Modern Meat: Synthetic Hormones, Livestock, and Consumers in the Post-WWII Era

Nancy Langston UW-Madison nelangst@wisc.edu

In 1960, a Food and Drug Administration (FDA) employee named Charles Durbin told a gathering of poultry producers: "Chemicals and drugs have revolutionized agriculture in the past 15 years. In animal husbandry growth-promoting chemicals permit the production of more meat with less feed; drugs and biological eliminate or control serious diseases.…pesticides help the farmer control insects that would otherwise seriously affect his livestock….Over 50% of the drugs used by the veterinarian and the feed mills weren't available to them in the early 1940's. Truly, agriculture has entered the chemical age." Durbin promised his audience that great things were afoot in animal husbandry: "more hormones may find their way into tomorrow's feed; enzymes have been shown to improve the chick's ability to handle barley; tranquilizers have been reported to help birds during times of stress; we must take a new look at the appetite stimulators; and the Food and Drug Administration willing, we will see more and better medicinals being used in poultry feeds." Yet, he cautioned his audience, they had to recognize that the modern "consumer is concerned with the question of chemicals in their food."

Durbin recognized that industry and government were struggling to shape the narrative of modern drugs and chemicals in agriculture. He told his audience; "We recognize that all constituents of food and feeds are chemical in nature, but unfortunately to many people the term 'chemical' denotes something poisonous or dangerous. It is up to all of us whether we be in Government, in industry, or the university to explain fully the story of drugs and chemicals in modern animal production."[1] This was a story they had begun to articulate decades earlier, but one which kept escaping from their control—for as disturbing new stories about the unpredictable

risks of agricultural chemicals kept bubbling into public consciousness, consumers were not easily soothed.

Durbin was speaking to poultry producers just months after the FDA had banned chicken implants of the synthetic hormone diethylstilbestrol (DES) under the 1958 Delaney Cancer Clause, which deemed no food additive safe "if it is found to induce cancer when ingested by man or animal." By 1960, what the journalist and rancher Orville Schell called "modern meat" was turning out to be not quite so nutritious or pure as envisioned. Partnerships between the FDA, USDA, scientists, universities, and industry, and farmers were unraveling as concern over DES erupted into consumer consciousness.[2]

This essay explores how the use of synthetic hormones in livestock helped to create partnerships between industry, researchers, and regulators, and then disrupted those partnerships as consumers became increasingly concerned over the consequences of chronic exposure to synthetic chemicals. This essay comes from a chapter in a book manuscript that explores the ways hormonal histories can illuminate our current conflicts over endocrine disruptors.

Diethylstilbestrol (DES), synthesized in 1938 by the English biochemist Charles Dodds, was the first synthetic estrogen and the first synthetic chemical known to act as an endocrine disruptor. As I argue elsewhere, the synthesis of DES emerged during a larger debate occurring in the 1920s and 1930s about the carcinogenic effects of estrogens.[3] Nearly all researchers agreed that natural estrogens were carcinogenic, and that DES had the potential to be at least as carcinogenic, if not more so-- because it was more potent at exciting "estrogenic effects." In 1940, the FDA denied the drug companies' new drug applications (NDAs) for DES to treat the symptoms of menopause in women. In initially rejecting DES, Commissioner Walter Campbell of the FDA argued that regulators must follow what he called the "conservative principle." Given

the scientific uncertainty over DES's mechanisms of action and metabolism and over the applicability of animal studies to women, the FDA refused to approve DES--not because scientists had any proof that the drug would harm women, but because they had no proof the drug would *not* harm women. FDA regulators essentially adopted the precautionary principle sixty years before that term came into common usage.

Within months, however, political pressures on the FDA forced the agency to reverse its decision, in 1941 approving its use in menopausal women. By 1947, the FDA had abandoned its position of precaution, telling critics of DES that it was up to them to prove that DES had caused harm, rather than up to the companies to show that DES was safe. That same year, the FDA approved the use of DES implants in poultry, even though many staff within the agency urged against it, concerned about the entirely new issues posed by daily exposure, from birth to death, to novel estrogens in foods.

DES quickly became critical in the development of industrialized livestock systems in America. Feedlots, for example, would not have been possible without the synthetic hormone. DES allowed farmers to fatten cattle on grains so cheaply and rapidly that it made grass finishing seem inefficient and old-fashioned. Just a few years following its approval for use in beef, DES was given to nearly 90% of feedlot cattle in America. After high levels of hormones were detected in treated chickens, concern over DES effects began to grow in various lay groups, including farmers who handled treated livestock, workers who manufactured the material, and consumers who were eating meat from treated livestock. Between 2 million and 5 million pregnant women eventually took the drug in America, exposing themselves, their children, and even their grandchildren to higher rates of reproductive cancers, infertility, and birth defects. The metabolic byproducts of DES—wastes with potent estrogenic activity--from feedlots and from

people made their way into broader ecosystems, exposing a wide range of wildlife to the hormone. Endocrine disruptors in the post-war era changed the internal ecosystems of human, livestock, and wildlife bodies, interconnecting our bodies with our environments in increasingly complex ways.

Decisions to approve DES and other synthetic hormones in livestock occurred in a post-war context of technological enthusiasm and anxiety. As Deborah Fitzgerald argues in *Every Farm a Factory*, the industrialization of American agriculture began two decades earlier, in the 1920s. "In response to a crisis in postwar wheat farming, a number of non-farmers-agricultural engineers, economists, bankers, and government officials-determined that existing farms were too small to be profitable." Specialists from the new field of agricultural economics "urged farmers to embrace the mentality of industrial farming and to utilize the tools and machines that made it possible. Informed by Taylorism, this influential cadre of agricultural engineers and economists preached efficiency, standardization, mechanization, and careful quantification of farm inputs and outputs."[4] Industrialization was driven by a complex set of relationships between the USDA, agricultural equipment manufacturers, university experts that envisioned a model farming landscape with farms as factories, and the farmers who actually tried to do much of this transformation, often at significant cost to their families and their farms.

Agricultural industrialization was a product of what James C. Scott terms "high modernist ideology"—"a strong, one might even say muscle-bound, version of the self-confidence about scientific and technical progress, the expansion of production, the growing satisfaction of human needs, the mastery of nature (including human nature), and above all, the rational design of social order commensurate with the scientific understanding of natural laws."[5] The enthusiastic promotion of DES in agriculture and medicine offers evidence of Scott's high modernist

ideology. Nevertheless, a more pragmatic, skeptical perspective on the chemical miracle was evident as well during the same era, particularly among scientists, regulators, and consumers.

The Second World War witnessed a boom in chemical technologies developed for military purposes. After the war, these technologies were rapidly appropriated for agriculture, boosted by the enormous growth of advertising to consumers in the post war era. Consumer marketing promoted a technological optimism, particularly for the "chemical miracle" promised by advertisers, doctors, and extension agents. This optimism existed in an uneasy relationship with cold war anxieties—anxieties about risk from the communist threat, risk from new chemical technologies, risk from changing roles of American women. The use of synthetic hormones in livestock exploded in part as a response to these anxieties, yet synthetic chemicals also served to increase these anxieties.

Meat had powerful symbolic meaning for Americans, particularly during wartime. As the social historian Amy Bentley argues, American meat consumption was framed in gendered terms: meat made men manly, strong, and able to fight. Without meat, soldiers might not be willing or able to fight. A military pamphlet extolled meat's virtues: "Meat is one of the major providers of life--essential complete proteins needed to repair body tissue as it wears out, and to furnish the building blocks for new muscle and sinew in husky, hardy men who follow the sea." Bentley writes that "being deprived meant much more than going without meat for dinner; the absence questioned the very health and strength of American society."[6]

During the war, when pharmaceutical companies requested that the FDA approve the use of diethylstilbestrol to treat certain veterinary conditions in livestock, the FDA agreed, in part because of the urgency of producing meat for fighting men. Yet because of concern about the potential sexual threats to men who might consume estrogen residues, the FDA explicitly forbade

treatment of livestock that might be eaten. Could eating estrogen-treated meat turn men into girls? No one wanted to find out during the war. Even after the war, when DES was eventually approved for use in livestock headed for the dinner table, journalists rushed to reassure consumers that the hormone wouldn't demasculinize men the way it did roosters. One popular article noted that when a rooster was fed DES, he would soon be transformed into a "fat, complacent fowl that would rather cluck than crow and will ignore the prettiest hen that passes by….Roosters forget about crowing and prefer to cluck like matronly hens. They have little interest in breeding and become content to sit quietly and get fat….Tests, *so far*, shown that the drug does not affect humans who eat the treated birds…Thus the housewife need not fear that if her husband eats a stilbestrol chicken he will give up golf and hunting and start knitting sweaters," [emphasis added].[7] The journalist's "so far" suggested that men who ate too much treated meat might indeed be wary of finding themselves a little less manly, more interested in domesticity than in dead animals. One of the great ironies of promoting DES for livestock was clear: meat meant manliness, but if you used a female hormone to make more meat, what might that do to the gender identity of consumers?

When companies tried to push against these restrictions on the use of synthetic hormones in livestock, the FDA initially insisted on precaution, arguing that the absence of evidence of harm did not prove safety. For example, in 1944 one company wrote the FDA requesting permission to use DES for chemical castration of roosters intended for human food. The FDA refused, pointing out that safety studies had not yet been done on possible residues in the meat, and asserting that the burden of the proof of safety lay on the chemical company, not on the consumer.[8] The company would have to submit a full New Drug Application (NDA) with tests demonstrating the safety of any residues. The burden of proof rested on the company, and until

the company submitted test showing residues to be safe, the drug could not be used, insisted the FDA.

The chemical companies refused to submit safety tests, providing instead theoretical arguments purporting to demonstrate why no residues could possibly exist or harm consumers. In March 1945, for example, the company Wick and Fry developed what they called "sex hormone pellets" to be inserted high into chicken necks. They wrote to the FDA that no danger should be expected to consumers, because modern housewives would surely throw chicken necks out, thus avoiding the pellets as well.[9] The FDA was not impressed with this logic, responding:

> Three possibilities, however, should not be overlooked…we have no way of knowing that the poultry men will do as your label directs. He may find it easier and quicker to implant the pellets a little lower in the neck. Second, the housewife, who has no way of knowing that her purchase may have a pellet in its neck, may be a frugal person who cuts off the head right behind the skull and uses the entire neck meat in the gravy. Third, it is very possible that several lots of treated birds would be bought by a chicken cannery…utilizing every available particle of flesh on the carcass. In view of these comments and the fact that small quantities of diethylstilbestrol may produce very undesirable effects in humans, you can appreciate that the use of the drug for the purpose of fattening or tenderizing poultry causes us real concern.[10]

In other words, the FDA here insisted on empirical evidence rather than theoretical arguments about what model housewives might do in ideal worlds. One staffer scribbled on a memo in response to this application: "Some people do use the heads of poultry for food!"

As the war came to an end, pressures began to build on the FDA to approve use of diethylstilbestrol in meat meant for human consumption. War-time rationing of meat had come to an end in America, but food shortages throughout Europe threatened to lead to famine, and many people worried about the collapse of peace. Grains were increasingly being used to feed livestock rather than people, but Americans proved unwilling to voluntarily reduce their meat consumption. Rather than re-institute rationing, the government encouraged research partnerships devoted to learning how to stimulate meat production at home while also having enough grain to prevent

famine overseas. The answer appeared to be hormones, which allowed for more efficient utilization of grain in meat production. Animals treated with estrogen gained weight on grain more quickly, so the hope was that new agricultural technologies might let Americans eat more meat without guilt.[11]

Professors from agricultural research stations, feed mills, and agricultural products companies wrote numerous letters to the FDA inquiring about diethylstilbestrol to be used in livestock. Poultry was the first target, for roosters and turkeys responded cheaply and quickly to DES.[12] Internal memos within the FDA showed that many staff continued to remain skeptical about the use of diethylstilbestrol in livestock. Staffers noted that the existing assay method for detecting DES residues in meat was not specific to the drug -- for example "all phenolic containing substances would give a positive test."[13] Throughout 1946, the FDA rejected NDAs submitted for poultry in 1946, stating that "no information has been offered to show the amount of diethylstilbestrol remaining in the tissues of treated birds. Until it can be clearly shown that no significant quantity of the drug remains in the tissues which might be capable of producing undesirable effects in human consumers, we will not be disposed to consider any application for the diethylstilbestrol use with this purpose."[14]

In January 1947, the FDA reversed course and agreed to allow diethylstilbestrol to be used in poultry implants. None of the problems discussed in the FDA correspondence from the previous several years had yet been fixed. The only research on estrogen residues submitted by chemical companies was one study from White Laboratories, which actually showed estrogen residues did indeed migrate from the pellets into meat intended for human consumption. The NDA claimed that “the amounts of the synthetic estrogens deposited in the tissues were

insignificant from the standpoint of human consumption," yet the company presented no evidence in favor of this assertion.[15]

What had changed? Why did the FDA suddenly allow DES implants in livestock, when regulators had resisted for years? Pressures to increase meat production after the war were certainly great. Concerns about risk of estrogens for men had initially led the FDA to resist these pressures, but by 1947, the FDA's approach to risk had shifted. As described in the preceding chapter, in 1947, drug companies had just succeeded, with the help of enthusiastic doctors, in persuading the FDA that DES should be used in pregnancy. While initially dubious of the Houston doctor Karl John Karnaky's extravagant claims for DES, by 1947, FDA staff had lost their skepticism about Karnaky's results. Because Karnaky had treated pregnant women with large doses of DES and no deaths had yet resulted from these extreme exposures, the FDA staff decided that small doses must be safe as well.

To understand the FDA's shift from precaution, we need to examine FDA relationships with Canadian regulators. In 1947, when challenged by Canadian government officials and researchers about the decision to allow DES in livestock, the FDA staff simply referred to Karnaky's work.[16] The Canadians, like the Americans, were negotiating concerns about the potential risks of synthetic hormones in meat. Immediately after the FDA staff approved the pellets, they received a concerned letter from the Canadians which urged the FDA to be extremely careful with the use of diethylstilbestrol in animal products. A staff member from the Canadian Department of National Health and Welfare wrote:

> I noticed in Chemical and Engineering News February 3, 1947, that the Food and Drugs Administration had reported approval of an application for use in interstate commerce of the method of artificially caponizing male chickens using the dimethyl ether of diethylstilbestrol….We have been working on the problem with the poultry division of the Department of Agriculture and our results show that there is a residue of the estrogen in the cockerels, sufficient to change the vaginal smear of the menopausal woman. Of course

> this is not evidence of any harmful effects but it is possibly an undesirable reaction for some people.…We were planning to publish these results and are wondering if any of the results from your division had been published and we had overlooked them.[17]

The Canadians had data showing enough residue was left in the meat to change the vaginal smears of a woman past menopause--in other words, that a synthetic estrogen implanted in chicken necks was powerful enough that the meat could change the vagina of the woman who ate that meat.

Why were Canadian regulators able to find evidence of residues and their effects, while American regulators could not? Canadian regulators conducted their own research to determine the safety of drugs and synthetic chemicals. In contrast, American regulators were required to use research performed by the companies and reported by the companies as well. This meant that any results not favorable to the drug companies of the chemical companies might never be reported to the federal government. Research design was also vulnerable to the desire of the companies not to find adverse results. The Canadian government's tests found estrogenic effects of residues, while the companies in the United States didn't test for such a thing, and yet continued to insist no such effect existed. The FDA attempted to defend its position by finding flaws in the Canadian research.[18] The implication was that, if Canadians were observing estrogenic effects, it might well be because their research protocol was flawed.

In July of 1947, Dr. Glover from the Canadian government wrote back to the FDA, insisting on their concern about DES in meat. Glover noted:

> In the Canadian Poultry Review, February 1947, an item appeared which indicated that your Branch was satisfied from the research work that had been done that the implantation of diethylstilbestrol tablets as recommended, for tenderizing the meat of cockerels will not affect the ultimate consumers. In the May issue of the same magazine a letter to the Editor from S. Bird, Assistant, Division of Poultry Husbandry, Central Experimental Farm, Ottawa, indicates that the feeding of tissues from treated birds has repeatedly produced estrous changes in clinical tests with aged woman. As we receive numerous inquiries

> regarding the efficacy and safety of so-called 'chemical caponizing' I should appreciate it very much if you would clarify the issue for me.[19]

The FDA replied that "the periodical you refer to is not immediately available to us," and went on to assure the Canadians that "according to our medical staff a 15 mg pellet of this drug when used as directed would not cause any particular harm in humans from consuming such treated birds." In response to this letter from the FDA, the Canadian official replied that "a technical paper dealing with the subject of the administration of female hormones is shortly to appear in *Endocrinology.*" This was the major journal in the field, so the FDA's earlier comment about Canadian journals being too obscure for the FDA to consider was no longer valid.[20]

After the October 1947 issue of *Endocrinology* appeared, the FDA did move to forbid the use of DES in chicken feed, while allowing DES to be used in pellets.[21] Rather than insisting that these Canadian experiments be repeated with pellets to see if pellets and feed had the same response, the FDA assumed that pellets would show no such response, based on the assurances they had received from the chemical companies that no effects were to be expected. For years the FDA continued to insist on something that made little scientific sense: that although diethylstilbestrol from feed would accumulate in fatty tissues and pose a danger to humans, DES administered in pellets simply wouldn't accumulate.

When challenged by members of its own scientific staff, the FDA attempted to explain this logic by arguing that "it is possible to exercise a rigid control over the dosage in the [pellet] process and under these circumstances the estrogen does not accumulate in those portions of the treated bird which are consumed by human beings."[22] A host of assumptions about the possibilities of scientific control are embedded in this statement. First, the statement assumes that technology can offer enough control to sidestep dilemmas posed by pollutants. Second, the statement assumes that people live in an ideal world, one designed by technicians: that no one

ever sells a chicken head, that consumers eat what they're supposed to eat, that companies do exactly what they promise to do, that pellets release a specific, measured, infallible dosage that can be carefully controlled. But none of these assumptions were based on empirical evidence. The FDA had never received or examined any data that showed that pellets did release a reliable and controllable dosage, or that this dosage did not accumulate in tissues, even if it were controllable and reliable.

Things Go Wrong

The FDA soon began to receive warnings that something might be seriously wrong with their hormonal chickens. Evidence accumulated that DES was causing problems with chemical plant workers, farm workers, restaurant workers, and consumers. In 1947, Arapaho Chemicals of Colorado wrote to the FDA: "Our Company has recently been approached in regard to manufacturing stilboestrol…as raw materials for pharmaceutical formulation. We know that these materials are all readily absorbed through the skin and by inhalation. It is our belief that the physiological effect of these materials would constitute a decided industrial hazard. In order to properly evaluate the advantages of undertaking the manufacture of synthetic estrogens, it is necessary that we obtain as much information as possible about them in regard to the seriousness of the health hazard involved, recommended precautions for handling, treatment of affected individuals, cumulative effects, etc. We are particularly concerned over the possibility of carcinogenesis through long continued contact with stilboestrol."[23]

The FDA responded: "we have your letter of June 26, 1947 requesting information concerning the health hazard involved and the precautions necessary on the manufacture of stilbestrol... It is our understanding that excessive exposure to the substances may cause marked disturbances of the menstrual function in women and have a devirilizing effect in men. For this

reason it might be feasible for you to consider the employment of old rather than young men. The question of carcinogenic potentiality of these substances is one which cannot be answered with finality at this time. We regret be unable to be of much of assistance to you in this matter and suggest that you write to the Bureau of Industrial Hygiene of the United States Public Health Service the National Institute of Health, Bethesda, Maryland for further information."[24]

At the same time that the FDA was reassuring the media and consumers that livestock treated with DES were perfectly safe for human consumption, the FDA was acknowledging that male workers might become infertile and grow breasts, while the women workers might have their menstrual cycles completely disrupted. Internal memos distributed within the FDA and response to these letters show that FDA staff were quite concerned. One staff member wrote to another: "I've had two previous inquiries along this line, the most recent being from Ortho products. The greatest complaint concerns woman who have deranged menstrual cycles, and excessive bleeding…There is no question as to the de-virilizing effects on males, and it may result in permanently lowered virility and sterility."

Yet, rather than reconsidering approval of the drug, the FDA began to argue that people who complained of sterilization and cancer were failing to provide "material proof" of their case, ignoring the fact that it was not their responsibility to do so. One FDA internal memo complained: "The data on carcinogenesis is meager and many published opinions are not properly backed up on the facts. I personally doubt if most or not all of the people who have raised the question have failed to provide material proof of their contention. The other side, however, has adequate proof of the lack of carcinogenic activity of the estrogen."[25] This memo suggests that the FDA was beginning to view absence of proof of harm as a form of proof of safety--an interpretation that FDA founder Harvey Wiley had long warned against during the early years of the agency.

The FDA, even in the presence of the strong warnings of problems, insisted to consumers, doctors, and scientists that the synthetic estrogens had been shown to be completely safe. For example, just ten days after these internal memos circulated about problems in workers exposed to DES, a professor at Cornell wrote the FDA asking if diethylstilbestrol in livestock was really safe.[26] In its response to this letter, the FDA did not tell the Cornell professor about the Canadian research, or about the concerns from chemical manufacturers. The FDA simply dismissed the entire question of residues by referring to Dr. Karnaky's work and claiming that if the amount of DES in the original pellet were safe for a human, then the residues must also be safe.[27] Karnaky had long been the most enthusiastic promoter of DES use in women, and his reductionist logic was extended to livestock implants: if huge doses didn't kill pregnant women, small doses must be safe for everyone. The FDA moved from an insistence that no residues be present, to a refusal to even require measurement of the residues or consider that residues might be problematic.

Troubling findings began accumulating. A restaurant worker in New York grew breasts after eating the heads of chickens implanted with DES pellets, and his case became immortalized in a medical textbook. Mink farmers began complaining to the FDA that their mink were made sterile by residues from the necks of the implanted chickens. FDA staff discounted these complaints, testifying that "a few mink ranchers have alleged that their breeding animals were rendered sterile after having been fed the discarded heads of poultry which were implanted with diethylstilbestrol pellets. As yet we have seen no satisfactory data of a factual or scientifically acceptable nature showing that the offal from birds implanted with these pellets will actually cause sterility in minks or any other animals."[28] Yet mink farmers could not be expected to produce scientific data; they were farmers, not scientists.

Finally, five months after repeated reports of problems, the FDA checked the chickens, found numerous cases where residue levels violated the law, and seized 50,000 pounds of chickens. These chickens continued high levels of DES residues, and some birds contained up to 4 pellets in a single neck.[29] FDA staff had vigorously denied the very possibility this might have been happening—without ever bothering to check before issuing their assurances. When empirical data was initially presented to them by mink farmers among others, FDA staff simply denied it. Yet when they went out and collected their own empirical data, they found that their scientific models of what ought to be happening were not supported by actual evidence.[30]

Hormones in Beef

Even as research staff within the FDA and scientific consultants hired to advise the agency were urging the FDA to ban DES for poultry, the FDA approved its rapid expansion into cattle feeding. What were the rationales at work that made residues from poultry dangerous, while residues from beef seem like a reasonable risk for consumers to bear? The differences stemmed from the differences between synthetic chemicals delivered via implanted pellets, versus those delivered via feed. These differences might seem technical, which indeed they were, and in the FDA's concern with their technical differences, they reveal a host of assumptions about the possibilities of scientific control over nature.

In *Cancer from Beef*, the historian Alan Marcus examines the growth and development of DES use in beef. Briefly, in 1947 a graduate student at Purdue named W. E. Dinusson experimented with DES implants in heifers. He had noted that spayed heifers put on weight more slowly than intact heifers, so he hypothesized that, if less estrogen than normal slowed growth rates, then more estrogen than normal might increase growth rates. He found that DES did lead to

faster growth in the young cows, but the hormonal side effects—vulvar swelling, riding, and mammary development were too great to recommend use. DES implants "produced a nymphomaniacal stance" in heifers, and "the meat and liver of these slaughtered experimental animals retained significant estrogenic activity, a factor that rendered safe human consumption problematic."[31] DES pellets seemed like a dead end for cattle.

Several years later, three researchers at Iowa State University (W.H. Hale, his graduate student C. D. Story, and Wise Burroughs), decided to see if DES could be used in feed, rather than in implants. DES supplementation in feed had not worked for chickens, because their livers quickly broke it down, and because chicken food consumption cannot be closely controlled. Cows and sheep, on the other hand, were ruminants, metabolizing nutrients in unique ways. Australian researchers had long noted that certain clovers with high levels of phytoestrogens led to striking changes in the ruminants that grazed upon them. Some sheep had repeated miscarriages, which suggested that plant estrogens might be harmful to fertility. But other sheep and cattle had higher growth rates when eating estrogenic clovers, suggesting that supplementing cattle feed with estrogens might have interesting possibilities, particularly in animals headed for the butcher, whose future fertility was of little concern. Hale noticed a report in a British journal that indicated oral DES was rapidly "detoxified" in chickens but not in cattle, which suggested oral feeding of DES, at higher levels, to cattle might be useful even though it was useless in chickens. So Hale and Story decided to do some experiments with DES added to cattle feed. In one study, they found that lower levels of DES in feed improved weight gain, but higher levels had no effect. "The responses in the first two studies are unexplainable," complained one critic, because they violated normal dose response theories—but given what we now know about the effects of endocrine disruptors, the results were not particularly surprising.[32]

In 1953 Wise Burroughs published a report showing that "cattle gains could be increased substantially and that feed costs could be reduced materially by placing 5 mg or more of DES in the daily supplemental feed fed to each steer." Burroughs concluded that DES feeding led to 35% increases in growth and a decreased feed cost of 20%--astonishing results if they could be reproduced.[33] Burrough's university, Iowa State, was certainly intrigued by the financial possibilities of this research. The university entered into complex patent negotiations with the drug company Lilly, and did its best to create an expectant market among farmers for the hormone. As Marcus writes: "On February 19, 1954, lowa State College's Wise Burroughs, professor of animal husbandry, announced at a special lowa Cattle Feeders' Day that he had discovered a growth-promoting cattle feed additive." Iowa State skillfully manipulated this attention: as one witness recalls, "publicity about an exciting new discovery resulted in a huge and unexpected crowd (over 1,000)."[34]

The FDA almost immediately granted approval on November 1, 1954—just a year after the initial report from the feeding studies. The key requirement introduced to protect consumers from residues was simple: DES had to be withdrawn from cattle feed 48 hours before slaughter, in the belief that no DES residues would remain in the meat after the withdrawal period. The drug company Lilly worked with the academic researcher Wise Burroughs to devise tests for the FDA that would show that measurable residues would not remain after 48 hours. Yet these residue tests were not particularly sensitive, for they could not measure levels in beef that were high enough to cause biological changes in tissues, even though it was possible to measure those levels in poultry. But the FDA was satisfied that, at least in ideal laboratory conditions, beef would be safe from DES residues if these withdrawal periods were followed. Their experience with poultry farmers had shown that few farm operations were able to meet the expectations set by laboratory

conditions in which an exact withdrawal period led to pure meat. Most chickens treated with DES were contaminated, not clean, either because of simple negligence or outright duplicity. But instead of learning from these chaotic poultry results, the FDA simply argued that the problem lay with farmers, not with the hormone.[35]

A month later, DES went on the market as Stilbosol. Manufacturers such as Lilly marketed DES feeds intensely, to extension agents, to farmers, and to the farmer's press, and "cattlemen turned to the enhanced feeds in droves."[36] By late 1955, less than a year after DES went on the market, fully half the cattle in America were receiving DES. Soon, 80 to 95% of cattle received DES.

The research, FDA approval process, and marketing of DES did not happen by accident; it emerged as part of a complex partnership between drug companies, universities, and federal agencies. Researchers at Purdue had initiated studies on DES in cattle, but the Purdue administration did not believe that commercialization of new technologies was part of the university's academic role. Iowa State College, however, was quite happy to work with drug companies to profit from academic discoveries. After a series of confidential meetings, the university decided to grant an "exclusive 5-yr license under the patent to Lilly on July 29, 1954."[37] As Alan Marcus writes: "Indeed, the case of DES seemed to be a model of the application of the partnership idea. A college scientist uncovered a new technique, pharmaceutical scientists produced the drug, feed-manufacturing scientists compounded the material as a premix, federal scientists approved its use, agricultural college scientists publicized it by demonstrating its utility, and farmers made use of it. That type of expert-based interaction had been the model for 'progress' since the 1920s. With respect to stilbestrol, little in the mid-1950s seemed to undercut faith in that model."[38]

Thanks to these partnerships, DES transformed beef production practices. In the words of one cattle feed researcher, the synthetic hormone "tipped the balance from grazing to cattle feeding and encourage creation of large commercial feedlots in the western, midwestern, and southern states. Beef production would forever be changed."[39] Beef consumption nearly doubled during the drug's heyday between 1954 and 1972. Yet even as consumers ate more meat, they also worried about what might be lurking in that meat.

Growing Consumer Concern

During the 1950s and early 1960s, the FDA and USDA came under increasing public pressure from consumers concerned about chemical residues in their food, particularly pesticides and hormones. In 1950, workers in the FDA's Division of Pharmacology found that DDT was showing up in the fat of ordinary Americans. Baby food manufacturers began testing their sources, and announced that they could not find vegetables free of pesticide residues.[40] When DDT residues were found in milk—that epitome of pure food good for children—consumer concern began to build, and soon meat became a focus of concern as well.

Meanwhile, the regulatory agencies were confronted with an increasingly industrialized food system, with producers that were transforming American agriculture into centralized agribusiness. How were these regulatory agencies to deal with these conflicting pressures, and with an increasingly conflicted sense of mission? Was their job to protect consumers and regulate industry? Or was their job to promote industry, to serve as partners with producers? The government tried to believe that, with science, they could do both simultaneously.

In a 1951 speech, an FDA representative, Dr. Collins, spoke to the livestock industry, warning his audience of growing consumer concern. Collins said: "increased public interest in

what goes into foods is shown by the recent activities of the House of Representatives' Select Committee to Investigate the Use of Chemicals in Food Products. But at the same time there has been a growing tendency among livestock people to employ drugs to promote fattening, stimulate milk production, and bring about other physiological changes in domesticated animals and poultry."[41]

As Collins noted, these two trends—increasing concern, and increasing chemical use--were about to collide. Regulatory agencies were caught between political pressures from lobbyists and growing pressure from consumers. To the FDA and livestock industry, the answer seemed to be in closer partnerships between industry and universities, with the federal agencies helping to foster scientific research and promote new drugs. In 1954, this faith in science as a way of defusing consumer concern about pesticides found expression in the Miller Pesticide Amendment. The Miller Pesticide Amendment allowed residues of toxic chemicals to be present on food, below a tolerance level assumed to be safe.[42] The FDA was responsible for setting tolerances, which were defined as the levels of residues that people could, in theory, tolerate without harm. The Amendment reflected a faith that scientists could measure and understand risk, and that anything that couldn't be measured simply didn't matter.

But as post-war anxiety about the chemical age grew, fed by anxieties over radiation exposure, nuclear technologies, pesticides, and new drugs, more researchers begin joining with consumers to express doubts that *any* residue of a known carcinogen could be safe. In 1955, a report in *Science* warned that mouse feed had been prepared in a mill that had previously been used to prepare cattle supplements, and that mouse feed had been cross-contaminated with DES, leading to "serious reproductive disturbances" in the mice."[43] This finding, published in the

leading American journal of science, began to stir renewed concerns over hormone residues in food.

At a 1956 FDA Symposium on Medicated Feeds, several scientists urged against feeding livestock DES, citing decades of experimental evidence that anticipated many of the findings of endocrine disruption research five decades later. In particular, they were concerned about results that showed small doses of DES could be more effective at inducing cancer than large doses, just as lower doses of DES were more effective at inducing weight gain in the cattle. This was particularly troubling for animal feed, because the arguments for the safety of small exposures through feed were all based on Karnaky's assumption that, since pregnant women didn't seem to be harmed the large doses of DES given to them, much smaller doses should surely be even safer.

As concern about the harmful effects of new synthetic chemicals increased during the 1950s, Congressman James J. Delaney's House Select Committee to Investigate the Use of Chemicals in Foods and Cosmetics conducted a two-year inquiry into chemical additives in food. Chemicals that might leach from plastic wraps and containers into food were included in the hearings, as was DES in livestock. Many researchers urged additional research on chronic toxicity and more controls in their use, arguing that not enough evidence showed DES or DDT were safe for general prolonged use.[44]

The FDA supported the need for changes in the law, calling for the need to show safety before a chemical could be marketed, just as was required for human drugs. In contrast, the USDA was sympathetic to industry, with economic entomologists on its staff arguing at the hearings that "research…could go on forever without disclosing all possible hazards from expected uses."[45]

Disputes over the law were not simply about science and safety; they were fundamentally about regulatory power. Industry representatives and farmers' groups continued to challenge the power of government to regulate their activities. "I know of no other industry which has to comply with more laws and regulations in order to sell its product," complained Lea S. Hitchner, president of the National Agricultural Chemicals Association. Samuel Fraser, secretary of the International Apple Association, characterized the push for new laws as a "grab for power which is to be secured under the whip of hysteria."[46] Consumer groups pointed out that, without strong regulation, nothing protected ordinary people from the growing power of industry.

In 1958, the Delaney hearings resulted in an amendment of the Federal Food, Drug and Cosmetic Act, which created new regulatory policies for food additives. The 1958 Food Additives Amendment required that the food products and packaging industry demonstrate to the FDA that all food additives--including packaging material and food related-materials such as can liners and plastic wrap--were safe. Nevertheless, as the historian Sarah Vogel argues, this amendment weakened American regulatory conceptions of risk. The earlier food laws of 1906 and 1938 had considered dangerous chemicals as "hazards per se"—in other words, hazards in and of themselves, regardless of exposure levels, and therefore theoretically restricted from entering food at any level. But the 1958 amendment reversed this standard, "based on the logic that many of the new industrial chemicals in use, in particular the pesticide DDT, increasingly detected in cow's milk, were 'necessary in production or unavoidable.'" The amendment formally conceptualized risk "as relative to the amount of exposure to a given chemical" in food, including pesticides, artificial colors, and plastics that might leach into food. Vogel writes: "With the passage of the 1958 law, the regulation of chemicals in foods shifted from the *per se* rule to the *de minimus* standard that inscribed into law the notion that chemical risks were a function not of the hazard

itself, but dependent upon the exposure. Safety, in turn, could be achieved not by questioning the hazard *per se*, but by minimizing the exposure."[47]

The 1958 Food Additives Amendment required that any company wanting to add a substance to animal feed had to show that it was safe for the animal, and that any parts of the animal meant to be eaten by humans did not contain residues, or that the residues it did contain could be shown to be safe for human consumption. In other words, if a livestock producer wanted to add cement dust to cow food to increase weight gain, that producer had to show that the dust was safe for the animal, and that the meat of the cow didn't contain any residues from that dust. If a producer wanted to add growth hormones to dairy cattle feed, they had to show that the hormones wouldn't hurt the animal and that residues wouldn't be present in the milk—or if they were present, that those residues would be safe for human consumption.[48]

Carcinogens were the exception, for no safe level of exposure was known to be achievable. Congressional Representative James Delaney (D-NY) persuaded Congress to pass what became known as the Delaney Cancer Clause. This had the potential to be revolutionary, for it stipulated that any substance known to cause cancer in test animals could not be added to food--in any quantity whatsoever. No one needed to prove that the quantities in question caused cancer; no one needed to prove that animal studies applied to humans. Essentially, this clause overturned the basis of the toxicology threshold tolerance paradigm, which had assumed that the dose makes the poison, and that therefore a small enough quantity of a carcinogen wouldn't cause harm. Instead of assuming, as earlier FDA policy had assumed, that if we couldn't measure it, it simply didn't exist, the Delaney Cancer Clause recognized that risks might exist beyond our power to measure or model them.[49]

The industry was furious about the Delaney Cancer Clause. In 1961, the National Institute of Animal Agriculture's annual conference highlighted the growing tensions. The agricultural producers were in an uproar, and the FDA came to try to calm them down. The Vice President of Lilly thundered that the Delaney Clause "is a triumph of superstition over science.... if the spirit of this clause were exercised to the extreme, if we eliminated from our lives everything that causes cancer in animals, mankind would be reduced to sitting around in the dark, naked and hungry, waiting to die of cancer."[50]

Speaker after speaker at this meeting sounded the same theme: while the public might be scared, that was silly, and to fight the communist threat, scientists, industry, and government had to join together to reassure a nervous public. Nothing less than the free market system was at stake. The President of the American Meat Institute, Homer Davison, said: "Fundamentally we all believe in one basic philosophy. That philosophy accepts the free market system as a base. From there it argues that by increasing the production of animal protein foods, the health of agriculture and the health of people can and will be improved. Thus our thesis is very simple."[51] More meat, in other words, meant fewer communists. The freedom of the nation depended on cheap, abundant meat, and that meant hormones.

By 1962, livestock producers had gained enough support in Congress to modify the Delaney Clause with the so-called DES proviso.[52] The exemption permitted the use of DES in livestock meat production, provided no detectable residues were found in the edible tissue. It may seem simple to say: a chemical is allowed in livestock feed, just so no residues end up in the meat. But to regulate based on this constraint assumes many things: first, that someone is actually testing for residues. Second, that tests exist which can detect biologically relevant levels of residues. Third, that those tests are reliable and reproducible, so that a negative finding is

trustworthy. Fourth, that farmers follow the set protocols exactly for giving the animals the right animal of the chemical, and then for withdrawing it from their feed. Fifth, that a small sample of meat on the market—say, 1% or less--is a reliable indicator of all the meat on the market. And finally, that animals submitted for sampling are being treated in the same way as other animals. All these assumptions rested on the fundamental assumption that industry would act in the best interests of consumers, rather than their stockholders, without strong government oversight, an assumption which had not been supported by historical evidence, to put it mildly.

The drug companies assured the FDA that they had done numerous tests that proved all traces of the drug would be gone after 48 hours, so no residues could possibly appear. On these grounds, the FDA agreed, even though they already had plenty of evidence in the poultry cases that theoretical arguments meant little in the real world, and that it wasn't easy to closely monitor what producers did in livestock operations, particularly when Congress refused to allocate funds for monitoring.

The FDA and USDA did not begin testing meat regularly for residues until 1965, and once the FDA and USDA did begin testing, they found violations every single year. Moreover, the testing protocols used by the USDA were only capable of detecting DES down to levels of 10 ppb. Yet by 1964, DES had been shown to cause tumors in mice when fed at 6.5 ppb, and it was impossible to find a "no effect" level where residues were safe. "Hence even meat shown to be clear of DES by the USDA's method could still contain dangerous quantities of DES."[53]

At first, the USDA only sampled 600 cattle each year—out of the tens of millions of cattle treated with DES--and those samples were not tested with particularly sensitive assays. Still, in 1965, 0.7 % of those cattle showed residues of DES; 1.1 percent in 1966, 2.6 percent in 1967, and 0.6% in 1970. These were clear violations of the law, showing that the DES proviso

wasn't working, for millions of cattle were indeed showing up in the nation's food supply with residues of a carcinogen. Yet, rather than banning the chemical, the FDA simply decided that the problem lay with cattle growers who weren't obeying the regulations properly (rather than with the unsupportable assumptions of the regulations themselves).

Instead of altering regulations or strengthening enforcement, the USDA cut its residue testing program in half in 1970, and the FDA inexplicably allowed more than twice the amount of DES to be used in cattle. "This order means that most of the cattle going to market may have received twice as much DES as formerly,"[54] the FDA noted in a glowing press release to livestock producers. Consumer groups found this turn of events infuriating.

During the Delaney hearings, the FDA staff had recognized the need for better consumer protection from unregulated hormone and pesticide residues. The Delaney Cancer Clause was a formal, legal expression of precaution. Yet in its focus on carcinogens, it also distracted attention from other potential endpoints such as endocrine disruption. The shape of the 1958 amendment reflects this split: for carcinogens, the law required almost complete precaution; for everything else, the law allowed a much relaxed threshold model.

When it came time to regulate, the FDA increasingly found itself speaking for industry, rather than for consumers. What explains this puzzle? During the war, when companies tried to pressure the FDA to extend approval of DES, the FDA responded with pragmatic skepticism. Instead of engaging in a theoretical argument with Wick and Frye about whether residues might possibly make their way from pellets into food, the FDA insisted on empirical tests of actual meat. The FDA figured that it was important to consider how people actually raised, slaughtered, processed, and ate animals. By the 1950s and 1960s, however, regulators sided with the industry, agreeing that they didn't need to do empirical tests of safety, relying instead on assumptions about

model organisms in model worlds. One of the key shifts that happened after the war was a turn in the regulatory agencies toward a faith in quantification and simplified theoretical representations of the world, accompanied by a turn away from the messy complexities of actual cows, people, and farms.

For boosters, hormones promised not only cheaper meat, but potential victory against communism, and even against the uncertainties of nature itself. In one stirring address, Frederick Andrews, an animal scientist at Purdue University who did some of the earliest research on DES in beef, exhorted his fellow meat scientists to join the struggle:

> Unfortunately, many of our contemporaries think of chemicals as inherently bad. These are the people who think of nature as beautiful, sympathetic, and basically pure and who resent the advances in knowledge which have led to the development of chemical fertilizers, hormones and antibiotics…They believe that the Age of Chemistry is unnatural, dangerous, and on the whole, opposed to nature….[But] nature has not been particularly gentle. From the origin of the simplest living forms until the present, all living things have been pitted against each other in a cruel struggle for existence… For man is now pitted against man as well as against the elements of nature….Those of us who are working with the life processes, whether concerned with insect and weed control, crop production, milk, meat or egg production, are fully aware of our responsibilities….Where dangers exist, we will eliminate them, for we have every intention of keeping the people of the United States the best fed, best housed, and best medically cared for in the world.[55]

Andrew's vision of a world free from famine, thanks to scientific technologies and government partnerships, was shared by many.

As mentioned earlier, this essay is part of a larger project which asks: Why have we retreated from the precautionary principle, failing to regulate many chemicals such as DES that were known to cause harm even during debates over their initial regulatory approval? Many environmentalists argue that greed drives the abuse of toxic chemicals. Greed drives Syngenta to push the endocrine-disrupting herbicide atrazine and suppress data suggesting toxicity. Greed drives General Electric to slow the cleanup of PCBs it dumped into the Hudson River. And greed

drives the chemical companies in their relentless efforts to minimize the risks of bisphenol-A in common plastics.

While economic incentives certainly shape corporate and individual behavior, understanding why endocrine disruptors quickly permeated the environment and our bodies requires that we do more than just follow the money. Scientists who promoted hormone use within the USDA and FDA were not driven by hopes of financial gain; rather, they were motivated by their faith in scientific expertise and their belief that technological innovation meant progress. When my father's family, in their enthusiastic pursuit of better farming through chemistry, listened to extension experts and used vats of toxic chemicals they could hardly afford, they weren't driven by greed, but rather by a complex mixture of faith in scientific agriculture, the desire to create a good place on earth through hard work, a belief in technology, and a frustration with seeing their efforts destroyed by a few bad years of insect damage. Natural variation was not their friend. Chemicals seemed to smooth the vicissitudes of climate, markets, insects, disease, and disorder itself. As increasing evidence of hormonal toxicity came to public attention in the 1960s, this faith in the chemical miracle proved difficult to disrupt, even among the consumers and workers whose health was most undermined by the synthetic chemicals that saturated their daily lives.

Endnotes.

NARA, FDA: National Archives and Records Administration at College Park, Maryland. RG 88, Records of the Food and Drug Administration.

1. Durbin and Weilerstein, "The Food and Drug Administration, Poultry Feed Additives and Drugs," NARA, FDA.

2. For more on hormones and these partnerships, see Orville Schell, *Modern Meat: Antibiotics, Hormones, and the Pharmaceutical Farm* (NY: Random House, 1984).

3. Langston, Nancy. "The Retreat from Precaution: Regulating Diethylstilbestrol (DES), Endocrine Disruptors, and Environmental Health." *Environmental History* 13 (2008): 41-65.

4. Deborah Fitzgerald, *Every Farm a Factory: The Industrial Ideal in American Agriculture* (New Haven: Yale University Press, 2003). The quote is from a review of Mark Finlay, review of *Every Farm a Factory*, in *History* 32 (2004): 95.

5. James C. Scott, *Seeing Like a State: How Certain Schemes to Improve the Human Condition have Failed* (New Haven: Yale University Press, 1998), 4.

6. The military pamphlet is quoted in Amy Bentley, *Eating for Victory: Food Rationing and the Politics of Domesticity*. Champaign, IL: University of Illinois Press, 1998, 92-93. The second quote is from Bentley herself, 111.

7. Gail Compton, "Pill Turns Rooster into Tender Soul; Female Hormone Does Trick." Clipping from the Chicago Tribune, 2/1/48, Circulated and initialed by sixteen FDA staff, including Moskey, Larrick and Dunbar. FDA, NARA, A1, Entry 5, General Subject Files, 1938-1974. 1948. Folder 526.1.

8. C. W. Crawford, Assistant Commissioner of Food and Drugs, FDA, to Mr. James A. Austin, Jensen-Salsbery Laboratories, Inc., Kansas City, MO. 5/8/44. NARA, FDA. A1, Entry 5, General Subject Files, 1938-1974. 1944a. Folder 526.1-526.11..

9. Letter, Mr. Harry J. Wick, Wick and Fry, Indianapolis, IN. to FDA. 3/20/45, NARA, FDA, A1, Entry 5, General Subject Files, 1938-1974. 1945a. Folder 526.1.10.

10. Letter, P.B. Dunbar, Commissioner, FDA to Mr. Harry J. Wick, Wick and Fry, Indianapolis, IN. 10/15/45, NARA, FDA, A1, Entry 5, General Subject Files, 1938-1974. 1945a. Folder 526.1.10. Dunbar insisted that Wick and Fry had to "show by actual experiments with your own product that no diethylstilbestrol remains in the tissues of treated birds at the time of slaughter. The experiment should be conducted by experts qualified by scientific training and experience to evaluate the safety of drugs."

11. Bentley, in *Eating for Victory*, discussed the dilemma of post-war meat rationing and grain, but does not mention hormones as a solution. In "Growing with the 1960s," conference participants were enthusiastic about hormones as a solution to meat shortages and the threat posed by hungry consumers who might turn to communism if denied their steaks.

12. Moskey to Hutt, 9/13/45; Vogelman to FDA, 2/13/46; Moskey to Vogelman, 3/7/46; Ellerbusch to FDA, 1/25/46; all NARA, FDA. During this period -- October 1945 -- poultry makers were illegally beginning to implant chickens meant for slaughter with diethylstilbestrol pellets. For example, on October 8, 1945, the FDA office in Washington DC wrote to a

Midwestern office ordering it to seize diethylstilbestrol pellets that had been made in a Kansas City laboratory for fattening and tenderizing poultry. Dunbar to Central District Administration, 10/8/45, NARA, FDA.

13. Anonymous handwritten memos from FDA staffer for circulation, initials unreadable (WBW? And W?P), 2/5/1946, NARA, FDA.

14. Dunbar to Bryce, 2/13/46, NARA, FDA.

15. Dunbar to Bryce, 1/28/47; Moskey,memorandum of interview with Sondern and various staff of White Laboratories, 1/30/47, NARA, FDA.

16. Moskey to Robertson, 7/21/47, NARA, FDA.

17. Pugsley to Vos, 2/20/47, NARA, FDA.

18. Dr. Moskey of the medical division of the FDA replied to the Canadians: "this administration has conducted no research or investigational work along this line and we would greatly appreciate receiving a copy of any report you may publish up your own investigations." Moskey to Pugsley, 3/6/47, NARA, FDA. Not all of the response remains in the files, but what remains states: “There are some points in the Canadian work that need clarification. Such as, how much of the fowl (leg, wing, breast) was consumed, at how frequent intervals, for what period of time, how was the estrogen applied, pellets, feeding, and over how long a period?” Anonymous unsigned letter to Vos, regarding the 2/20/47 letter from Pugsley, 2/27/47, NARA, FDA.

19. Glover to Office of the FDA, 7/28/47, NARA, FDA.

20. Moskey to Glover, 8/6/47, NARA, FDA. Glover to Moskey, 8/8/47, NARA, FDA.

21. Moskey to Austin, 9/10/47, NARA, FDA.

22. Crawford to Montgomery, 4/13/48, NARA, FDA.

23. Waugh to Stormont, 6/26/47, NARA, FDA. The letter was circulated within the FDA for handwritten and initialed comments

24. Handwritten comments by FDA staff on the letter from Stormont to Waugh, 7/9/47, NARA, FDA.

25. Waugh to Stormont, FDA, 6/26/47, NARA, FDA.

26. The professor asked: "has experimental evidence been submitted to substantiate this question that the level left in the tissue of the bird is below the amount necessary to affect people who have consumed a bird? I would greatly appreciate any information along this line that you can divulge, particularly with reference to the residual effect of this hormone in the tissue of the birds." Robertson to Moskey, FDA, 7/11/47, NARA, FDA.

27. The chicken case is discussed in N. Wade, “DES: A Case Study Of Regulatory Abdication,” *Science* 177 (1972): 335–7. For the mink discussion, see Moskey wrote: “It was the consensus of our medical staff that even if 15 mg pellets of diethylstilbestrol were to be accidentally swallowed by humans, there would be no harmful effect, and it was on this that we made the new drug application's effective. In this connection you may be interested in the two articles published by Dr. K. Y. Karnaky in the Southern Medical Journal….While it is our understanding that there are experiments being conducted to determine the amount of synthetic estrogens left in the tissues of birds from prolonged administration of diethylstilbestrol in the feed, we are not aware that the results of such experiments have as yet been published.” Moskey to Robertson, 7/21/47, NARA, FDA. The scientific research on mink responses to being fed DES saturated chicken include: Howell and Pickering, “Suspected synthetic oestrogen poisoning in mink,” and Sundqvist et al, “Reproduction and fertility in the mink.”

28. Collins, “Drugs for Food-Producing Animals and Poultry are a Problem,” NARA, FDA.

29. The speech stated: “recent investigations have shown the directions for use are not being followed in all cases, with the result that pellet residues are very often found in the edible tissues. Examination of treated poultry when marketed disclosed that many of the treated birds contained pellet residues in the edible neck area. The Food and Drug Administration also developed evidence that partially unabsorbed pellets were present in some birds after they were completely dressed by butchers for home consumption," Collins, “Use and Abuse of Diethylstilbestrol Pellets in Poultry,” NARA, FDA.

The seizures of poultry in August 1951 shocked many. By mid-August of 1951, about 50,000 lbs. of chickens had been seized by the federal government. As Collins told his audience, "most of the birds examined from the seized lots contained partially unabsorbed residues 1 in. or more below the base of the skull... residues examined chemically contained from 2.9 to 9.3 mg check these numbers of diethylstilbestrol. A few birds were found to contain more than one pellet residue. Two pellets, one broken pellet and a fragment of a fourth pellet, with a total diethylstilbestrol content of 24.3 mg, were found improperly implanted in a single neck," ibid.

30. After residues were discovered, the FDA moved to ban DES use in poultry. Nine years passed before the FDA finally called for a “voluntary discontinuance.” DES in chickens was eventually banned in 1959, but the poultry industry took the FDA to court and the ruling was not upheld until 1966. “Hormones & Chickens,“ *Time*.

31. Alan Marcus, *Cancer from Beef: DES, Federal Food Regulation, and Consumer Confidence*, Baltimore: Johns Hopkins University Press, 1994.Marcus, *Cancer from Beef.* For the first quote, see A. P. Raun,and R. L. Preston. “History Of Diethylstilbestrol Use In Cattle.” *American Society of Animal Science* (2002) www.asas.org/Bios/Raunhist.pdf (accessed July 23, 2008). The second quote is from Marcus, *Cancer from Beef*, 78. See also Terry G.Summons, “Animal Feed Additives, 1940-1966.” *Agricultural History* 42 (1968): 305-313, and Alan Marcus, “The Newest Knowledge of Nutrition: Wise Burroughs, DES, and Modern Meat.” *Agricultural History* 67 (1993): 66-85.

32. Raun and Preston, “History of Diethylstilbestrol Use.” Summons writes: “In 1951 animal nutritionists at Iowa State University noticed that lambs on a certain fattening ration gained weight much faster than anticipated. The gains resembled those obtained from hormone pellet implantation. Puzzled, researcher Wise Burroughs and his co-workers at Iowa State brought samples of the lamb ration into the laboratory for analysis. They found that certain parts of the ration showed hormone activity. Further research indicated that a wide variety of cattle and sheep feeds occasionally showed hormone activity.”

33. Raun and Preston, “History of Diethylstilbestrol Use.” Burroughs et al*,* "The Effects of Trace Amounts of Diethylstilbestrol,” 66, 67.

34. Marcus, “The Newest Knowledge of Nutrition.” See also Raun and Preston, “History of Diethylstilbestrol Use.”

35. As the science journalist Nicholas Wade wrote in 1972, in his *Science Magazine* expose of the FDA’s failures to regulate DES: DES “The methods available in 1959 were good enough to pick up DES residues in poultry but not in sheep or cattle. The Delaney anticancer law of 1958 says unambiguously that no known carcinogen shall be allowed in food, so the FDA had no option but to prohibit the use of the hormone in poultry. It was clearly only a matter of time before detection methods

improved sufficiently to pick up DES residues in beef and mutton. The FDA was not hurrying, however, and in 1962 someone persuaded Congress to emasculate the Delaney law as it affected DES.

The new clause, a piece of fine-print chicanery known as Section 512 (d) (1) (H) of the Food, Drug, and Cosmetic Act, said that it is okay to feed carcinogens to meat animals, as long as no residue is left in the meat when the chemical is used according to label directions that are "reasonably certain to be followed in practice." In other words, if you find DES in meat, that's the fault of the farmer for disobeying the "reasonable" regulations. So don't ban DES, jail the farmer." Wade, "DES: A Case Study of Regulatory Abdication."

36. Marcus, "The Newest Knowledge of Nutrition," 66, and *Cancer from Beef*, 22-25. This essay lists examples of various press reports, including "Can Hormones Increase Cattle Gains?"; "Putting Feed Research to Work,"; "Hormones-New Beef-Gain Booster,"; "Faster, Cheaper Beef Gain,"; "'Dynamite' Drug Boosts Gain."

39. Burroughs et al, "The Effects of Trace Amounts of Diethylstilbestrol," and Burroughs et al, "The Influence of Oral Administration of Diethylstilbestrol to Beef Cattle."

37. Raun and Preston, "History of Diethylstilbestrol Use," citing T. W. Perry, personal communication. They write: "At that time, J. F. Downing had the responsibility for finding and developing new animal products for the recently formed Agricultural Products Division of Eli Lilly and Co., Inc. The president of Specified, Inc. (an agriculture/pharmaceutical company) in Indianapolis, IN, Downing's previous employer, was returning to Indianapolis after attending a Cattle Feeders Day program at the University of Minnesota. Seated in front of him on the plane were two people discussing the results of the DES studies at Iowa State. As soon as the plane landed, the president of Specified, Inc., called Downing and passed on what he had heard. Downing immediately contacted the Lilly patent counsel, called Burroughs, and arranged a meeting at Iowa State the following day. Iowa State had made contact earlier with a potential DES manufacturer for development of the product but had received a noncommittal response. Lilly, also a manufacturer of DES, came to the meeting ready to make a commitment to further research and development. Lilly also possessed some manufacturing technology that was critical to the safe handling of the drug. As a result of this meeting, and after the president of Iowa State University, J. H. Hilton, met confidentially with interested parties in agriculture, Iowa State College granted an exclusive 5-yr license under the patent to Lilly on July 29, 1954 (Willham, 1996). Lilly worked with Iowa State College in developing the data needed for the approval of DES by the Food and Drug Administration (FDA)."

38. Quotes: Marcus, "The Newest Knowledge of Nutrition," 66- 67, and Marcus, *Cancer from Beef*, 25.

39. For figures on the rapid growth of the DES beef market, see Raun and Preston, "History of Diethylstilbestrol Use."

40. Diana Dutton, *Worse Than the Disease: Pitfalls of Medical Progress*. Cambridge: Cambridge University Press, 1988, 65.

41. Collins, "Drugs for Food-Producing Animals and Poultry are a Problem," NARA, FDA.

42. Multiple authors, "Growing with the 1960s," NARA, FDA, 70. In 1954 the Miller amendment, or the Pesticide Residue Amendment, was passed. For the first time, this required that producers had to show residues to be safe, not just necessary. The applicant also had to

provide scientific evidence and testing techniques on which safety was determined -- and most importantly the burden of proof was shifted from the government to the producer. Producers that wanted to promote a pesticide chemical first had to secure evidence about its toxicity to animals and the amount that would remain on food after its use. Producers that wished to sell a pesticide used on livestock "must first secure evidence about its toxicity to animals, the amount required for the particular purpose, and the amount that will remain on the food after its use. These facts are submitted along with others to the Food and Drug Administration with a request to set a formal tolerance for safe residues of the chemicals on a specific raw agricultural product," Durbin and Weilerstein, "The Food and Drug Administration, Poultry Feed Additives and Drugs," NARA, FDA.

43. Dutton, *Worse Than the Disease*, 61.

44. U.S. Congress, "Chemicals in Food Products." Ann Vileisis in *Kitchen Literacy: How We Lost Knowledge of Where Food Comes From and Why We Need to Get It Back*. Washington DC: Island Press, 2007, has an excellent discussion of consumer concerns and the Delaney hearings, 160ff.

45. Thomas Dunlap, *DDT: Scientists, Citizens, and Public Policy*. Princeton: Princeton University Press, 1981, 67 and 69.

46. Dunlap, *DDT,* 68. With consumers expressing growing concerns about carcinogens in food, the chemical industry mobilized to prevent regulatory action. The Manufacturing Chemists' Association (MCA) hired the firm Hill and Knowlton (H&K), strategists from the tobacco industry, to lobby Congress, and they were successful in weakening regulatory action. Michaels and Celeste Monforton, "Manufacturing Uncertainty."

47. Sarah Vogel, "From the 'Dose Makes The Poison' to the 'Timing Makes The Poison': Conceptualizing Risk in the Synthetic Age." *Environmental History* 13 (2008): in press. Vogel quotes House Hearings before a Subcommittee of the Committee on Interstate and Foreign Commerce House of Representatives. *Federal Food, Drug, and Cosmetic Act (Chemical Additives in Food) on H.R. 4475, H.R. 7605, 7606, 8748, 7607, 7764, 8271, 8275.*84th Congress, 2nd Session. Statement of George Larrick, February 14, 1956.

48. Cook, "The Food and Drug Administration and Feed Additives," NARA, FDA, 3.

49. Moreover, the FDA's interpretation of the Delaney Cancer Clause explicitly argued that animal studies on cancer were sufficient for interpreting human effects. An FDA staffer, Alfred Barnard, argued in one speech to the Nebraska Livestock Feeders Association that to require a clear demonstration that a chemical caused cancer in humans would be, in effect, turning people into guinea pigs: "I am sure that no one here would argue that we return to the out-dated and now essentially outlawed practice of using the American public as a reservoir of guinea pigs for experimentation with carcinogenic or otherwise untested or suspect substances," Barnard, "Foods and Feeds- Current Problems," NARA, FDA. The Delaney clause originally prevented manufacturers from using food additives known to cause cancer in animals. The clause was extended in the 1960s to prohibit the presence of carcinogenic pesticides in processed foods (but not in fresh produce). This had the perverse effect of outlawing some pesticides in canned foods that were permitted in fresh produce US may relax rules on carcinogens in food.

For the reversal of the Delaney Clause in 1996, see Kleiner, "US May Relax Rules on Carcinogens in Food."

50. Multiple authors, "Growing with the 1960s," NARA, FDA, 57.

51. Ibid, 7.

52. This was part of the 1962 Kefauver-Harris Amendment, which required that drugs be shown to be effective, not just safe. Vogel, in "From the 'Dose Makes the Poison," writes: "In 1962, the Delaney clause was effectively watered down by the DES Proviso, an amendment to the 1958 food law, which permitted the use of a carcinogen, diethylstilbestrol (DES), a drug used to increase livestock meat production, provided no detectable amount of the chemical was found in the edible tissue. Risk, according to this proviso, was tied to quantifiable detection." For a fuller treatment, see Wargo, *Our Children's Toxic Legacy,* 106-07.

53. Wade, "DES: A Case Study of Regulatory Abdication."

54. FDA Memorandum, "FDA Position on Diethylstilbestrol (DES) Synthetic Hormone for Beef and Sheep," 1/20/71, included in U.S. Congress, Senate Committee on Labor and Public Welfare, Subcommittee on Health, chaired by E. Kennedy. Hearings on "Regulation of Diethylstilbestrol (DES), 92 Congress, Second Session, July 20, 1972 (Washington DC: US Government Printing Office, 1972), 153.

55. Ibid, 26.