all planted acres - 1996-2014					
	Herbicide Tolerant Soybeans	Herbicide Tolerant Cotton	Bt cotton	Bt corn	Herbicide Tolerant Corn
1996	7	2	15	1	3
1997	17	11	15	8	4
1998	44	26	17	19	9
1999	56	42	32	26	8
2000	54	46	35	19	7
2001	68	56	37	19	8
2002	75	58	35	24	11
2003	81	59	41	29	15
2004	85	60	46	33	20
2005	87	61	52	35	26
2006	89	65	57	40	36
2007	91	70	59	49	52
2008	92	68	63	57	63
2009	91	71	65	63	68
2010	93	78	73	63	70
2011	94	73	75	65	72
2012	93	80	77	67	73
2013	93	82	75	76	85
2014	94	91	84	80	89

[66] USDA Economic Research Service, *Adoption of Genetically Engineered Crops in the United States, 1996-2014*, http://www.ers.usda.gov/data-products/adoption-of-genetically-engineered-crops-in-the-us/recent-trends-in-ge-adoption.aspx#.VDcSFBbjnbw.

Box #3. Growth of GM Crop Production in the USA (graph) [67]

US GM Crop Market Percentage, 1996–2014:



With this recent explosive growth in adoption of GM technology, as alluded to earlier, some consumers, scientists, and ecologists worry about allergenicity, exposure to novel and hazardous proteins, animal physical abnormalities that translate to poor human health, and in extreme cases cancer, organ atrophy, and immune disruption. The earliest instance of a public health scare related to GM food was an eosinophilia myalgia syndrome outbreak in the US in the summer and fall of 1989. The outbreak was caused by an L-Tryptophan supplement derived from GM bacteria.[68] Fifteen thousand cases were reported with thirty seven deaths.[69]

The L-Tryptophan supplement was manufactured by Showa Denko K.K. of Japan. Although manufacturing occurred without problems previously, some consumers have since blamed GM technology as the source of the impurities that most likely caused the outbreak.[70] However, those concerns more properly speak to the approval of supplements, not to GM technology, because the industrial process of creating the supplement has largely been officially

---

[67] This graph is drawn from data shown in Box #2, *supra*.

[68] EM Kilbourne et al., *Tryptophan Produced by Showa Denko and Epidemic Eosinophilia-Myalgia* Syndrome, 46 JOURNAL OF RHEUMATOLOGY SUPPLEMENT, pp. 81-8 (Oct. 1996), http://www.ncbi.nlm.nih.gov/pubmed/8895184.

[69] *Information Paper on L-Tryptophan and 5-hydroxyl-L-Tryptophan*, U.S. Food and Drug Administration, Center for Foods Safety and Applied Nutrition (Feb. 2001), http://www.nemsn.org/Articles/FDA-Info.pdf.

[70] See *id.* (discussing the manufacturing and impurities); *see also* Smith, *supra* note 54, at 109-25 (blaming GM technology for EMS).

blamed and not the genetic engineering itself.[71] There is even a possibility that other impurities in the L-Tryptophan supplement could have caused the epidemic, so again there is no certainty that GM technology is culpable.[72]

However, GM apprehensions persisted in 2000 when taco shells nationwide inexplicably contained a GM corn called Starlink.[73] Starlink had not been approved from consumption because Cry9C, the protein it produced to kill insect pests, is similar to known human allergens.[74] International markets suffered as corn bound for Asia was rejected and Kraft Foods promptly recalled more than two and a half million boxes of taco shells.[75] Aventis CropScience, the manufacturer of Starlink, ended up paying one hundred million dollars to take back all of its corn.[76] Even higher costs came from the damage to public perception though.

Numerous individuals complained of adverse reactions from consuming Starlink contaminated taco shells, but the Center for Disease Control did not substantiate any of the reports.[77] However, that did not matter because regulatory science had failed the public's trust. GM food now seemed even riskier considering the failure of substantial safeguards. Starlink had a required six hundred sixty foot buffer zone between it and conventional corn to prevent cross pollination, and Aventis was required to give specific instructions to farmers regarding safe handling of Starlink followed by contracts with the growers to abide by EPA regulations.[78] If a nationwide contamination is still possible with so many precautions, then what will happen if a GM crop acutely toxic to humans is planted? How will another outbreak be prevented? The agribusiness and regulatory reaction to these events has been to downplay the dangers of GM technology.

---

[71] U.S. Food and Drug Administration, *supra* note 69.

[72] Rossanne Philen et al., *Tryptophan Contaminants Associated with Eosinophilia-Myalgia Syndrome. The Eosinophilia-Myalgia Studies of Oregon, New York, and New Mexico*, 138 AMERICAN JOURNAL OF EPIDEMIOLOGY, issue 3, pp. 154-59 (April 1993), http://aje.oxfordjournals.org/content/138/3/154.abstract?sid=d5688ad5-06ae-4090-bd05-770ce807bc23.

[73] Joshua Cannon, *Statutory Stones and Regulatory Mortar: Using Negligence Per Se to Mend the Wall Between Farmers Growing Genetically Engineered Crops and Their Neighbors*, 67 WASHINGTON AND LEE LAW REVIEW, issue 2, pp. 653-93 (2010), http://scholarlycommons.law.wlu.edu/wlulr/vol67/iss2/6/; Elizabeth Hill, *Nature's Harvest or Man's Profit: Environmental Shortcuts in the Deregulation of Genetically Modified Crops*, 44 TEXAS TECH LAW REVIEW, pp. 353-90 (2012), https://litigation-essentials.lexisnexis.com/webcd/app?action=DocumentDisplay&crawlid=1&doctype=cite&docid=44+Tex.+Tech+L.+Rev.+353&srctype=smi&srcid=3B15&key=a714d655fc153b83fa085c35bc4c56be.

[74] Cannon, *supra* note 73.

[75] *Id.*

[76] *Id.*

[77] *Id.*

[78] *Id.*

More recently in 2006, there was another unexplainable contamination of conventional rice by the GM LibertyLink rice.[79] LibertyLink, like Starlink, was also not approved for human consumption, but still managed to appear to the US food market.[80] The USDA did determine that the contamination was not likely because of cross pollination.[81] Just like with Starlink's outbreak, international markets suffered, consumer fears fomented, and retaliatory lawsuits from farmers were filed.[82] Bayer CropScience, the producer of LibertyLink and what ironically previously bought Aventis, ended up paying $750 million to settle the cases against it.[83]

What is important to take from L-Tryptophan, Starlink, and LibertyLink is that contaminations can spread, even with buffers and other safeguards. Such regulatory failure is likely because of the volatile nature of plant genetics that sometimes cannot be accounted for, and the inherent dangers of a vertically integrated food system that can spread pathogens and viruses just as much as genes. Accidental contaminations even continue today, demonstrating that regulatory institutions have failed to adapt to GM technology.[84] However, do not assume that this then incriminates all GM food. There may be valid socioeconomic concerns over GM food that should not be wholly disregarded, but they are ignored for the purposes of properly assessing public health risks only, and as of right now there have been no substantiated, hazardous GM outbreaks. The only instances of toxicity or carcinogenic properties of GM food have been in laboratories, and it is still impossible to assess that work without politics or fear.

C. Politicized Scientific Testing of Genetic Engineering's Safety

In 1998, Arpad Pusztai of the Rowett Research Institute was asked to comment about his research on GM food to the Royal Society. However instead of providing the Society with his data and comments, Pusztai went straight to the media and told the public that his research on a GM potato proved that the GM crop was toxic, and that he would not consume GM products as a result.[85] His research was later published in Lancet, but then retracted. Pusztai received a gag

---

[79] *Id.* at 672-674; Hill, *supra* note 73.

[80] Hill, *supra* note 73.

[81] Cannon, *supra* note 73.

[82] *Id.*

[83] Nicole Cerullo, *Settlement of Genetically Modified Rice Lawsuits with Bayer CropScience*, PR Web, July 4, 2011, http://www.prweb.com/releases/2011/7/prweb8619423.htm.

[84] *See* Roxanne Hegeman, *Monsanto Sued Over Genetically Modified Wheat*, USA TODAY, June 4, 2013, http://www.usatoday.com/story/money/business/2013/06/04/farmer-monsanto-genetically-engineered-wheat/2388957/; *see also Bayer Extends GM Rice Settlement Deadline to November*, Arkansas Democrat-Gazette, Oct. 25, 2011, *http://www.arkansasonline.com/news/2011/oct/25/bayer-extends-gm-rice-settlement-deadline-november/.*

[85] PPG Bateson, *Genetically Modified Potatoes*, 354 LANCET, issue 9187, pp. 1382 (Oct. 1999), http://www.thelancet.com/journals/lancet/article/PIIS0140-6736%2899%2999183-3/fulltext.

order from the Rowett Institute to not comment on his study or opinions, and was suspended.[86] The Audit Committee of the Rowett Institute disavowed Pusztai's conclusions.[87]

However, even with sanctions and official condemnation Pusztai's damage had already been done. Public relations for GM food suffered. Pusztai became a martyr for anti-GM efforts, leading to the creation of conspiracy theories.[88] Pusztai's conclusion led to a backlash with biotechnology companies completely disregarding any parts of Pusztai's work. Unfortunately both sides, in furthering either pro-GM or anti-GM positions, neglect to properly present the situation.

Pusztai tested the toxicity of a potato with a transferred gene to express a snowdrop lectin.[89] Feeding rats for only 10 days with the GM potato significantly reduced lymphocyte responsiveness, reduced the weight of vital organs such as the liver and kidneys, and a reduced rate of digestion leading to malnutrition. However, diets predominantly composed of conventional potatoes would present the same findings due to protein deficiency, thus validating the critiques against Pusztai that his rats were already malnourished, but when Pusztai raised the overall protein levels from sixty one and sixty three g/kg to one hundred forty seven g/kg (more than necessary to sustain healthy growth) the ill results still held.[90]

However, none of this is surprising considering that insertion of the snowdrop lectin gene had concurrently been proven to be toxic, and that lectins can naturally impeded nutrient intake.[91] Furthermore, Pusztai's work should not be used to vilify all GM food or even just GM potatoes because further research on other GM potatoes has found them safe.[92] In sum, the proper response to the Pusztai incident is that his GM potato exhibited signs of toxicity but it has never been approved for human consumption so there is no immediate concern, the signs of toxicity do not necessarily translate to toxicity for humans, Pusztai's reaction was

---

[86] Jonathan Rhodes, *Genetically Modified Foods and the Pusztai Affair*, 318 BRITISH MEDICAL JOURNAL, issue 7193, pp. 1284 (1999), http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1115659/.

[87] *The Audit Committee's Response to Dr Arpad Pusztai's Alternative Report of 22October 1998*, Rowett Research Institute (1999).

[88] *See*, *e.g.,* Smith, *supra* note 54, at 5-44.

[89] *Review of Data on Possible Toxicity of GM Potatoes,* The Royal Society (June 1999), https://royalsociety.org/~/media/Royal_Society_Content/policy/publications/1999/10092.pdf.

[90] *Id.*

[91] KV Rao et al., *Expression of Snowdrop Lectin (GNA) in Transgenic Rice Plants Confers Resistance to Rice Brown Planthopper*, 15 THE PLANT JOURNAL, issue 4, pp. 469-77 (Aug. 1998), http://www.ncbi.nlm.nih.gov/pubmed/9753773.

[92] Wataru Hashimoto et al., *Safety Assessment of Genetically Engineered Potatoes with Designed Soybean Glycinin: Compositional Analyses of the Potato Tubers and Digestibility of the Newly Expressed Protein in Transgenic Potatoes*, 79 JOURNAL OF THE SCIENCE OF FOOD AND AGRICULTURE, issue 12, pp. 1607-12 (1999), http://onlinelibrary.wiley.com/doi/10.1002/%28SICI%291097-0010%28199909%2979:12%3C1607::AID-JSFA408%3E3.0.CO;2-T/abstract.

unprofessional, and that the GM and conventional potato were not substantially equivalent and therefore more research on a case by case basis should have been recommended, as opposed to the actual GM food debate rhetoric as a black and white, all safe or all not safe, spectrum.

One researcher who does advocate case by case scrutiny is Gilles-Eric Seralini from the University of Caen in France. Unfortunately, like Pusztai, politics and misinformation surround him as well. In 2006, he and two other researchers questioned the experiment methodology of Monsanto in 2005 that allowed for the approval of a GM corn called MON 863. When they tried to replicate the results and statistics Seralini et al. found that MON 836 had a dose and gender specific effect on rats over a ninety day feeding period.[93] For male rats there was a 3.3% decrease in weight and 3.7 for female, signs of kidney toxicity were observable in 24% to 40% of females and in 31% to 35% of males.[94] Of the two experimental groups, both experienced these symptoms after being a fed a diet of either 11% or 33% MON 863, but the latter group experienced the symptoms first.[95] It was thought that these signs of toxicity are because of pesticide residues excreted by the GM corn itself.

In response, John Doull et al. reevaluated Seralini's works compared to the tests at Monsanto and concluded that MON 863 is actually safe.[96] Doull et al. disagreed with Seralini's methodology of performing different statistical tests than those performed by Monsanto, and repeatedly pointed out that Seralini did not use reference weights from control groups to analyze the experimental rats.[97] Doull et al.'s conclusions are in accordance with other studies in European Union, German, French, and Philippine regulatory agencies that also reevaluated Seralini's work.[98]

However, Seralini appears to have not been dismayed by rebuttals because he continues to publish papers to support his claim that some GM crops show signs of toxicity and therefore that GM food should be considered on a case by case basis for regulatory approval. In 2009 Seralini, as part of Spiroux de Vendomois et al., performed rat feeding trials of GM corn MON 810, MON 863, and NK 603 for fourteen weeks.[99] Those GM corn varieties had already been

---

[93] Gilles-Eric Seralini et al., *New Analysis of a Rat Feeding Study with a Genetically Modified Maize Reveals Signs of Hepatorenal Toxicity*, 52 ARCHIVES OF ENVIRONMENTAL CONTAMINATION AND TOXICOLOGY, issue 4, pp. 596-602 (2007) [hereinafter Seralini-2007], http://www.ncbi.nlm.nih.gov/pubmed/17356802.

[94] *Id.*

[95] *Id.*

[96] John Doull et al., *Report of an Expert Panel on the Reanalysis by Seralini et al. (2007) of a 90-day Study Conducted by Monsanto in Support of the Safety of a Genetically Modified Corn Variety*, 45 FOOD AND CHEMICAL TOXICOLOGY, issue 11, pp. 2073-85 (2007), http://www.ncbi.nlm.nih.gov/pubmed/17900781.

[97] *Id.* at 2079-2082.

[98] *Id.* at 2083.

[99] Joel Spiroux de Vendomois et al., *A Comparison of the Effects of Three GM Varieties on Mammalian Health*, 5 INTERNATIONAL JOURNAL OF BIOLOGICAL SCIENCES, issue 7, pp. 706-26 (2009), http://www.ijbs.com/v05p0706.htm.

approved for consumption, and this was the longest in vivo test on GM food performed on mammals so this study was meant to carry more weight than previous work.[100]

The Spiroux de Vendomois et al. study followed Seralini's previous methodology and concluded that all of the GM corn showed signs of toxicity.[101] The gender and dose related effects also held. Males were more sensitive to NK 603 with 83% of kidney toxicity reported in the group fed a 33% GM corn diet at week fourteen of the experiment.[102] Males also experienced the most heart and liver weight gain on the NK 603 diet. The effects of MON 863 were also similar to NK 603 with males expressing more signs of toxicity.[103] Females appeared to more sensitive to MON 810 with similar signs of toxicity as in the NK 603 groups.

Spiroux de Vendomois et al. concluded with the strong words that, "the tendency for physiological disturbance is characteristic of almost all rats of all GM-fed treatment groups, and physio-pathological profiles differ according to dose or sex."[104] The authors then ended with recommendations that more studies be performed, and a critique of two studies by Bruce Hammond, from Monsanto, that concluded that GM corn varieties MON 810 and MON 88017 were safe.[105] There appears to be no formal response to Seralini's 2009 study, but Hammond's research on MON 88017 was subsequently, independently substantiated.[106]

Following Spiroux de Vendomois et al.'s study, Seralini once again looked at NK 603 using the same methodology of rat feeding trials, but this time held trials for two years.[107] The results confirmed Seralini et al.'s previous suspicions of renal toxicity, but this time tumors were exhibited by nearly all of the rats.[108] Seralini et al. supposed that the glyophosphate produced by

---

[100] *Id.* at 707.

[101] *Id.* at 706.

[102] *Id.* at 709-711.

[103] *Id.* at 711-713.

[104] *Id.*

[105] *Id.* at 713-717; Charles Healy et al., *Results of a 13-Week Safety Assurance Study with Rats Fed Grain from Corn Rootworm-Protected, Glyophosphate-Tolerant MON 88017 Corn*, 46 FOOD AND CHEMICAL TOXICOLOGY, issue 7, pp. 2517-2524 (2008), http://www.ncbi.nlm.nih.gov/pubmed/18492601; Bruce Hammond et al., *Results of a 90-Day Safety Assurance Study with Rats Fed Grain from Corn Borer-Protected Corn*, 44 FOOD AND CHEMICAL TOXICOLOGY, issue 7, pp. 1092-1099 (2006) [hereinafter Hammon-2006], http://www.ncbi.nlm.nih.gov/pubmed/16487643.

[106] Yann Devos et al., *Bt-Maize Event MON 88017 Expressing Cry3Bb1 Does Not Cause Harm to Non-Target Organisms*, 21 TRANSGENIC RESEARCH, issue 6, pp. 1191-24 (2012), http://www.ncbi.nlm.nih.gov/pubmed/22576225.

[107] Gilles-Eric Seralini et al., *Long Term Toxicity of a Roundup Herbicide and a Roundup-Tolerant Genetically Modified Maize*, 30 FOOD AND CHEMICAL TOXICOLOGY, issue 11, pp. 4221-31 (2012) [hereinafter Seralini-2012], http://www.sciencedirect.com/science/article/pii/S0278691512005637.

[108] *Id.* at 4225-4229.

NK 603, despite being below authorized levels, was responsible for the signs of toxicity, tumors, and high mortality.[109] Just as in 2006 study, there was an almost immediate backlash from those in the scientific community.

This time scientists, and governing bodies such as the European Food Safety Authority and Germany's Federal Institute for Risk Assessment, are asking that Seralini release the rest of the data used in his latest experiment, but Seralini counters that he will not release his data until the European Food Safety Authority releases all of the information used in the regulatory approval of NK 603.[110] Seralini released the information to only a select group of journalists, but each one had to sign an unusual confidentially agreement preventing them from discussing the paper with other scientists until the embargo period on his study ended.

Seralini has however released a book and a film about his study. Having such materials already made for distribution so soon after finishing a study leads one to infer that he already knew what his study was going to conclude long before it was completed or worse, that it was predetermined.[111] In addition, Butler points out that Seralini used Sprague-Dawley rats, which are predisposed to developing tumors, and that those rats was improper in a two year study because those rats only have a two year life span.[112] Also, counter to Serlaini's contention that he followed Organization for Economic Co-operation and Development (OECD) guidelines,[113] Seralini only used ten rats per group as opposed to the sixty five or more that are required when using rats with such inherently high mortality rates.[114]

Hammond and other critics voice those same concerns, point to lack of information, using mismatched statistical methods, and stating conclusions on GM corn and glyophosphate that have no basis based on Seralini et al.'s low statistical power.[115] Hammond points out, along with the previously mentioned violation of OECD test guidelines and incomplete data presentation, that Seralini actually changed the purpose of the study from just testing toxicity to carcinogenicity.[116] A reasonably prudent scientist, if she needed to change her test parameters

---

[109] *Id.* at 4230.

[110] Declan Butler, *Hyped GM Maize Study Faces Growing Scrutiny*, 490 NATURE pp. 158 (2012), http://www.nature.com/news/hyped-gm-maize-study-faces-growing-scrutiny-1.11566.

[111] *See id.*

[112] *Id.*

[113] Seralini-2012, *supra* note 107, at 4222-4223.

[114] Butler, *supra* note 110.

[115] Bruce Hammond et al., *Letter to the Editor*, 53 FOOD AND CHEMICAL TOXICOLOGY, pp. 459-64 (2013) [hereinafter Hammond-2013], http://www.sciencedirect.com/science/article/pii/S0278691512007892; David Tribe, *Letter to the Editor*, FOOD AND CHEMICAL TOXICOLOGY, pp. 467-72 (2013), http://www.sciencedirect.com/science/article/pii/S0278691512007879; Dung Tien & Ham Huy, *Comments on "Long Term Toxicity of a Roundup Herbicide and a Roundup-Tolerant Genetically Modified Maize*, FOOD AND CHEMICAL TOXICOLOGY, pp. 443-44 (2013), http://www.sciencedirect.com/science/article/pii/S0278691512007995.

[116] Hammond-2013, *supra* note 115.

would have started over as opposed to changing the experiment midway. Otherwise it looks like a scientist is skewing results or the experiment to fit a desired result. Other scientists concur with those criticisms and bring up other statistical errors.[117] The European Food Safety Authority ended up rejecting Seralini's study.[118]

D. Conclusion on Genetically Modified Foods

Studies that hint at GM food's toxicity (to humans) have largely been discredited. One might then reasonably conclude that GM food is not a public health concern regarding its consumption. This is not to say, though, that GM food is not concerning at all.[119] For one thing, pesticide use has increased because of GM crops. In addition, some observers would argue that the practice of patenting genes is economically disadvantageous for farmers.

Moreover, the Nature Institute has developed extensive information and commentary about various non-human-health effects of GM crops,[120] reflecting the view that a consideration of the effects of GM food crops should not be exclusively anthropomorphic in nature. That is, instead of looking only at the effects GM development has or might have on humans, we should evaluate also the effects that GM development might have on other species and on the non-living components of the ecosphere.

However, if for present purposes we concern ourselves only with human-health concerns that come from a supposed danger inherent to GM technology, we can conclude at least tentatively that such a danger has not been seen. Meta-analyses of peer reviewed studies looking at GM food toxicity confirm this inference.[121]

---

[117] Tribe, *supra* note 115; Tien & Huy, *supra* note 1155.

[118] Hammond-2013 et al., *supra* 115.

[119] In fact, Frederick Kirschenmann has offered a note of caution on this point relating to genetic engineering. Noting that it (and other approaches) might "have the potential to produce positive effects", he says remains true that agriculture "is based on complexities that cannot be readily managed in large-scale monocultures". In this regard, he notes that "Richard Manning has concluded that we will never be successful in our efforts to feed the world if we do not take the complexity and diversity of local cultures and local ecologies into consideration." Kirschenmann goes on to report that Manning concludes that while genetic engineering might provide a limited tool in food production for an expanding human population, it will not be the solution". Kirschenmann quotes Manning:

> The genetic engineering business is going to get all the headlines, but these simple matters [attending to the needs of local cultures and local ecologies] are potentially far more earth-shaking. What must happen, and to a degree is happening, in agriculture is also an information revolution. If there was a key mistake of the green revolution, it was in simplifying a system that is by its very nature complex.

Kirschenmann, *supra* note 8, at 175 (quoting Richard Manning's 2000 book *Food's Frontier: The Next Green Revolution*).

[120] See, e.g., Craig Holdrege, *Understanding the Unintended Consequences of Genetic Manipulation* (2008), on the website of the Nature Institute at http://natureinstitute.org/txt/ch/nontarget.php.

[121] Jose Domingo & Jordi Bordonaba, *A Literature Review on the Safety Assessment of Genetically Modified Plants*, 37 ENVIRONMENT INTERNATIONAL , issue 4, pp. 734-42 (2011), http://www.researchgate.net/publication/49812743_A_literature_review_on_the_safety_assessment_of_genetically_modified_plants.

Still, it is important to emphasize that most of the studies that exonerate GM foods either (i) are funded by biotech companies that make GM products or (ii) are performed by their researchers.[122] There is a lack of truly independent review, and this obviously could be a problem should any dangerous GM product be made. Thus, GM research is like pharmaceutical studies that are more likely to report positive results for their sponsors than independently funded studies.[123] This reality becomes especially concerning considering that Monsanto, the largest GM crop producer, recently had many of its studies verifying the safety of its products retracted because of the corrupt practices of contracted laboratories.[124]

Despite this, at the present time the idea of a GM danger to human health is still mere speculation. At the moment, genetically modified crops are not a public health danger in of themselves, and public health advocates would be better off targeting other risks – such as the substantiated outbreaks of food borne diseases explored in the following section.

III. Food Borne Illness and Health

Food borne illnesses constitute a serious public health concern and cost. It is estimated that up to 76 million such illnesses, including 325,000 hospitalizations, and 5,000 deaths, occur in the USA each year, costing $10 million to $80 billion each year.[125] As illnesses that by definition come from food, they are inextricably related to the agricultural practices that made that food. There are many factors that come into play between the field and the plate, such as poor food handling, cooking, and shipping, but as with most problems we should first approach the ultimate source – the bacteria that instigate the illnesses. My survey will start with Escherichia coli (E coli), a pathogen that has recently received a lot of media attention, as well as Salmonella, Campylobacter, and Listeria, exploring why they are health concerns related to agriculture and why they became so. Finally, this note will then compare those pathogens to other public health risks to determine the extent of concern that food borne illnesses should elicit.

A. Food borne Pathogens and Antibiotic Resistance – Escherichia coli

E coli, specifically the genotype O157:H7, is a gram negative, pathogenic bacterium frequently dispersed throughout via the food supply. Symptoms of an E coli infection include diarrhea, cramping, hemorrhagic colitis, sepsis, and possibly fatal hemolytic uremic syndrome

---

[122] *Id.* at 734.

[123] Behrokh Maghari & Ali Ardekani, *Genetically Modified Foods and Social Concerns*, 3 AVICENNA JOURNAL OF MEDICAL BIOTECHNOLOGY, issue3, pp. 109-17 (2011), http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3558185/.

[124] *Testing Fraud: IBT and Craven Laboratories*, Monsanto (June, 2005), http://www.monsanto.com/products/Documents/glyphosate-background-materials/ibt_craven_bkg.pdf.

[125] David Nyachuba, *Foodborne Illness: is it on the Rise?*, 68 NUTRITION REVIEWS, issue 5, pp. 257-69 (2010), http://www.ncbi.nlm.nih.gov/pubmed/20500787. For a reference to estimates showing more than 46 million foodborne illnesses, see *CDC 2011 Estimates: Findings*, Centers for Disease Control (Jan. 8, 2014), http://www.cdc.gov/foodborneburden/2011-foodborne-estimates.html.

affecting kidney function.[126] More than 80% of urinary tract infections worldwide are caused by E coli,[127] but even worse are those extremely rare scenarios involving intestinal bacterial infections leading to blood clots that travel to the brain causing seizures or paralysis.[128] Hamburgers and other meats are closely associated vectors for the pathogen as it develops in the digestive tract of livestock, but recent recalls of infected spinach attest to possible alternative carriers.[129] Produce likely becomes contaminated with fecal matter when excrement is used as fertilizer, as a result of rain runoff, or when food handlers fail to exercise proper hygiene practices.

The US public is much more aware of E coli since the winter of 1993. Jack in the Box had been alerted by the Washington Health State Department in January that an E coli outbreak had been traced to their burgers. Four hundred cases were reported throughout Washington, Idaho, and Nevada, but the most significant public scare was the death of three toddlers in the Seattle area attributed to Jack in the Box hamburgers.[130] Twenty thousand pounds of tainted meat were recalled and Jack in the Box responded to the public relations nightmare by passing the blame onto their meat suppliers, competing businesses who also possibly served E coli laced meat, and lax Washington state health officials. They also capitalized on the crisis by emphasizing their newfound commitment to proper cooking, cleaning, and demanding that their suppliers also comply with stricter screening and health standards.[131] Jack in the Box successfully went from foul food provider to public health crusader, saving its market and educating the public on one of the problems involved in the US food system.

Since the Jack in the Box episode, the Center for Disease Control estimates that there are 2,268 O157-type illnesses each year.[132] With so many cases, E coli is a main contributor to the ten to eighty three billion dollars spent each year in the USA on foodborne illness treatment.[133]

All foodborne illness costs are incurred by a vertically integrated food, increasingly globalized food system, more dining outside the home, microbial antibiotic resistance, and an

---

[126] Ross Lowe, et al., *Factors Influencing the Persistence of Escherichia coli O157:H7 Lineages in Feces from Cattle Fed Grain Versus Grass Hay Diets*, 56 CANADIAN JOURNAL OF MICROBIOLOGY, issue, pp. 667-675 (2010), http://www.nrcresearchpress.com/doi/abs/10.1139/w10-051#.VD04Vxbjnbw.

[127] Caroline Vincent et al., *Food Reservoir for Escherichia coli Causing Urinary Tract Infections*, 16 EMERGING INFECTIOUS DISEASES, issue 1, pp. 88-95 (2010), http://wwwnc.cdc.gov/eid/article/16/1/09-1118_article.

[128] Michael Moss, *The Burger that Shattered Her Life*, N.Y. TIMES, Oct. 3, 2009, at A1.

[129] Lowe, *supra* note 126, at 668.

[130] *Crisis Communication Strategies*, U.S. Department of Defense (June 28, 2002), http://www.ou.edu/deptcomm/dodjcc/groups/02C2/Jack%20in%20the%20Box.htm.

[131] *Id.*

[132] *See CDC 2011 Estimates*, *supra* note 125.

[133] Nyachuba, *supra* note 125, at 257.

increasing vulnerable portion of the consumer population.[134] Therefore treating E coli may end up preventing Listeria and Salmonella, more morbid pathogens, leading to an overall healthier public.

A large scale food production system contributes to E coli proliferation by increasing the speed of meat production, inevitably leading to more excrement inadvertently leaking onto meat in the rushed removal of digestive tracts during butchering, including the ground flesh from several hundred cows to make many burgers instead of just one,[135] and the complex nature of centralized production making oversight and tracing food origins increasingly difficult and pathogenic impacts more significant.[136] The US food production model also calls for economies of scale meaning livestock are fed cheap commodity crops such as corn. This is problematic because corn based diets acidify the stomachs of ruminants, encouraging E coli cultures to grow.[137]

Restaurant dining poses the additional risk of not being in control of the cooking process, possibly exposing patrons to unsafe handling practices, and the prolific use of antibiotics in agriculture creates an environment where E coli in livestock are evolving into strains similar to those found in humans.[138] This is actually how O157:H7, the most problematic strain, developed. Finally, changing US demographics, nearly 20% of the population being over the age of 65 by 2030, signifies an increasing sector that will suffer more from E coli outbreaks and whose treatment will cost more.[139]

B. Salmonella and Campylobacter

Salmonella is a gram negative, rod shaped bacterium whereas Campylobacter is a gram negative, spiral shaped bacterium. They are both often associated with poultry[140], and infections often display the same symptoms, so for analytical purposes they are considered together, staring with Salmonella.

---

134 *Id.* at 258-262.

135 Carl Schroeder et al., *Isolation of Antimicrobial-Resistant Escherichia coli from Retail Meats Purchased in Greater Washington, DC, USA*, 85 INTERNATIONAL JOURNAL OF FOOD MICROBIOLOGY, issue 1-2, pp. 197-202 (2003), http://www.sciencedirect.com/science/article/pii/S0168160502005081..

136 Moss, *supra* note 128.

137 Lowe, *supra* note 126, at 671-673.

138 Nyachuba, *supra* note 125, at 261-262.

139 *Id.* at 262.

140 Although Campylobacter has just recently found to persist in cow manure meaning that it shares many of the same health concerns and issues related to E. coli described above. G. Inglis et al., *Prolonged Survival of Campylobacter Species in Bovine Manure Compost*, 76 APPLIED AND ENVIRONMENTAL MICROBIOLOGY, issue 4, pp. 1110-19 (2010), http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2820969/.

There are actually three types of Salmonella. There is Salmonella enteritidis that is then split into phage type four (Europe) and phage type eight (North America), both of which are associated with raw or undercooked eggs.[141] The third type is Salmonella typhimurium that is found in the guts of pigs, sheep, and cattle.[142] Salmonella is found in about 40.3% of ground sausage, 46% of ground beef, and 56.3% of poultry.[143] This does not guarantee infection, and proper cooking eliminates most Salmonella strains, but it still means that a consumer has about a substantial chance of buying Salmonella contaminated meat.[144]

Salmonellosis, a Salmonella infection, is often exhibited by diarrhea, fever, and abdominal cramps twelve to seventy two hours after infection, lasting for four to seven days.[145] Most people recover without complications so there is probably substantial underreporting of cases.[146] The CDC estimates that there are actually about 1,027,561 Salmonella related illnesses in the USA each year.[147] Still, only a small fraction has to be hospitalized each year (19,336), and of those there are only a supposed 378 deaths per year from salmonellosis. Salmonella is spread via the vertically integrated food system that I discussed in the E. coli section so I need not elaborate on that more, and can instead turn to Campylobacter.

People who contract campylobacteriosis, like salmonellosis, experience diarrhea, cramping, abdominal pain, and fever.[148] Symptoms have a much more delayed appearance though of two to five days after exposure. Campylobacter is found in the gastrointestinal tracts of farm animals, but it appears to be the most adapted to avian guts.[149] This is probably why Campylobacter and Salmonella are often coupled. Roughly 13% of fresh pork is contaminated with Campylobacter, 27% of cattle, and, 66.2% of poultry.[150] Campylobacter has also been known to be found in raw milk, untreated surface water, and pets.[151]

---

[141] M. Teuber, *Spread of Antibiotic Resistance with Food-Borne Pathogens*, 56 CELLULAR AND MOLECULAR LIFE SCIENCES, issue 9-10, pp. 755-763 (1999), http://link.springer.com/article/10.1007/s000180050022#page-1.

[142] *Id.*

[143] *Id.*

[144] *Id.*

[145] *What is Salmonellosis*, Center for Disease Control and Prevention (Apr. 5, 2012), http://www.cdc.gov/salmonella/general/index.html.

[146] *Id.*

[147] *CDC 2011 Estimates*, *supra* note 125.

[148] *Campylobacter*, Center for Disease Control and Prevention, National Center for Emerging and Zoonotic Infectious Diseases (June 3, 2014) [hereinafter CDC-Campylobacter], http://www.cdc.gov/nczved/divisions/dfbmd/diseases/campylobacter/.

[149] Teuber, *supra* note 141, at 758.

[150] *Id.*

[151] *Id.*

However, even with a startling dispersal rate, resulting diseases and death are not as astonishing. The US experiences about 845,024 Campylobacter infections each year, with 8,463 hospitalizations, but only seventy six deaths, which probably reflecting those with weakened immune systems or other people with other predispositions such as pregnant mothers and the elderly.[152] These infections are also facilitated via the already elaborated on centralized food production system.

C. Listeria

Listeria monocytogenes has only just emerged as a major concern in microbiology over the past decade.[153] It is a rod shaped, gram positive bacterium[154] that can cause meningitis, septicemia, and even abortion, coupled with a relatively high fatality rate around 20–30%.[155] Listeria is actually quite ubiquitous, being found in everything from vegetables to water, but it is usually found in meat, especially raw and ready-to-eat seafood products.[156]

There were Listeria scares in 1998 and 1999 related to read-to-eat meats made by a Sara Lee subsidiary.[157] The USDA and meat industry officials have since countered that products recalled because of Listeria have declined from forty in 2002 to fourteen in 2003.[158] Listeria has not continued this declining trend in recent years, though.[159] Also, the problem goes beyond meat because the latest Listeria outbreaks have been with cantaloupe out of Colorado in 2011 and ricotta cheese from Italy in 2013.[160] Listeria's persistence is because of its spread via the same vertically integrated food system described earlier, and because Listeria is found on most surfaces. Therefore, a colony can establish itself simply because of poor hygiene and cleaning practices. Listeria then follows the same concerns and pathways I emphasized with E. coli, Salmonella, and Campylobacter, but Listeria is much deadlier.

---

152 CDC-Campylobacter, *supra* note 148.

153 Teuber, *supra* note 141, at 759.

154 *Listeria (Listeriosis)*, Center for Disease Control and Prevention (July 29, 2014) [hereinafter CDC-Listeria], http://www.cdc.gov/listeria/ (follow "Causes" hyperlink).

155 Teuber, *supra* 153.

156 *Id.*

157 Schneider, *supra* note 11, at 637.

158 *Id.*

159 *Id.*

160 CDC-Listeria, *supra* note 154 (follow "Outbreaks" hyperlink).

It is the substantially higher fatality rate that leads to Listeria being portrayed as a health crisis in popular media outlets.[161] It probably deserves it though since it accounts for most of the resulting estimated deaths from food borne illnesses in the USA. However, only an estimated 1,600 Americans contract listeriosis each year.[162] Of those, the majority are hospitalized.

D. Antibiotic Resistance in General

When considering food borne illnesses and their origins in modern agriculture, one must also consider the tools used to combat those bacteria responsible – antibiotics. The development of antibiotics in the 1940s has indisputably been a boon for humanity. However it has also allowed for the intensification of animal agriculture, and therefore consequentially, antibiotics are partly responsible for the public health concerns detailed in this appendix.[163] There is even a building body of scientists, doctors, and consumers who fear that antibiotics pose a public health danger in of themselves by creating antibacterial resistant pathogens through their overuse.

E. The Current State of Antibiotic Resistance

It is now estimated that 70% of all antibiotics in the USA are administered to farm animals.[164] This sustains livestock in often disease ridden, unsanitary conditions, and encourages weight gain.[165] Prescriptions are now required, but up until 2012 agricultural operators had been able to indiscriminately administer antibiotics without prescriptions.[166] All of this is done without actually approaching the question why we should perpetuate a food production system with so many negative externalities.

Antibacterial resistance is created in the guts of animals as they are administered the antibiotics in their feed. When an antimicrobial is used it will eliminate living bacterial cultures, sometimes by disrupting cell wall construction or the conversion of glucose into energy, but either way those bacteria die. However, sometimes a small amount may survive because they acquired a gene, via mutation or plasmid transfer, that prevents the antibacterial's effectiveness. Those bacteria can then reestablish a new population with a complete resistance to the previously used antimicrobial. In small amounts this evolution should not be a problem, and would in no

---

[161] See William Neuman, *Listeria Outbreak Traced to Cantaloupe Packing Shed*, NEWYORK TIMES, Oct. 19, 2011, at B1.

[162] *National Enteric Disease Surveillance: The Listeria Initiative*, Center for Disease Control and Prevention, National Center for Emerging and Zoonotic Diseases, Jan. 10, 2013, http://www.cdc.gov/listeria/pdf/ListeriaInitiativeOverview_508.pdf.

[163] Blatt, *supra* note 26, at 117; Ulrike Thoms, *Between Promise and Threat, Antibiotics in Foods in West Germany 1950 – 1980*, 20 NTM, issue 3, pp. 181-214 (2012), http://link.springer.com/article/10.1007%2Fs00048-012-0073-x/fulltext.html.

[164] Blatt, *supra* note 26, at 159.

[165] Thoms, *supra* note 163, at 188-189.

[166] Gardiner Harris, *U.S. Tightens Rules on Antibiotics Use for Livestock*, N.Y. TIMES, Apr. 11, 2012, at A19.

way outpace the progress of antibiotics, but with a continued high use of antibiotics the chance of fully established antibiotic resistant bacteria becomes a real threat.

Relating back to agriculture, resistant bacteria can transfer from the intestines of livestock to retail meat through fecal contamination, and continued handling of animal tissues.[167] Raw milk samples have shown contamination of food borne pathogens at 50%, and egg shells will inevitably be contaminated with fecal bacteria during the laying process.[168] We have also known since 1959 that bacteria can share resistance genes via plasmid transfers.[169] This means that antibiotic resistance can transfer to bacteria in our own guts, increasing the risk of harm to consumers by making a once harmless disease immune to treatment. You will notice that many of the public health risks and disease vectors between antibiotic resistance and the bacteria discussed above are the same. That is because many of the bacteria discussed above have begun to exhibit antibiotic resistance, thus amplifying health concerns.[170]

A study in 2002 reported that there was a startling antibiotic resistance amongst four hundred seventy two E coli isolates taken from ground and whole retail beef, chicken, pork, and turkey in Washington, DC from 1998 to 2000.[171] Large resistance rates included the 59% resistant to tetracycline, 45% to sulfamethoxazole, 44% to streptomycin, 38% to cephalothin, and 35% to ampicillin.[172] Whereas lower resistance rates included the 12% resistant to gentamicin, 8% to nalidixic acid, 6% to chloramphenicol, 4% to ceftiofur, and 1% to ceftriaxone.[173] About 16% of the sample showed resistance to only one antimicrobial, while 23% were resistant to two, another 23% to three, 12% to four, 7% to five, 3% to six, 2% to seven, and finally 2% to eight. Luckily only three of the E coli isolates contained Shiga toxins, but two of those had acquired antibacterial resistance, one being resistant to six microbials.[174]

---

[167] Schroeder et al., *supra* note 135, at 197; Teuber, *supra* note 141, at 756.

[168] Teuber, *supra* note 141.

[169] Thoms, *supra* note 163, at 199.

[170] In addition to the specific instances of antibiotic resistance referred to below – focusing on E Coli, salmonella, and campylobacter – a wide range of other pathogens have developed resistance to antibiotics. They include *Clostridium difficile*, *Neisseria gonorrhoeae*, *Acinetobactor*, *Candida*, extended-spectrum β-lactamase-producing *Enterobacteriaceae*, *Enterococcus*, *Pseudomonas aeruginosa, Shigella*, *Staphylococcus aureus*, *Streptococcus pneumonia*, tuberculosis, Group A *Streptococcus*, and Group B *Streptococcus*. See *Threat Report 2013*, Center for Disease Control (Sep. 16, 2013), http://www.cdc.gov/drugresistance/threat-report-2013/index.html**;** see also *Antimicrobial Resistance*, World Health Organization (May 2013), http://www.who.int/mediacentre/factsheets/fs194/en/.

[171] Schroeder et al., *supra* note 135, at 197.

[172] *Id.*

[173] *Id.*

[174] *Id.*

Another study of 47 E coli isolates in Spain in 2001 found that 53% were resistant to nalidixic acid and 13% to ciprofloxacin.[175] These developments in E coli are especially startling given E coli's nature of readily transferring resistance genes to other intestinal microflora, not just E coli.[176] This can possibly make any latent diseases inside one's own gut, into a seemingly unstoppable illness.

Salmonella typhimurium resistance to commonly used tetracylines has jumped from 0% in 1948 to 98% in 1998.[177] One of the most commonly used antibiotics against Campylobactor, Quuinolone, is now ineffective against Campylobacter strains in the Netherlands, the UK, Finland, Spain, Germany, Canada, Sweden, France, Japan, and the USA.[178] Consumer Reports have also found that, of those supermarket chickens infected with Campylobacter and Salmonella, 67% and 84% contained Campylobacter and Salmonella strains, respectively, resistant to one or more common antibiotics.[179]

F. Historical Development of Antibiotics and Resistance Thereto

To get a better understanding of this issue let us look at the historical roots and progression of antibiotic resistance. At the beginning of the 20th century, bacteriology and scientific hygiene had the most focus among the medical disciplines.[180] Fear over infections without effective treatment fueled research, but even more motivation was found in warfare. The original focus was on educating women to have a safe, clean home and to prevent food spoilage, but with Alexander Fleming's work, which was later rediscovered by Ernest Boris Chain and Howard Florey during the Second World War, the medical profession turned to antibiotics. Tyrothricin and gramicidin worked but were also toxic when ingested. Therefore, when penicillin was discovered to not have those negative side effects, its development was seen as crucial for the war effort.[181]

Penicillin research, like many scientific achievements, helped to convey a hope that modern, systematic, and peer reviewed progress would always solve social ills; a belief that anything is possible as long as enough money is available.[182] Like the Green Revolution, Chain and Florey's work was funded by the Rockefeller Foundation which made the newly made

---

175 *Id.* at 200.

176 *Id.* at 198.

177 Teuber, *supra* note 141, at 755.

178 *Id.* at 759.

179 Blatt, *supra* note 26, at 117. For a reference to other instances of antibiotics resistance, see note 170, *supra*, and sources cited there.

180 Thoms, *supra* note 163, at 183.

181 *Id.*

182 *Id.*

antibiotic business a commercial success. Overproduction and resulting declining prices followed leading into the cheap and available use in agriculture.[183]

Antibiotics were first used in agriculture to cure infections in the milk industry. At the time, dairies were still suffering from poor public opinion due to the discovery that milk could transfer brucellosis and tuberculosis in the 19th century.[184] The negative connotations of "white poison" finally went away though during the 1939/40 World Fair in New York.[185] "The Dairy World of Tomorrow" was being exhibited with a newly developed automatic milker called a Rotolactor meant to be more hygienic than hand milking. However mastitis, udder infections, developed in sixteen of the cows, and so they were quickly treated with a new antibiotic, gramicidin. Though four cows had not recovered, gramicidin's success seemingly confirmed the utopian fantasy of continually progressing agriculture with antibiotics playing a key part in that future.[186]

From then on antibiotics were continually used in animal treatment, and even tested in animals before being used on humans.[187] The veterinarian then became an important part of the linear progression towards modern agriculture, despite any possible conflict of interest in promoting antibiotics, selling them, and then prescribing without any governmental oversight or tracking.[188] Open acceptance was not absolute for long, though. Consumers in the 1950s already became concerned over food safety related to eating the antibiotics themselves, as opposed to disease resistance. For example, Germany banned the use of antibiotics as food preservatives in its 1958 food regulation.[189]

However, the application of antibiotics to promote agriculture was more accepted by the USA where risks were weighed less compared to the economic advantages stressed by the veterinarians.[190] Small farmers may not have been fully convinced of the benefits of antibiotic use, but the demands of increasing the food supply after two World Wars, and potential profit, drowned out most critiques.[191] Concerns were also ignored on the grounds that they originally came from right-wing life reformers and ecologists who argued that antibiotics could disturb the bacterial equilibrium in one's own stomach. This was ignored for supposedly being unscientific

---

183 *Id.*

184 *Id.*

185 *Id.* at 185-186.

186 *Id.* at 186.

187 *Id.*

188 *Id.* at 197.

189 *Id.* at 188.

190 *Id.* at 189.

191 *Id.* at 189-90.

and backward, but the irony is that those claims have since been proven true, and, as the examples above demonstrate, we are observing a marked increase in antibiotic resistance.[192]

As science advanced and antibiotic resistance was discovered, people grew skeptical of antibiotic use in agriculture, but the demands of larger production scales prevented any real industrial or policy change. In 1977 the FDA tried banning the use of some antibiotics in agriculture, but the House and Senate appropriations committees, controlled by agricultural interests, passed resolutions against such a ban.[193] Being unsuccessful the FDA has taken a compromising stance instead of banning. Recently in 2010, the FDA approached the issue of antibiotics in food, but only by issuing draft guidance on judicious use.[194] The guidance suggested that antibiotics should only be used to treat, control, or prevent infections, and alongside the oversight of a veterinarian.[195] This guidance ignores the fact that veterinarians incentivized to prescribe antibiotics may be part of the problem, and that prevention has been one of the motivators in overusing antibiotics so this guidance changes nothing. Environmental interest groups have since been unsuccessful in challenging the FDA's voluntary governance approach.[196] There is also a disconnect between public opinion of antibiotics in the medical setting as opposed to in food production. The overuse of antibiotics in both medical practice and animal husbandry can lead to immune bacteria clusters so rationally people should call for reduced use overall, but this is not always the case. Antibiotics in medical treatments are still highly regarded, probably because the near miraculous like nature of the discovery of penicillin that we still remember today, while antibiotics in agriculture are vilified.[197]

Ignoring antibiotic overall is especially dangerous given recent trends. Between 2003 and 2010 the use of antibiotics went from seven hundred twenty four to nine hundred worldwide.[198] Within the last half century dozens of new antibiotics have been licensed, but despite these "new" products, and increased use thereof, most antibiotics today are merely minor chemical variants on those antibiotics that bacteria have already formed resistances.[199] There has only been one new class of antibacterial agents, oxazolidinones, since the 1970s.[200]

---

[192] *Id.* at 190-91.

[193] Sabrina Tavernise, *Farm Use of Antibiotics Defies Scrutiny*, N.Y. TIMES, Sept. 3, 2012, at D1.

[194] Editorial, *Antibiotics and Agriculture*, N.Y. TIMES, June 29, 2010, at A30.

[195] Thoms, *supra* note 163, at 190-191.

[196] *See Nat. Res. Def. Council v. Food & Drug Admin.*, 760 F.3d 151, 171-72 (2d Cir. N.Y. 2014) (holding that the FDA retains the discretion to continue allowing the use of animal drugs despite a risk to human health).

[197] *Id.* at 202.

[198] *Id.* at 203.

[199] Marc Lipsitch et al., *Antibiotics in Agriculture: When is it Time to Close the Barn Door?*, 99 PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, issue 9, pp. 5752-54 (2002), http://www.pnas.org/content/99/9/5752.full.

[200] *Id.*

We thus have a food production environment saturated with antibiotics, but no novel change in their structure or rate of use, making it even easier for resistance genes to establish and spread. However we also have alongside that an increasing use of antibiotics in medicine and everyday cleaning products making it is hard to determine how exactly antibiotic resistance is forming. Is it primarily through agriculture or just everyday use? It is with this doubt that those against the reduced use of antibiotics in agriculture present their counterargument.

G. Food borne Pathogens and Antibiotics Rebuttal: Comparison, Summation, and Diagnosis

Despite media hype, and everything pointed out above, it appears that rates of food contamination and resulting diseases are not increasing according to Center for Disease Control data.[201] The largest estimate of 5,000 deaths from food borne illnesses in the USA each year,[202] is nothing compared to the 36,036 Americans who die from car crashes each year.[203] We can use this same rhetorical argument to dismiss concerns over most foodborne pathogens by looking at the much larger numbers of deaths resulting each year from heart disease (600,000), cancer (577,190 in 2012), gun violence (30,000), and even toasters (several hundred worldwide).[204] Then again, maybe it is obtuse to compare kitchen appliances and chronic diseases to vector borne pathogens. If that is the case then let us look at another celebrity disease – the West Nile Virus.

The West Nile virus is characterized with neurological symptoms such as encephalitis, meningitis, gastrointestinal inflammation, fever, seizures, hallucinations, and memory loss in more extreme cases.[205] West Nile virus was first introduced into the USA in New York during the summer of 1999[206], and as it established itself in all forty eight continental states by 2005

---

[201] Nyachuba, *supra* note 125, 15 261-262.

[202] *Id.*

[203] *Fatality Analysis Reporting System Data Tables*, National Highway Traffic Safety Administration, http://www-fars.nhtsa.dot.gov/Main/index.aspx**.**

[204] *Heart Disease Facts*, Center for Disease Control and Prevention (Sept. 26, 2014), http://www.cdc.gov/heartdisease/facts.htm; *Cancer Facts & Figures 2012*, American Cancer Society, 2012, http://www.cancer.org/acs/groups/content/@epidemiologysurveilance/documents/document/acspc-031941.pdf; Georgina Olson, *More Than 30,000 Die from Gunshot Wounds Each Year in the United States*, Woodrow Wilson International Center for Scholars, Oct. 25, 2010, http://www.wilsoncenter.org/sites/default/files/Olson_21.pdf; Michael Hanlon, *Killers in Your Kitchen: Gender-Bending Packaging, Exploding Floor Cleaners and Toasters More Deadly than Sharks*, DAILY MAIL, Jan. 22, 2010, http://www.dailymail.co.uk/femail/food/article-1245151/Killers-kitchen-Gender-bending-packaging-exploding-floor-cleaners-toasters-deadly-sharks-.html#axzz2JzETnqid.

[205] Don Weiss et al., *Clinical Findings of West Nile Virus Infection in Hospitalization Patients, New York and New Jersey, 2000*, 7 EMERGING INFECTIOUS DISEASES, issue 4, pp. 654-58 (2001), Weiss et al., *Clinical Findings of West Nile Virus Infection in Hospitalization Patient*.

[206] Carmen Tedesco, Marilyn Ruiz, & Sara McLafferty, *Mosquito Politics: Local Vector Policies and the Spread of West Nile Virus in the Chicago Region*, 16 HEALTH AND PLACE, issue 6, pp. 1188-95 (2010),

with an exponentially increased infection rate.[207] From an initial sixty two cases in 1999 there has been a high of 9,862 viral infections in 2003, totaling 39,557 Americans affected since the introduction, and averaging 2,637.13 infections per year, 111.2 of them fatal each year.[208] The virus is now mostly reported in coastal states like New York, California, and Mississippi being the most afflicted.[209]

Unlike E coli, Campylobacter, Salmonella, and Listeria, predominantly orally transmitted infections, the West Nile virus is spread via mosquitos which receive the virus from birds that then spread the disease to humans as they continue feeding. Humans, and other larger animals, are dead end hosts as they usually do not reach a viral load sufficient to spreading the infection to new mosquito vectors therefore newly infected birds sustain virus populations.[210] This host specific nature of West Nile virus signifies that physical and political ecology are the limiting factors involved in its spread.

Physical ecology refers to biological and environmental forces that limit or promote disease vector populations such as hydrogeography and climate. Hydrogeography and climate considerations include those areas with standing water, in which mosquito larvae develop, and rainy seasons, which reduce West Nile cases in humans because bird populations, mosquitos' preferred host, are not motivated to search for more water during those times. In dry, hot periods birds venture further from their local geographies and mosquitos consequentially search out alternative hosts, increasing human West Nile virus infections.[211] Anthropomorphic climate change then poses the unique public health danger of increasing drought severity and frequency which promotes West Nile.[212] Humans also further spread the virus by creating more urban spaces where birds, and consequentially people, are more susceptible to infection.[213]

---

http://www.researchgate.net/publication/45650367_Mosquito_politics_local_vector_control_policies_and_the_spread_of_West_Nile_Virus_in_the_Chicago_region.

[207] Michael Walsh, *The Role of Hydrogeography and Climate in the Landscape Epidemiology of West Nile Virus in New York State from 2000 to 2010*, 7 PLOS ONE, issue 2, pp. 1-6 (2012), http://www.plosone.org/article/info%3Adoi%2F10.1371%2Fjournal.pone.0030620.

[208] *West Nile Virus Disease Cases and Deaths Reported to CDC by Year and Clinical Presentation, 1999-2013*, Center for Disease Control (May 29, 2014) [hereinafter CDC-West Nile], http://www.cdc.gov/westnile/resources/pdfs/cummulative/99_2013_CasesAndDeathsClinicalPresentationHumanCases.pdf.

[209] *West Nile Virus*, Center for Disease Control and Prevention (Jan. 30, 2015), http://www.cdc.gov/ncidod/dvbid/westnile.

[210] Tedesco et al., *supra* note 206.

[211] Walsh, *supra* note 207.

[212] Catherine Cooney, *Climate Change & Infectious Disease, is the Future Here?*, 119 ENVIRONMENTAL HEALTH PERSPECTIVES, issue 9, pp. A395-98 (2011), http://ehp.niehs.nih.gov/119-a394/.

[213] Shannon LaDeau et al., *West Nile Impacts in American Crow Populations are Associated with Human Land Use and Climate*, 26 ECOLOGICAL RESEARCH, issue 5, pp. 909-16 (2011), http://www.researchgate.net/publication/225225816_West_Nile_virus_impacts_in_American_crow_populations_are_associated_with_human_land_use_and_climate.

Political ecology is the combination of cultural ecology and political economy.[214] In the context of West Nile, this means that its spread is place specific, predicated on public attitudes towards proposed solutions, social patterns that may promote or discourage viral infections, and economic capital to confront the problem. Communities averse to pesticidal programs succumb to West Nile more often than those embracing chemical programs, as do impoverished areas with lower tax bases and public knowledge of the problem.[215] Also, social behaviors such as draining standing water campaigns can inhibit the virus. There is then a philosophical, monetary, and educational rift that can determine the spread of the disease.

A discussion of the factors promoting West Nile's spread may seem like a long digression, but the point of discussing West Nile is to show that its causes have largely been ignored since its emergence in the USA, while beef recalls due to E. coli and other pathogens still make headline news. This is despite the fact that West Nile, though decreasing in frequency, still kills more people each year than most food borne pathogens, and West Nile can be more readily alleviated than the institutional causes of food borne pathogens. For a side-by-side comparison, see the table in Box #4. Preventing West Nile involves mostly simply convincing people to accept pesticide programs while attempting to fix the spread of food borne pathogens requires the costly, complete overhaul of our vertically integrated food system.

Box #4. Comparison of Pathogen-Caused Illnesses and Deaths [216]

Pathogen Comparisons (US Estimations)			
	Illnesses per Year	Hospitalizations per Year	Deaths per Year
E. coli, O157	3,268	2,138	31
Salmonella, nontyphodial	1,027,561	19,336	378
Campylobacter	845,024	8,463	76
Listeria	1,600	1,400	255
Total Foodborne Pathogens	47,800,000	127,839	3,037
West Nile	2,637.13	Not Available	111.2

[214] Tedesco et al., *supra* note 206, at 1189.

[215] *Id.*

[216] The data for E. coli were taken from Elaine Scallan, et al., *Foodborne Illness Acquired in the United States – Major Pathogens, Table 3*, Center for Disease Control (2011), http://wwwnc.cdc.gov/eid/article/17/1/p1-1101-t3. The data for Salmonella, Campylobacter, and total foodborne pathogens were taken from *CDC 2011 Estimates*, *supra* note 125, while Listeria's estimates were found in *National Enteric Disease Surveillance: The Listeria Initiative*, Center for Disease Control (Aug. 2014), http://www.cdc.gov/listeria/pdf/ListeriaInitiativeOverview_508.pdf. Finally, estimates regarding West Nile virus can be found at CDC-West Nile, *supra* note 208.

Returning to antibiotic resistance specifically, deciding whether or not it is a public health issue related to agriculture is muddled by a concurrent, excessive use of antibiotics in everyday practice. Other conflicting evidence has been shown in Nebraska where antibiotic resistant Salmonella transferred to humans simply by being near cattle.[217] These are valid points, but dismissing concerns over agricultural use of antibiotics solely based on them seems like an industry public relations ploy of simply creating doubt without actually addressing a concern.

Maybe it does not matter if antibiotic resistance is a result more of agricultural or medical use. Molecular analysis of resistance genes reveals that food bacteria are not necessarily separated from human counterparts, and genes have been proven to transfer in vitro, in a test tube, and in vivo, in actual digestive tracts.[218] This means that lowering antibiotic use in agriculture can still help prevent more antibiotic resistance development, but such initiatives need to be accompanied by judicious antibiotic use in medical and commercial products.

Consumers and scientists may be right to be worried about food borne pathogens, but that concern should be nuanced with an understanding that there are far more pressing public health dangers to tackle. This by no way means that one should not concerned about these regarding diseases regarding agriculture, far from it, but approaching other public health and environmental concerns may help more people in the short term, so addressing them should be a priority. Antibiotic overuse needs to be addressed immediately however, but it is not just an agriculture issue. I feel that this holistic view, considering other dangers in comparison, is a more accurate and fair approach than doomsday foretelling or worse, denying there is a problem at all, as some industry groups do.[219]

Having explored E coli, Campylobacter, Listeria, and antibiotic resistance, which threatens to make the aforementioned bacteria stronger, let us now turn to a related, but separate topic – CAFOs.

IV. Confined Animal Feeding Operations

Although animal agricultural production involves several of the public health issues already discussed in this article, CAFOs deserve special consideration because of their unique impacts on water quality and quality of life in impoverished rural communities. There are even instances of the waste from Arkansas chicken CAFOs entering into the Illinois River watershed that supplies water for twenty two public water utilities in Oklahoma.[220] The waste emitted by

---

[217] Dan Ferber, *Superbugs on the Hoof*, 288 SCIENCE, issue 5467, pp. 792-94 (2000), http://www.sciencemag.org/content/288/5467/792.summary.

[218] Teuber, *supra* note 141, at 755.

[219] *Scientists Question ABC News Report Linking Antibiotic Resistant Bladder Infections to Chicken; Say Chicken is Safe*, National Chicken Council (July 11, 2012), http://www.nationalchickencouncil.org/scientists-question-abc-news-report-linking-antibiotic-resistant-bladder-infections-to-chicken-say-chicken-is-safe/.

[220] *Watch that Bird's Rear*, ECONOMIST, July 14, 2005, at 44, http://www.economist.com/node/4174555.

these poultry operations equals the amount generated by ten point seven million people, over one and half times total populations of Arkansas and Oklahoma combined.[221]

A. Public Health Concerns Related to CAFOs

Starting with the nitrous nutrients, such as nitrate, found in excess in farm animal excrement, high density animal farming results in massive amounts of manure which is washed between slats in the floors where it collects into waste lagoons.[222] For example, in North Carolina, a state with one of the highest concentration of hog CAFOs, 124,000 metric tons of nitrogen and 29,000 metric tons of phosphorus are generated in the manure of hogs each year.[223]

Manure, rich in nitrogen and other nutrients, serves as a perfect medium for pathogens like E. coli, Salmonella, Streptococcus, and pathogenic protozoans to grow and multiply.[224] Those pathogens can then be a threat to a community's water supply should a CAFO have an accidental excrement release. However, even if the waste is contained, the stench emitted from the waste, including ammonia, hydrogen sulfide, and volatile organic compounds, still causes tension, depression, fatigue, anger, and increased asthma rates among nearby rural populations.[225]

Also, even with primary treatment and containment measures, CAFO waste lagoons are nonetheless often emptied onto neighboring agricultural fields to act as fertilizer.[226] Secondary treatment of livestock waste to remove excess nutrients is seldom practiced.[227] This means that

---

[221] Blatt, *supra* note 26, at 118.

[222] Michael Mallin & Lawrence Cahoon, *Industrialized Animal Production: A Major Source of Nutrient and Microbial Pollution to Aquatic Ecosystems*, 24 POPULATION AND ENVIRONMENT, issue 5, pp. 369-85 (2003), http://link.springer.com/article/10.1023/A%3A1023690824045#page-1; Sacoby Wilson et al., *Environmental Injustice and the Mississippi Hog Industry*, 110 ENVIRONMENTAL HEALTH PERSPECTIVES, supplement 2, pp. 195-201 (2002), http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1241163/pdf/ehp110s-000195.pdf.

[223] Mallin & Cahoon, *supra* note 222, at 369.

[224] *Id.* at 370-71; Steve Wing, Dana Cole, & Gary Grant, *Environmental Injustice in North Carolina's Hog Industry*, 108 ENVIRONMENTAL HEALTH PERSPECTIVES, issue 3, pp. 225-31 (2000), http://citeseerx.ist.psu.edu/viewdoc/summary;jsessionid=02F098184D0DD9262F3485FC90A9CEAD?doi=10.1.1.280.8784.

[225] Rachel Horton et al., *Malodor as a Trigger of Stress and Negative Mood in Neighbors of Industrial Hog Operations*, 99 AMERICAN JOURNAL OF PUBLIC HEALTH, supplement 3, pp. S610-15 (2009), http://www.ncbi.nlm.nih.gov/pubmed/19890165; Kelly Donham et al., *Community Health and Socioeconomic Issues Surrounding Concentrated Animal Feeding Operations*, 115 ENVIRONMENTAL HEALTH PERSPECTIVES, issue 2, pp. 317-20 (2007), http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1817697/; Anthony Ladd & Bob Edwards, *Corporate Swine and Capitalist Pigs: A Decade of Environmental Injustice and Protest in North Carolina*, 29 SOCIAL JUSTICE, issue 3, pp. 26-46 (2002), http://www.socialjusticejournal.org/archive/89_29_3/89_03Ladd.pdf.

[226] Heather Williams, *Fighting Corporate Swine*, 34 POLITICS AND SOCIETY, issue 3, pp. 369-98 (2006), http://pas.sagepub.com/content/34/3/369.abstract.

[227] Mallin & Cahoon, *supra* note 222, at 370-371.

CAFOs are all but guaranteed to release excess nitrogen loads unsuited for their environments, leading to eutrophication, fish kills, exponential toxic dinoflagellate growth, and contaminated groundwater.[228]

In terms of public health, nitrogen contaminated water can lead to shortness of breath and methemoglobinemia, commonly known as blue baby syndrome, in those infants who directly drink the water, and teratogenic or developmental abnormalities in those infants who receive the nitrogen indirectly through their mothers while they are in the womb.[229] However, these effects are only associated drinking water levels higher than the current maximum nitrate contaminant level of or ten parts per million, meaning that current water quality restrictions, if followed, appear to adequately protect public health. [230]

However, current regulations have failed to address environmental justice concerns associated with intensive animal production as people in rural communities disproportionately bear the environmental burden of CAFOs, relative to more affluent, urban populations. In states with the highest amount of hog CAFOs, impoverished and minority populations experience a disproportionate amount of risk associated with CAFO waste lagoons. In North Carolina, where groundwater pollution is especially dangerous due its high water table and hurricanes, hog production is concentrated in areas with higher percentages of poverty, nonwhite populations, and homes dependent on domestic wells.[231] This pattern also holds for Kansas, Oklahoma, and Missouri in rural areas where communities have less social and political capital to oppose hog CAFOs.[232] In Mississippi it has been demonstrated that CAFOs are consistently concentrated in areas of higher poverty rates and nearer African American demographic communities, as much as over three times as many hog operations.[233] The environmental inequity is then quite apparent at the state level. Although other anthropogenic sources of nitrogen have contributed to those values, the accumulation of CAFOs in watersheds used to monitor national nitrogen pollution indicates that intensive animal production is a significant contributor.[234] Nonetheless, debate still occurs as to the extent of CAFOs allegedly harmful public health impacts.

---

[228] *Id.* at 378-80.

[229] Anna Fan and Valerie Steinberg, *Health Implications of Nitrate in Drinking Water: An Update on Methemoglobinemia Occurrence and Reproductive and Developmental Toxicity*, 23 REGULATORY TOXICOLOGY AND PHARMACOLOGY, issue 1, pp. 35-43 (1996), http://www.sciencedirect.com/science/article/pii/S0273230096900069; *Basic Information about Nitrate in Drinking Water*, US Environmental Protection Agency (Feb. 5, 2014), http://water.epa.gov/drink/contaminants/basicinformation/nitrate.cfm.

[230] *See* Fan and Steinberg, *supra* note 229.

[231] Wing et al., *supra* note 224, at 229-231.

[232] Williams, *supra* note 226, at 372-82.

[233] Wilson, *supra* note 222, at 199-200.

[234] Mallin & Cahoon, *supra* note 222, at 378-380.

B. Critiques of the Concerns

One dispute as to whether CAFOs are indeed damaging to public health is connecting nitrates in drinking water to methemoglobinemia. It is already established that methemoglobinemia is connected to nitrates, and that CAFOs produce an exorbitant amount of nitrates. This is accepted by most regulatory professionals and scientists, but simple gastrointestinal infection and inflammation that overproduce nitric oxide in the guts of infants can explain methemoglobinemia as well.[235]

This latter scenario explains why infants suffering from diarrhea and methemoglobinemia will excrete ten times more nitrates daily than they ingest through food or water.[236] Infants' gastrointestinal infections being the true source of excess nitrates also suggests that regulatory standards on nitrate water levels could be lowered without endangering public health.[237] This suggestion is further supported by no blue baby syndrome cases being reported when drinking water is below the maximum contaminate level of nitrogen or nitrate.[238] However, explaining away the source of nitrates speaks nothing to nitrites, which are still toxic, and still potentially released by CAFOs.[239] Furthermore, nitrates are converted to nitrites, and vice-versa, depending on which microbes are consuming the nutrients. Therefore, disputing the ultimate source of methemoglobinemia is a red herring, distracting people from the real issue; no matter whether one focuses on nitrites or nitrates, there is still excrement in the water.

As for people living near CAFOs suffering tension, depression, fatigue, anger, and increased asthma rates, a cynic could respond that, since rural populations have dropped continually throughout the 20th century, fewer people today are experiencing these problems and therefore that we should focus on issues that afflict more people. Progress as usual will remove more people from the countryside, thereby eliminating said tension, depression, fatigue, anger, or increased asthma rates. Arguing that the public health impacts related to CAFOs are not as troubling as they affect fewer people is a valid observation, but a rejectable one as well because it is essentially ignoring the problem.

---

[235] Alexander Avery, *Infantile Methemoglobinemia: Reexamining the Role of Drinking Water Nitrates*, 107 ENVIRONMENTAL HEALTH PERSPECTIVES, issue 7, pp. 583-86 (1999), http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1566680/.

[236] *Id.* at 585.

[237] *Id.* at 589; Lorna Fewtrell, *Drinking-Water Nitrate, Methemoglobinemia, and Global Burden of Disease: A Discussion*, 112 ENVIRONMENTAL HEALTH PERSPECTIVES, issue 4, pp. 1371-74 (2004), http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1247562/.

[238] Fan & Steinberg, *supra* note 229.

[239] Avery, *supra* note 235.

Another defense of CAFOs is that new dietary supplements for the animals and technological innovation will fix CAFO externalities.[240] This is simply pure technological optimism: it is logically fallacious, naïvely hopeful, and will not solve the institutional problems in an otherwise unsustainable agriculture system.

Moving on, since CAFO health effects are presented as an environmental justice issue it falls prey to many of the inherent downfalls of the environmental justice movement itself. Pursuing environmental justice goals may actually exacerbate inherent environmental policymaking breakdowns such as economic inefficiency, muddled policy priorities, and there is a gap between expert and public risk perceptions.[241] Advocates against CAFOs may argue for less or even the elimination of intensive animal husbandry, but that implies that local food production will have to take its place which could reduce food access and increase costs via inefficiency.[242]

Environmental justice campaigns can often times be more interested in community advocacy and holding back private capital than in actual health concerns.[243] The members of a suspicious public may be apt to point blame at the incoming corporate hog operation for public health problems, but they may be ignoring other health factors.[244] Individual actions, such as choosing to smoke or not, and indoor air pollution do more harm to people today then the heavily vilified outside pollutants.[245] This means that health problems attributed to CAFOs could just as easily be explained by smoking, an activity that that the rural participate in more than the urban, indoor air quality, or other poor decisions affecting your immediate person and environment.[246] Fighting polluters has a compelling narrative, but the real answer may be unexciting personal changes.[247]

However, critiquing the pursuit of environmental justice as a collateral attack on the public health concerns would appear disingenuous. Even if environmental justice is flawed as a

---

[240] Oluyinka Oluksoi & Olayiwola Adeola, *The Possibility for Reducing Water Pollution Resulting from Concentrated Animal Feeding Operations and the Impact of Phytase*, Aug. 2007, http://www.extension.purdue.edu/extmedia/ID/cafo/ID-355-W.pdf.

[241] Foreman, *supra* note 37, at 3-4.

[242] Steven Sexton, *Does Local Production Improve Environmental and Health Outcomes?*, Giannini Foundation of Agricultural Economics, University of California, http://giannini.ucop.edu/media/are-update/files/articles/v13n2_2.pdf.

[243] *Id.* at 5.

[244] *Id.* at 40.

[245] *Id.* at 131.

[246] Mark Doescher et al., *Prevalence and Trends in Smoking: A National Rural Study*, 22 JOURNAL OF RURAL HEALTH, issue 2, pp. 112-8 (2006), http://www.ncbi.nlm.nih.gov/pubmed/16606421.

[247] Foreman, *supra* note 37, at 66-70.

concept, such a conclusion speaks nothing to the noxious and nitrous emissions from CAFOs. Those emissions are still occurring, and their impacts are still apparent.

C. Conclusion on CAFOs

The causally related health impacts of living near a CAFO are well understood. Health disparities between rural and more affluent, urban populations are also apparent, but the origins of those disparities and instances of health deterioration may be debatable. The counter arguments capitalize on that and point out that environmental justice may create more problems or divert focus on personal health responsibility. However, as valid as those arguments may be they, again, do not deny the plausibility of harm from drinking fecal contaminated water or breathing noxious gas. The criticisms of the public health concerns pertaining to CAFOs are more like distractions rather than rebuttals. Finally, let us end by exploring what may arguably be the greatest public health danger related to agriculture – obesity.

V. Obesity and Agriculture

A. Introduction

Obesity has become a major platform issue for those in and out of the public health field in recent years. With over two thirds, nearly 68%, of US adults being either overweight or obese, this newfound national attention is understandable and commendable, but unfortunately there are two faults in the mainstream thought.[248] The first is misplacing blame for the American obesity epidemic. The public can harshly, and incorrectly, assume that other peoples' obesity is the sole product or their effort or lack thereof.[249] The second is not realizing that America's obesity is byproduct of our modern agricultural model.[250]

---

[248] *See* Melanie Warziski et al., *Psychosocial Correlates of Weight Maintenance Among Black & White Adults*, 36 AMERICAN JOURNAL OF HEALTH BEHAVIOR, issue 3, pp. 395-407 (2012), http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3454451/.

[249] David Wallinga, Heather Schoonover, & Mark Muller, *Considering the Contribution of US Agricultural Policy to the Obesity Epidemic: Overview and Opportunities*, 4 JOURNAL OF HUNGER & ENVIRONMENTAL NUTRITION, issue 1, pp. 3-19 (2009) [hereinafter Wallinga-2009], http://www.tandfonline.com/doi/abs/10.1080/19320240802706817?journalCode=when20#preview; Eubanks, *supra* note 10, at 279-300.

[250] Although this article focuses almost entirely on obesity in the USA, obesity is unquestionably a global issue. In 2014 it was determined that over a quarter of the world's human population is either overweight or obese. Betsy McKay, *Study Finds Nearly 29% of the World is Overweight or Obese*, WALL STREET JOURNAL, May 29, 2014, http://online.wsj.com/articles/nearly-30-of-world-population-is-overweight-1401365395; *Overweight and Obesity Viz* (May 2014), http://vizhub.healthdata.org/obesity/. But excess weight is not equally spread across the globe. Instead, with much variety, it appears that the range of percentages is quite vast between countries – with those populations that experience extreme levels of poverty having little excess body fat, and more developed nations accounting for higher body mass indices. See *Global Database on Body Mass Index*, World Health Org. (Aug. 21, 2014), http://apps.who.int/bmi/index.jsp. See also *Global Health Observatory Data Repository*, World Health Org. (2013), http://apps.who.int/gho/data/node.main.A897; *Overweight/Obesity, 2008 Prevalance of Overweight, Ages 20+, Age Standardized: Both Sexes*, World Health Org. (2011), http://gamapserver.who.int/gho/interactive_charts/ncd/risk_factors/overweight_obesity/atlas.html. For information about obesity in the world's most populous state – China – see Youfa Wang et al., *Is China Facing an Obesity*

Population changes through biological effects like genetics or individual choice take much longer to manifest, especially on a country wide scale, and there is no measurable epidemic of laziness to explain unprecedented obesity rates over the past 30 years.[251] Instead, the environment and agriculture must be more critical, and indeed let us explore the obesogenic environment America has created through agricultural policies.[252]

B. Background and Historical Development

In the most basic sense, one becomes overweight when he or she consumes more calories than they expend in a day over an extended time period such that excess fat is accumulated.[253] One is then obese once he or she reaches a body mass index of 30 kg/m^2 or more.[254] This excess weight and adipose tissue puts extra stress on the human body, increasing the risk of coronary heart disease, diabetes, cancer, hypertension, dyslipidemia, stroke, liver and gall bladder disease, sleep apnea, osteoarthritis, gout, impotence, and gynecological disorders such as infertility.[255]

The effects of food over consumption are even more concerning considering that the four leading causes of death in the USA are all diet related – heart disease, cancer, stroke, and diabetes. As such those causes of death are all directly related to obesity, a diet related ailment, and therefore the marked increase in obesity over the past thirty years has seen subsequent rises in all four conditions.[256] The direct and indirect costs of obesity in the USA have been estimated at $117 billion including medical expenses and lost worker productivity, but that figure does not begin to comprehend the immeasurable amount of emotional and psychological damage dealing with the social stigma of being obese.[257] Because of the cost and chronic ailments obesity is a major public health policy concern, but that does not mean that its pertinence is not contested.

Epidemic and the Consequences? The Trends in Obesity and Chronic Disease in China, 31 INTERNATIONAL JOURNAL OF OBESITY 177, 177 (2007), http://www.nature.com/ijo/journal/v31/n1/full/0803354a.html (noting that between 1992 and 2002 the percentage of Chinese overweight or obese rose from 14.6 to 21.8%). See also *Trends*, World Obesity (2014), http://www.worldobesity.org/aboutobesity/resources/obesity-data-portal/resources/trends/ (follow "Trends in overweight and obesity in Chinese adults" hyperlink).

[251] Michael Power, *The Human Obesity Epidemic, the Mismatch Paradigm, and our Modern 'Captive' Environment*, 24 AMERICAN JOURNAL OF HUMAN BIOLOGY, issue 2, pp. 116-22 (2012), http://www.ncbi.nlm.nih.gov/pubmed/22287210; Wallinga-2009, *supra* note 249, at 3-4.

[252] Wallinga-2009, *supra* note 249, at 3-4.

[253] Power, *supra* note 251.

[254] Warziski et al., *supra* note 248.

[255] *Overweight and Obesity*, Center for Disease Control and Prevention (Sept. 9, 2014), http://www.cdc.gov/obesity/data/trends.html.

[256] *Id.*; Wallinga-2009, *supra* note 249; Eubanks, *supra* note 10, at 287-93.

[257] Wallinga-2009, *supra* note 249, at 3.

Some individuals become obese because they cannot stick to a low fat diet, and therefore some argue that obesity should not be a public health issue at all.[258] Accordingly, obesity is simply a matter of personal gumption, and there is no need to considering balancing the imperatives of an overall healthy country with the individual liberty to eat whatever one wants. However viewing obesity as simply a personal choice ignores the fact that people do not act, or eat, in a vacuum.[259]

Obesity is exacerbated by barriers to healthy eating such as taste concerns, beliefs that healthy food is too expense, distances away from sources of health food, not having enough time or motivation to eat healthier, and stressful life events like divorce or job insecurity.[260] These barriers are compounded with reports of people eating more food they dislike, usually calorically dense yet nutritionally lacking, to compensate for times of want.[261] This behavior of eating more than immediately necessary appears to be an evolutionary holdover from our days as gather-hunters when high-calorie food supplies were not abundant. Now, however, we have retained our digestive system built for occasional feasts of fat laden meals, but in an environment surfeit with high calorie, cheap food. In addition, humans are also disadvantaged with a brain chemistry, which also evolved in a time period with no guaranteed food supply, which actively seeks calorie dense, salty, and sweet foods.[262]

As modern agriculture enabled humanity to leave the gatherer-hunter lifestyle, and enter an age of caloric wealth, food production is systematically connected to obesity. Moving away from our tradition of eating mostly grains and plant matter, modern agriculture has quadrupled meat intake worldwide over the past forty five years. As a result, meat now forms over half of humanity's calorie intake.[263]

The pattern of agriculture's progress leading to oversupplies of food and waistlines is seen throughout recent history. At the beginning of the 20th century, the American West was opened to prospecting farmers willing to make the venture and stake out land. Promises of easy farming and wealth enticed many westward, and as a result there was a boom in crop availability. Crop prices fell given the extra supply, but the cruel nature of agricultural economics dictates that farmers produce more in a self-defeating attempt to make up in volume what has been lost in price per bushel.[264] This pattern continued until prices were so low that surplus grain and

---

[258] *See* Warziski et al., *supra* note 248, at 405

[259] Wallinga-2009, *supra* note 249, at 3-8.

[260] Warziski et al., *supra* note 248, at 401-06.

[261] Brandi Franklin et al., *Exploring Mediators of Food Insecurity and Obesity: A Review of Recent Literature*, 37 JOURNAL OF COMMUNITY HEALTH, issue 1, pp. 253-64 (2012), http://www.ncbi.nlm.nih.gov/pubmed/21644024.

[262] Power, *supra* note 251, at 116-18.

[263] Blatt, *supra* note 26, at 111.

[264] Michael Pollan, *The (Agri)Cultural Contradictions of Obesity*, N.Y. TIMES MAGAZINE, Oct. 12, 2003, http://www.nytimes.com/2003/10/12/magazine/12WWLN.html?src=pm&pagewanted=1.

livestock were removed from the market in an attempt to drive prices back up, but most farmers were still forced off their land as the costs of early twentieth farming became too great.[265] As a result, newly available land was then incorporated into those farming operations large enough to survive the market crash, laying the groundwork for the heavily consolidated farm operations of today.

Thus, the Great Depression was actually a cruel irony in that it was a time of environmental refugees and bread lines, and yet there was actually a surplus of food that could have staved off the nation's hunger. Thankfully, the New Deal of the Roosevelt administration offered farm support to feed the masses and reconcile the paradox of plenty.[266] New Deal programs like the Agricultural Adjustment Act and Soil Conservation Service encouraged farmers to plant more crops to provide for ever more populated urban centers with subsidized prices and upfront costs, while also paying farmers for putting some lands into fallow.[267]

The New Deal, combined with the economic boost of two World Wars, succeeded in feeding the hungry and keeping the remaining farmers afloat, without drastically increasing national obesity rates, but then this cycle of boom and bust repeated itself forty years later. With the benefit of post-war mechanization, farming took over parts of the industrial military complex that were not necessary for fighting anymore, including the nitrogen from dynamite that then became fertilizer. Production surged making the USA a food exporter nation, but that proved to be a weakness in 1972 when an unexpected famine stricken Soviet Union increased the demand for US grain exponentially.[268] The smaller, less centralized farming structure of the time could not handle the demand. Domestic prices soared, and Earl Butz, then Secretary of Agriculture under President Nixon, shifted subsidy support from farmers to consumer prices in response to public outcry over the cost of food. Butz used his "fence row to fence row" mantra, telling farms to either get big or get out, to drive prices down with even more commodity production.[269]

The cycle of boom and bust of the 1930s thus repeated itself, with smaller farms becoming economically obsolete, bigger agricultural corporate models being able to expand their operations, and the rural workforce being driven into already stressed and impoverished urban areas. Meanwhile there was once again a surplus of commodity crops that needed to go somewhere. Though it may be impossible to physically force the human body to consume more than it needs, Americans have still been able to put away at least an extra 200 calories of these commodities per person since 1977, [270] half of which being from sugar and fat derived from the crops.[271] The newly subsidized commodities were soy, corn, wheat, and cotton which

---

265 Donald Worster, DUST BOWL 34-43 (1979).

266 Pollan, *supra* note 264.

267 Worster, *supra* note 265.

268 Pollan, *supra* note 264.

269 Wallinga-2009, *supra* note 249, at 6-8; Pollan, *supra* note 264.

270 Pollan, *supra* note 264.

271 Wallinga-2009, *supra* note 249, at 4.

agribusiness ingenuity has transformed into frying oil, partially hydrogenated soybean oil, high fructose corn syrup, the breading and filler for chicken nuggets, and many other processed foods that play on our biological drive to eat calorie dense food. These cheap grains were also, and are still today, fed to livestock to make cheap meat and dairy. The Chicken McNugget, the Dollar Menu, and increased portion sizes are then physical manifestations of this profit-driven, publically supported system that seeks to dispose of surplus grain at the expense of public health.[272]

The effects of this system are even more startling when you consider that the USDAbases nutritional guidelines on our grain, dairy, and meat surplus which are then used in the school lunch and Supplemental Nutrition Assistance Program (SNAP) programs meant to benefit impoverished people. Combine that knowledge with the realization that people of color are generally poorer than whites and it is understandable why poverty stricken people and certain races are vulnerable to obesity. Unfortunately, the 1996 Farm Bill, removing all federal agricultural supply management, possibly cemented this market structure, and meanwhile there are few subsidies for healthy food. The American diet is consequentially deficient in fruits and vegetables containing macro- and micronutrients that commodity crops and animal products lack.[273]

Agricultural policies are then the stage upon which health disparities for vulnerable groups manifest. The USA has become an obesogenic environment in which cheap calorie dense food is readily available, which combined with human impulses to eat those foods because of our gatherer-hunter history, explains the stark increases in obesity observed over the past thirty years.[274]

C. Vulnerable Gender

For women, obesity poses the extra challenge of disproportionate amounts of harm, lesser education, and less economic opportunities. A recent study of Boston area hospitals indicated that poorer groups experience obesity more than others, but when those results were stratified for gender the results held significance only for women.[275] Similar results were seen again in the results of the Center for Disease Control's National Health and Nutrition Examination Survey from 1999 to 2002.[276] In addition, when it comes to trying to mitigate obesity's health impacts, pregnant obese women have unfortunately been shown to be more likely to remain obese after

---

[272] Pollan, *supra* note 264.

[273] David Wallinga, *Agricultural Policy and Childhood Obesity: A Food Systems and Public Health Commentary*, 29 FOOD AND AGRICULTURE, issue 3, pp. 405-10 (2010) [hereinafter Wallinga-2010], http://www.ncbi.nlm.nih.gov/pubmed/20194980; Wallinga-2009, *supra* note 246, at 8.

[274] Wallinga-2010, *supra* note 273, at 8.

[275] Franklin et al., *supra* note 261, at 255-62.

[276] *Id.* at 261.

giving birth.[277] Also, daughters of obese mothers, because of fetal programing, are more likely to become obese than sons.[278]

Data from the Wisconsin Longitudinal Study over three decades demonstrate that heavy-set women receive less post-secondary education than thinner counterparts, putting them at a disadvantage at finding a higher paying job.[279] It is hypothesized that the women in this analysis received less schooling because of the demands of raising children, and because of society's higher demand for skinny women as opposed to skinny men. Another study looking at the first fifteen years of employment, from the Panel Study of Income Dynamics, supports that hypothesis with correlations existing between reduced women's wages, probability of marriage, and excess body fat. At the same time research indicates that career and education prospects for men are only nominally affected by obesity.[280]

Heavy set women are also less likely to get married, and more likely to get divorced which has the potential of putting more women in economic insecurity than men. One could argue that the delayed family planning would allow heavier women to have more time to secure career advantages and placement, but this has not been substantiated.[281] Women's extra vulnerability to obesity even exists despite the "sacrifice theory," which predicts that poverty stricken and stressed mothers will give their meals to their children.[282] Isolated incidences of sacrifice most certainly occur, but it does not appear to have a large enough counter obesity rates.

D. Vulnerable Children

Children are also more vulnerable to obesity than their adult counterparts. [283] Nursing infants of obese mothers are exposed to biochemical signaling, programing their bodies to be more inclined to gain weight as they grow up, and as they mature they are dependent on the food choices provided by care givers, which may not always be health promoting.[284] If environmental and social stressors succeed in making a child obese, then he or she is much more likely to

---

277 *Id.* at 262.

278 Power, *supra* note 251, at 120.

279 Christy Glass, Steven Haas, & Eric Reither, *The Skinny on Success: Body Mass, Gender, and Occupational Standing Across the Life Course*, 88 SOCIAL FORCES, issue 4, pp. 1777-806 (2010), http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2951322/.

280 *Id.* at 1780-1781.

281 *Id.* at 1781-1782.

282 Franklin et al., *supra* note 261, at 262.

283 Eubanks, *supra* note 10, at 286; Pollan, *supra* note 264.

284 Power, *supra* note 251, at 120.

remain obese into adulthood.[285] This will lead to the aforementioned health complications, but may also be detrimental to the physical transition into adulthood by upsetting the body's homeostatic tendencies.[286]

Children also face the social stigma associated with obesity that can cause them to be excluded from social groups, resulting in less self-confidence, and then possibly lead them to continue unhealthy behavior, such as overeating, as a coping mechanism.[287] Eating as a coping mechanism then creates a positive feedback loop leading to more obesity, more social stigma, and finally more eating to feel better. There are disagreements in the literature on how much of an effect this has on children's future occupations and income levels, most reinforcing the idea that being a fat woman is worse than being a fat child, but there is still the measurable chance that overweight teenagers are less likely to receive a college education.[288] Also, even in this debate, all sides can still agree and realize that a pre-teenage girl is then especially disadvantaged.

E. Vulnerable Races

Obesity has been demonstrated to disproportionately affect African American populations, relative to whites, regarding the burdens of hypertension, diabetes, cardiovascular disease, and other obesity related conditions.[289] A meta-analysis of fourteen published studies from 1999 to 2006 demonstrated that obesity significantly occurs more often in non-Hispanic white and Hispanic groups than among blacks,[290] and increasing Hispanic populations in the USA have resulted in higher obesity rates in those areas where Hispanics have concentrated.[291] A study of 496 adolescent females demonstrates that black and Latino girls are more vulnerable to obesity than their white peers, and racial disparities even continue when comparing the state of European Africans and African Americans.[292] These observations and more document the

---

[285] David Huh et al., *Female Overweight and Obesity in Adolescence: Developmental Trends and Ethnic Differences in Prevalence, Incidence, and Remission*, 41 JOURNAL OF YOUTH ADOLESCENCE, issue 1, pp. 76-85 (2012), http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3413457/.

[286] Lisa Nicholson and Christopher Browning, *Racial and Ethnic Disparities in Obesity during the Transition to Adulthood: The Contingent and Nonlinear Impact of Neighborhood Disadvantage*, 41 JOURNAL OF YOUTH ADOLESCENCE, issue 1, pp. 53-66 (2012), http://connection.ebscohost.com/c/articles/69887473/racial-ethnic-disparities-obesity-during-transition-adulthood-contingent-nonlinear-impact-neighborhood-disadvantage.

[287] Alexandra Brewis, *Stigma and the Perpetuation of Obesity*, 118 SOCIAL SCIENCE AND MEDICINE, pp. 152-158 (2014), http://www.sciencedirect.com/science/article/pii/S0277953614005206.

[288] *Id.* at 153.

[289] Warziski et al., *supra* note 248, at 395-396; X. Chen and Y. Wang, *Is Ideal Body Image Related to Obesity and Lifestyle Behaviors in African American Adolescents?*, 38 CHILD, CARE, HEALTH, AND DEVELOPMENT, issue 2, pp. 219-28 (2012), http://www.ncbi.nlm.nih.gov/pubmed/21434968.

[290] Franklin et al., *supra* note 261, at 254

[291] Huh et al., *supra* note 285, at 77.

[292] *Id.* at 79-83.

actualized disparities between racial groups indicating that minority groups are more vulnerable to obesity than whites.

Interestingly, racial vulnerabilities to obesity are even shown in cultural acceptance. Data from 402 low income African Americans indicates that ideal body images, for themselves and their partners, are larger than those of affluent and Caucasian individuals.[293] That research is in line with observations that overweight and obese black teenagers are more likely to perceive themselves as healthy, as opposed to overweight Caucasians who suffer from lower self-esteems from realizing their true health condition.[294] This indicates that there may be an African American cultural preference for larger partners resulting from African American populations already being disproportionately overweight, which in turn condones and endorses behaviors that could lead to obesity.

However, focusing only on racial or cultural differences as markers for obesity misses the point of the research entirely. Affluent whites and blacks are both just as likely to remain at healthy weights after a diet program, and therefore race itself cannot be the key factor in determining obesity. Race may instead be an indicator of prevailing disparities between socioeconomic groups.[295] Another study concluding that disadvantaged neighborhoods are obesity predictors for African American or Hispanic individuals, but not Caucasian, further support that observation that economics play a larger role in body weight, rather than race inherently leading one to being overweight.[296] In the Nicholson and Browning study, black and Hispanic populations were more concentrated in poorly infrastructured urban centers, which produced a statistically significant relationship between neighborhood disadvantage and weight attainment. This means that socioeconomics must be considered as well when discussing obesity.

F. Vulnerable Socioeconomic Groups

Peer reviewed literature has consistently confirmed a strong correlation between the food insecurity suffered by those in poor socioeconomic groups and the paradoxical obesity these marginalized populaces experience at a higher rate relative to the general US population.[297] There is even a relationship indicating that food stamps exacerbate obesity as food stamps do not provide impoverished people the ability to buy healthier food.[298] One dollar buys 1,200 calories of potato chips and cookies but only 250 calories of carrots, and when facing the dilemma of what to buy with limited funds, impoverished people on SNAP will invariably tend to purchase

---

293 Chen et al., *supra* note 289, at 222-26.

294 *Id.* at 220.

295 Warziski et al., *supra* note 248, at 401.

296 Nicholson & Browning, *supra* note 286, at 62.

297 Franklin et al., *supra* note 261, at 253.

298 *See* Eubanks, *supra* note 10, at 288.

the higher calorie option.[299] Given that prices for produce have risen 40% over the past twenty years, while soda prices have declined 20%, it becomes even more understandable why poor people may eat against their own best interests.[300]

The sad economics that drive impoverished people to buy unhealthier food explain the inverse relationship between social class status and weight.[301] However, other contributing factors to the disproportionate impact obesity has on poor groups involve the stressors of impoverished neighborhoods including lack of access to healthy food and violence.[302] The obesity impacts of impoverished neighborhoods stressors follows social disorganization theory, hypothesizing that obesity is an adaptive response to stressful environments,[303] and coincides with biological theories of human evolution and dietary developmental response to the availability of processed, post-industrial era foods.[304] This also puts the previous explanations of gender and race disparities into perspective as symptoms of deeper economic rifts.

G. Obesity and Agriculture – Conclusion

Overweight and obesity rates in the USA have progressed to the point of an epidemic, making it possibly the gravest public health concern pertaining to agriculture. Their causes are largely explained by the agricultural policies of the past century. There is simply no standing for an argument that obesity rates have not exponentially increased or that an abundance of calorie-dense, nutritionally-lacking food is not a problem. The only possibly effective retort would be to invoke personal responsibility, on grounds that people are ultimately obese because of their own food choices. However, one can also not ignore the institutional structures, originating from agricultural policy, that have shaped modern food choices.[305] The gender, age, racial, and economic disparities regarding obesity are evidence that personal responsibility alone cannot explain US obesity.

VI. Closing Observations

As noted at the outset, my aim in this article has been to explore a cluster of key public policy issues involved in industrial agriculture – specifically, those issues involving implications of modern agriculture on human health. In particular, I have examined five areas of public concern: (1) agricultural chemicals, (2) genetic engineering and genetic modification, (3) food-borne illness, (4) confined animal feeding operations, and (5) obesity. While I have not attempted here to propose solutions but rather to draw from the relevant policy, technical, and

---

299 *Id.*

300 Wallinga-2009, *supra* note 249, at 11.

301 Glass et al., *supra* note 279, at 1778.

302 Nicholson & Browning, *supra* note 286, at 53-54.

303 *Id.*

304 Power, *supra* note 251, at 116-18.

305 Marion Nestle, FOOD POLITICS 360-74 (2007).

professional literature in examining the legitimacy of concern expressed on these various topics, I would offer a few closing remarks that this survey brings to mind. My closing remarks touch on matters of balance, vulnerability, and limits.

First, in fashioning new national and global policies in the aftermath of the Green Revolution, it will be crucial to consider matters of *balance*. The foregoing discussions suggest that more weight has been accorded to (1) the volumes of production (of food grains, of livestock) and numbers of mouths that can be filled in an ever-expanding human population than has been accorded to (2) the quality of the food produced and the quality of the lives of those humans it feeds. Discussions of agricultural policy should give special attention to the question of how an appropriate balance is to be determined and then struck. Indeed, another type of balance – namely, profit versus protection – might also be given closer scrutiny in a new agricultural policy.

Second, certain elements of the human population are subject to much greater *vulnerability*: they can be injured more easily, and their interests more readily disregarded. I highlighted this concern over vulnerability in my discussion above of obesity. More broadly, matters of vulnerability also apply in a fashion not addressed in this article – the vulnerability of other species with which humans share this planet and that are much more dramatically affected than humans are by agricultural policies and practices described above, especially those relating to agricultural chemicals.

Third, a consideration of the human-health aspects of modern agriculture and agricultural policy should be informed by an appreciation of *limits*, in several respects. What might have seemed to some observers as the boundless promise of the Green Revolution must surely be regarded now as bounded and limited by several realities. These include ecological realities – humans cannot pursue vast extensification or intensification of agricultural production without hewing to the ecological limits and processes of the natural world. They also include the incompleteness of our own scientific knowledge and technical expertise – despite rapid advances of recent decades, humans remain woefully ignorant of how the natural world works and therefore of what cascading consequences might come from introducing changes into it (massive volumes of chemical fertilizers, for instance, or genetic modifications).

In short, I believe that the project of shaping new national and global agricultural policies should give close attention to the specific human-health concerns highlighted in this article, and that certain themes – focusing on balance, on vulnerability, and on limits – should also inform that project as well.