NATURE'S HORMONE FACTORY

ENDOCRINE DISRUPTERS IN THE NATURAL ENVIRONMENT

Jonathan Tolman

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EXECUTIVE SUMMARY

For millions of years plants have been quietly producing chemicals. Through countless generations they have been perfecting a potpourri of chemicals, some benign some deadly. As the ability to detect, isolate, measure and test chemicals found in nature has progressed a starling fact has emerged: hundreds of plants appear to produce endocrine disrupters.

Many of the plants that produce phytoestrogens and other endocrine disrupters are edible. In laboratory test more than 43 plants and foods found in the human diet have been shown to be estrogenically active. Many phytoestrogen containing plants are common elements of our diet. Such grains as corn and wheat form a significant part of the human diet. Many legumes have also shown a surprising capacity for phytoestrogen production.

Although much more work has been done on phytoestrogens, some work has been done on plant chemicals which are known to effect the production of sperm. Only a handful have been discovered in the human diet. Of these the most common is cottonseed oil. Although cottonseed oil is rarely sold as a vegetable oil, it is commonly used in manufactured snack foods.

A great deal of attention has recently been given to the fact that synthetic chemicals have exhibited estrogenic effects in laboratory studies. The chemicals most prominently cited are PCBs and DDT, both of which have been banned in the United States. Compared with phytoestrogens, the concern over synthetic estrogens may be somewhat overstated. The estrogenic effects from the phytoestrogens in our diet are an estimated 40 million times greater than those from synthetic chemicals. To date, however, there is no concrete evidence that either pose a risk to human health.

Commonly Consumed Foods Shown to Have Estrogenic Effects

BARLEY
CABBAGE
CAROB BEAN
CARROT
CELERY
CHICK PEA
CINNAMON
CLOVES
COCONUT OIL
COFFEE BEAN
COMMON OAT
CORN
CUMIN
FENNEL
FIELD BEAN
GARLIC
GINSENG
GRAPEFRUIT JUICE
GREEN PEA
HOPS
KIDNEY BEAN
LIME
LIQUORICE
OLIVE OIL
PALM OIL
PARSLEY
PARSNIPS
PEANUT OIL
PINEAPPLE
PLUM
POMEGRANATE
POTATO
RHUBARB
RICE
SAFFLOWER OIL
SAGE
SESAME SEED
SOYBEAN
SUGAR BEET
SUNFLOWER OIL
WHEAT
WILD CHERRY
WILD GINGER
WILD LEEK

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THE SILENT SPRING

For sheep ranchers spring is a busy time. As the ewes in their flock begin to lamb there is a great deal of work to do to ensure the next generation of healthy sheep. But in the 1940s the sheep ranchers in Australia began to notice a peculiar and frightening trend. At first there was a rash of stillborn lambs. Then the ewes became sterile. Each spring there were fewer and fewer lambs. For the ranchers it was literally a silent spring. By the mid forties the sheep ranching industry in Australia was in a state of crisis and faced certain financial ruin unless the cause of the mysterious infertility in the sheep could be found. What could be causing this disastrous sterility? Genetic mutations? Radiation? Poisonous chemicals?

The Australian Department of Agriculture was called in. A cadre of veterinarians and scientists investigated all possible sources of the sterility. By 1946 they had discovered the source of the sheep’s sterility — clover.[1]

The innocuous looking clover had been producing large quantities of estrogen mimicking compounds.

Unbeknownst to the ranchers, the innocuous looking clover (*Trifolium subterraneum*) that they had recently begun planting in their fields to feed their sheep had been producing large quantities of estrogen mimicking compounds. It took another decade before scientists finally pinned down the exact chemicals which were causing the sterility, genistein and formononetin.[2] A structural comparison for genistein and formononetin shows why they were causing the sterility. They are surprisingly similar in structure to estrogen and synthetic estrogen, diethylstilbestrol, DES (see Figure 1). These two compounds mimic the steroidal nucleus of the natural female hormone estrogen. Although they are in fact rather weak estrogens, the plants make up for that fact by producing them in comparatively huge quantities: 5 percent of dry weight in the clover fodder.[3]

The estrogen mimicking nature of chemicals found in plants is not restricted to the clover *T. subterraneum*. A survey of clover found that 18 different species of the plant produced estrogen mimicking substances, or “phytoestrogens,” in quantities as high as those found in *T. subterraneum*.

Figure 1 — Isoflavonoids as Estrogenic Mimics

Source:Gerald A. Rosenthal and Daniel H. Janzen, Herbivores, Their Interaction with Secondary Plant Metabolites, *Academy Press 1979.*

Moreover, sheep are not the only animals on which the reproductive effects of phytoestrogens have been observed. Estrogenic effects also have been observed in quail which feed on pastures rich in leguminous species.[4] In years of good rainfall, legumes that are eaten grow luxuriously and are relatively low in phytoestrogens. However, in drought years the levels of phytoestrogens are increased with respect to the weight of the leaves. Consequently, egg laying by female quails is curtailed. There appears to be a self-regulating mechanism whereby the increase in quail population is kept low when food availability is limited. In other words, in order to enhance their survival, the plants reduce the number of quail in the next generation.

As the presence of phytoestrogens in clover and legumes became known, scientists began to wonder if other plants were also producing hormone mimickers which could disrupt the reproduction of animals. And even more importantly, were any of these plants in the human diet?

An answer to this question came from another area half way around the world from where scientists were struggling with the fertility of sheep, in Tibet. A clue emerged from the fact that in the history of Tibet the population has been extremely stable, often for as long as 200 years. During those times the Tibetan diet largely consisted of barley and peas. Could the peas or barley be affecting the fertility of the Tibetans?

A survey of clover found that 18 different species of the plant produced estrogen mimicking substances.

When scientists fed mice a diet consisting of 20 percent peas, litter sizes dropped by 50 percent. When the mice were fed diets consisting of 30 percent peas, the mice failed to produce any young at all.[5]

THE ORIGINAL CHEMICAL INDUSTRY

As the ability to detect, isolate, measure and test chemicals found in nature has progressed, a startling fact has emerged: plants are phenomenal chemical manufacturers. The Chemical Manufacturers Association (CMA) headquartered in Washington, D.C. can claim to have huge chemical companies like Dow and Dupont, but compared to the plant kingdom they are second rate amateurs. The botanical world is the original CMA.

For millions of years plants have been quietly producing chemicals. Through countless generations they have been perfecting a potpourri of chemicals for a host of objectives. Some chemicals are used to directly deter animals from eating the leaves or seeds of the plants. Strychnine, a very effective poison, comes from the seed of *Strychnos toxifera*. Hundreds of plants produce an astounding array of powerful poisons, from exotic tropicals such as curare' to the common flower foxglove, all deadly if eaten.

The botanical world is the original CMA.

Producing deadly toxins is a rather straight forward method for a plant to avoid becoming some animal's lunch. Some plants, however, have developed more subtle methods of chemical combat, not death but disruption — reproductive disruption.

Over the last few decades, scientists have been analyzing plants and the chemicals they produce. Most of this research is aimed at finding new and useful drugs. One consequence of all these studies has been that a large number of plants have been studied for their reproductive effects. A survey of literature compiled by the University of Illinois at Chicago's Department of Medical Chemistry, showed that 149 chemicals have been isolated from plants and have demonstrated estrogenic effects in laboratory studies. A total of 173 plants have been shown to have active estrogenic effects (see Appendix A).

Although much research has focused on estrogenic effects, antispermatogenic effects have also been studied. 55 compounds extracted from plants have been shown to inhibit the production of sperm. And 31 plant species have been tested and shown to be antispermatogenic (see Appendix B).

Table 1 — Lab Testing of Soybeans

Criteria	**Diet 1** (control)	**Diet 2** (genistein)	**Diet 3** (commercial soybean meal)
Total # of females	33	34	35
Avg. of young per female	4.9	3.2	4.5
% of females dropping litters	82	59	77
Avg. # of young per litter	6.0	5.4	5.8
Avg. weight per litter (g)	8.5	7.9	8.6

Source: M.W. Carter, Gennard Matrone, and W.W.G. Smart Jr., "Effect of Genistin on Reproduction of the Mouse," Journal of Nutrition, ***55**, p. 639-645. (1955)*

LAB TESTING OF PHYTOESTROGENS

The reproductive effect of low levels of genistein has been tested in laboratory mice beginning in the 1950s. Mice fed artificially elevated levels of genistein in the diet suffered decreased reproduction compared with control populations (see Table 1). In the control group, 82 percent of the females produced litters. A second group was fed a diet which consisted of 0.2 percent genistein. Of this second group, only 59 percent of the females produced litters.

Table 2 — Estrogen Effect of Vegetable Oil

Kind of Oil	Level Fed (%)	Mean Uterine wt (mg)
None (control)		9.5
Mineral	10	8.6
Castor	10	9.4
Cottonseed	10	10.1
Safflower	10	13.6
Wheat germ (sample 1)	10	13.6
Cod-liver	10	13.9
Corn	10	14.2
Linseed	10	14.6
Wheat germ (sample 2)	10	15.0
Peanut	10	15.9
Olive	10	16.7
Soybean (sample 1)	10	16.8
Soybean (sample 2)	10	17.7
Coconut	10	19.0
Rice bran	10	22.5

Source: A.N. Booth, E.M. Bickhoff and G.O. Kohler, "Estrogen-like Activity in Vegetable Oils and Mill By-products," Science, *June 1960, 1807-8.*

A third group was fed commercial soybean meal which contained 0.1 percent genistein, of which 77 percent of the females gave birth:[6]

Other early studies have also been conducted on the estrogenic effects of soybean oil. In these tests, the estrogenic effect was measured in terms of increased uterine weight in mice. One such study compared a host of different processed oils, both edible and non-edible. The oils were mixed into the feed to constitute ten percent of the diet. Of the two soybean oil samples used one increased the mean uterine weight 77 percent and the other 86 percent. Only two other oils produced more estrogenic responses in the mice, coconut oil and rice bran oil[7] (see Table 2).

Since these early studies, additional tests have continued to determine the reproductive effect of a number of phytoestrogens. Many of the most recent studies have used newborn mice to assess how these substances affect the development of the reproductive tract. One such study, of another common phytoestrogen, coumestrol, found that "Neonatal coumestrol treatment is effective in causing a number of morphological alterations in the female reproductive tract."[8]

Other studies have been conducted with other phytoestrogens in fetal and neonatal mice. One such study concluded, "Genistein influences estrogen-dependent development by modifying both morphological and neuroendocrine endpoints." In other words, phytoestrogens such as genistein and coumestrol act as endocrine disrupters.[9]

Many phytoestrogen containing plants are common elements of our diet.

PHYTOESTROGENS IN FOOD

Although hundreds of plants appear to produce endocrine disrupters, the concern for human exposure is limited principally to those plants in the human diet. Unfortunately, dozens of edible plants produce phytoestrogens and other endocrine disrupters. In laboratory tests, more than 43 foods found in the human diet have been shown to be estrogenically active (see Table 3).

Many phytoestrogen containing plants are common elements of our diet. Such grains as corn and wheat form a large part of our diet. For example, many legumes have shown a surprising capacity for phytoestrogen production, it was not unexpected that phytoestrogens would be found in the legumes we eat.

But the soybean has garnered particular attention. Soybeans are the third largest crop in the United States, Every year nearly 60 million acres of soybeans are planted in the United States alone. Nearly 2 billion bushels of soybeans are harvested every year.

Numerous studies have shown that soybeans contain significant levels of the phytoestrogens genistein and daidzein. Dry soybeans on average contain 1,107 milligrams of genistein per kilogram and 846 milligrams of daidzein per kilogram.[10] If one considers only the U.S. crop of soybeans and only the amount found in the seeds, the total quantity of genistein produced each year is roughly 130 million pounds, and an additional 100 million pounds of daidzein.

Table 3 — Commonly Consumed Foods From Plants, or Foods Containing Chemicals Shown to Have Estrogenic Actitivity in Laboratory Animals

WILD LEEK	ALLIUM AMPELOPRASUM (LILIACEAE)
GARLIC	ALLIUM SATIVUM (LILIACEAE)
PINEAPPLE	ANANAS COMOSUS (BROMELIACEAE)
PEANUT OIL	ARACHIS HYPOGAEA (LEGUMINOSAE)
COMMON OAT	AVENA SATIVA (GRAMINEAE)
SUGAR BEET	BETA VULGARIS (CHENOPODIACEAE)
CABBAGE	BRASSICA OLERACEA (CRUCIFERAE)
SAFFLOWER OIL	CARTHAMUS TINCTORIUS (COMPOSITAE)
CAROB BEAN	CERATONIA SILIQUA (LEGUMINOSAE)
CHICK PEA	CICER ARIETINUM (LEGUMINOSAE)
CINNAMON	CINNAMOMUM ZEYLANICUM (LAURACEAE)
COCONUT OIL	COCOS NUCIFERA (PALMAE)
COFFEE BEAN	COFFEA ARABICA (RUBIACEAE)
WILD GINGER	COSTUS SPECIOSUS (ZINGIBERACEAE)
CUMIN	CUMINUM CYMINUM (UMBELLIFERAE)
CARROT	DAUCUS CAROTA (UMBELLIFERAE)
PALM OIL	ELAEIS GUINEENSIS (PALMAE)
FENNEL	FOENICULUM VULGARE (UMBELLIFERAE)
SOYBEAN	GLYCINE MAX (LEGUMINOSAE)
LIQUORICE	GLYCYRRHIZA GLABRA (LEGUMINOSAE)
SUNFLOWER OIL	HELIANTHUS ANNUUS (COMPOSITAE)
BARLEY	HORDEUM VULGARE (GRAMINEAE)
HOPS	HUMULUS LUPULUS (CANNABACEAE)
OLIVE OIL	OLEA EUROPAEA (OLEACEAE)
RICE	ORYZA SATIVA (GRAMINEAE)
GINSENG	PANAX GINSENG (ARALIACEAE)
PARSLEY	PETROSELINUM CRISPUM (UMBELLIFERAE)
KIDNEY BEAN	PHASEOLUS VULGARIS (LEGUMINOSAE)
GREEN PEA	PISUM SATIVUM (LEGUMINOSAE)
WILD CHERRY	PRUNUS AVIUM (ROSACEAE)
PLUM	PRUNUS DOMESTICA (ROSACEAE)
POMEGRANATE	PUNICA GRANATUM (PUNICACEAE)
RHUBARB	RHEUM RHAPONTICUM (POLYGONACEAE)
SAGE	SALVIA OFFICINALIS (LABIATAE)
SESAME SEED	SESAMUM INDICUM (PEDALIACEAE)
POTATO	SOLANUM TUBEROSUM (SOLANACEAE)
WHEAT	TRITICUM AESTIVUM (GRAMINEAE)
FIELD BEAN	VICIA FABA (LEGUMINOSAE)
CORN	ZEA MAYS (GRAMINEAE)

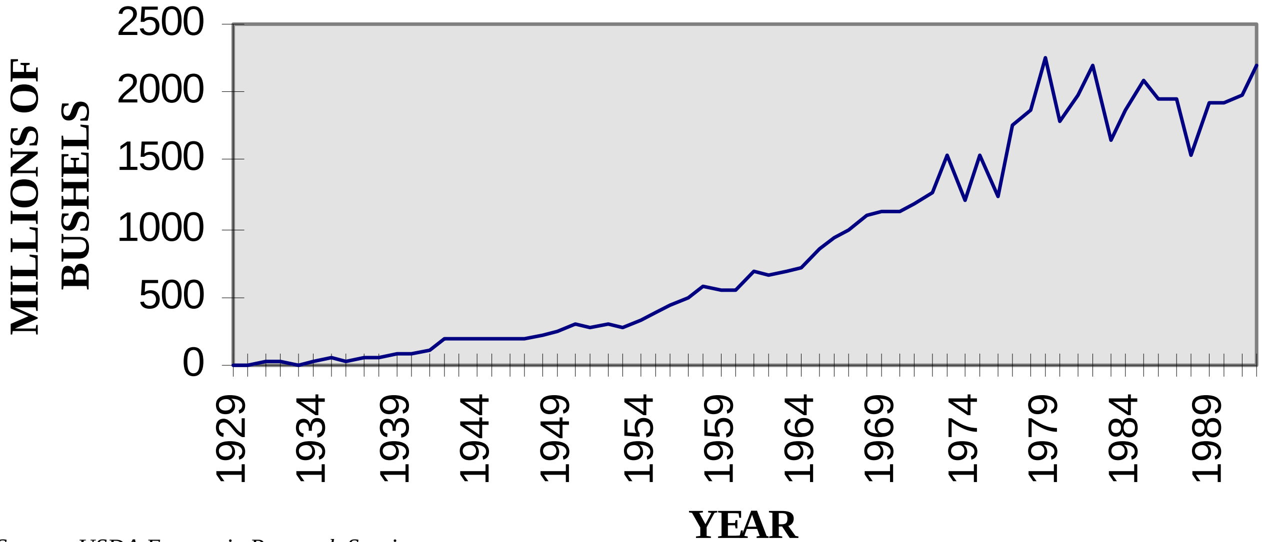
Although some of the soybeans end up as animal feed, much of the genistein and daidzein ends up on our dinner plates. When most people think of soybean foods they generally think of tofu or soy sauce. But soy products are not restricted to a few occasionally eaten foods: they are ubiquitous in the modern western diet. Soy flour, soy protein and most importantly soybean oil are found in hundreds of products eaten every day.

The Sprouting of The Bean

At the turn of the century, soybeans were a virtually unknown crop in the United States. By 1924, when the U.S. Department of Agriculture started publishing systematic annual statistics 448,000 acres of soybeans were harvested. Back then the yield per acre was also low, a mere 11 bushels per acre. Since then the acreage of soybeans harvested and the yield per acre have soared.

By the end of World War II, acreage had climbed to 10 million acres a year and the yield per acre had nearly doubled. As food processing

Figure 2 — U.S. Soybean Production



Source: USDA Economic Research Service.

modernized, more and more uses were found for the soybean and demand continued to increase. In 1979, soybean production peaked with 70 million harvested acres, producing 2.3 billion bushels. Since then production has continued to hover around 2 billion bushels a year (see Figure 2.)

Although they are ubiquitous in our diet, phytoestrogens are considered only weakly active.

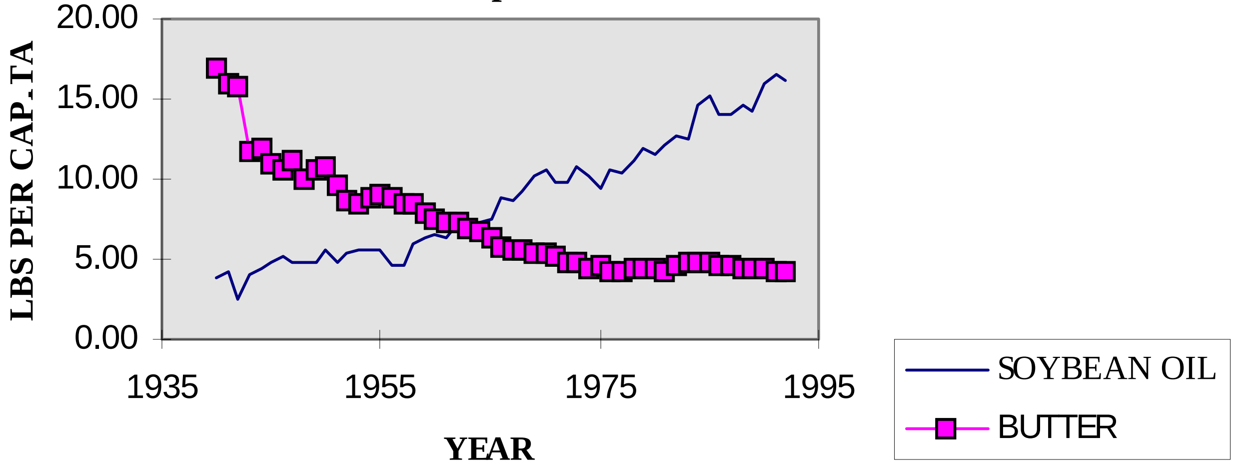
The majority of soybeans grown today are crushed for their oil. Soybean oil is by far the most common of the vegetable oils. Roughly 80 percent of the vegetable oil consumed in the United States is soybean oil. Annual per capita consumption of soybean oil is an estimated 65 pounds per year.[11] For instance, virtually all fried snack foods are manufactured with vegetable oil, predominantly soybean oil and is consumed more than butter. Virtually all infant formulas are a mixture of dairy and soy solids and proteins (see Figure 3).

ANTISPERMATOGENS FOUND IN FOOD

Although much more work has been done on phytoestrogens, some work has been done on plant chemicals which are known to effect the production of sperm. Only a handful have been discovered in the human diet (see Table 4). Of these the most common is cottonseed oil. Although cottonseed oil is rarely sold as a vegetable oil, it is commonly used in manufactured snack foods.

Since the 1970s most of the work on cottonseed oil as an antispermatogenic has been conducted in China, after Chinese authorities noted decreased fertility in some provinces in the late 1950s and eventually

Figure 3 — Per Capita Soybean Oil v. Butter Consumption in the United States



Source: USDA Economic Research Service.

linked it with the use of cottonseed oil in cooking.[12] Since then laboratory and human experiments have been conducted using cottonseed oil. In one study rats fed 0.5 milliliters of cottonseed oil a day produced no viable sperm at the end of the 28-day test period.[13]

XENO-ESTROGENS: CAUSE FOR ALARM?

A great deal of attention has recently been given to the fact that synthetic chemicals have exhibited estrogenic effects in laboratory studies. The chemicals most prominently cited are PCBs and DDT, both of which have been banned in the United States. Compared with the phytoestrogens, the concern over human exposure to synthetic estrogen mimicking compounds may be somewhat overstated.

Although they are ubiquitous in our diet, phytoestrogens are considered only weakly active. Most of the compounds which have been identified and tested have been found to have a relative potency when compared to synthetic estrogen of 0.001 to 0.0001.[14] In other words it takes between 1,000 and 10,000 molecules of these phytoestrogens to create the same effect as one synthetic estrogen molecule. The vast majority of phyto-estrogens appear to oc-

Table 4 — Commonly Consumed Foods From Plants or Containing Chemicals Shown To Have Antispermatogenic Activity In Laboratory Experiments

GARLIC	ALLIUM SATIVUM (LILIACEAE)
CELERY	APIUM GRAVEOLENS (UMBELLIFEREA)
COTTONSEED OIL	GOSSYPIUM HIRSUTUM (MALVACEAE)
SUNFLOWER SEED OIL	HELIANTHUS ANNUUS (COMPOSITAE)
COFFEE	CAFFEINE (ALKALOID)
TOBACCO	NICOTINE (ALKALOID)
GRAPEF	KAEMPFEROL (FLAVONOID)
CHOCOLATE	THEOBROMINE (ALKALOID)
TEA	THEOBROMINE (ALKALOID)
COLA	THEOBROMINE (ALKALOID)

Table 5 — Natural vs. Synthetic Estrogens

Source of Estrogens	Estrogen Equivalents (ug/day)
Birth Control Pill	18,675
Postimenopausal therapy	3,350
Phytoestrogens in food	102
Xeno-estrogens	0.0000025

Source: Steven Safe, Environmental Health Perspectives, *Vol. 103 No. 4., April 1995.*

cur in the family of chemicals known as flavonoids. Total flavonoid consumption in the human diet is estimated at approximately 1 gram per day.[15] Consequently the total daily estrogenic effect of phytoestrogens could be estimated at roughly 100 micrograms of estrogen equivalents (see Table 5).

Synthetic estrogen mimics or xeno-estrogens are considered even less estrogenically active than the phytoestrogens. Estrogenically active pesticides such as DDT, Dieldrin, and Endosulfan have been assigned a relative potency of 0.000001.[16] In other words it takes one million xeno-estrogen molecules to have the same effect as one synthetic estrogen molecule. Since total intake of these xeno-estrogens is significantly lower than the naturally occurring phytoestrogens (around 2.5 micrograms per day), the estrogen exposure from synthetic chemical has been estimated at 0.0000025 micrograms of estrogen equivalents per day.[17] In other words, the estimated estrogenic effects from the phytoestrogens in our diet are 40 million times greater than those from synthetic chemicals, but it is questionable that either is impacting human health.

Synthetic estrogen mimics or xeno-estrogens are considered even less estrogenically active than the phytoestrogens.

PLANT DEFENSE THEORY

A number of hypotheses have been developed to explain the functioning of plant defensive systems. Two of these hypotheses in particular bear examination. Because defenses are costly to produce:

1. Less well defended individuals have higher fitness than more highly defended individuals, when enemies are absent.
2. Commitment to defense is decreased when enemies are absent and increased when attacked.

A sizable body of literature has been accumulated which supports both of these hypotheses.[18] For example, varieties of insect resistant soybean produce a lower yield of seeds and accumulate nitrogen more slowly than insect susceptible varieties in the absence of herbivores including insects.

In the case of the second hypothesis, numerous studies have been conducted actually measuring the increased amounts of secondary metabolites. In the species *Senecio jacobaea*, when half of the leaves were removed the plant responded within two days by increasing the amount of total leaf alkaloids and N-oxides in the remaining leaves, 40 to 47 percent.

Table 6—Effect Of Herbivore Or Simulated Herbivore Damage On Plant Defenses

Plant Species	Damage	Plant Response
Lycopersicon esculentum Tomato	Insect grazing/mechanical damage	Systemic increase in concentration of proteinase inhibitor, 12-100hrs
Solanum tuberosum Potato	Insect grazing/mechanical damage	Systemic increase in concentration of proteinase inhibitor, 12-100hrs
Beta vulgaris Beet	Infestation by beet fly	29-100% increase in mortality of beet fly, 24 days
Medicago sativa Alfalfa	Attacked by spotted aphid/ grazing	Lowered fertility in sheep, "Clover disease," 6 weeks
Senecio jacobaea Toyon	50% of leaves mechanically removed	Total leaf alkaloids and N-oxides increase 40-47% in undamaged leaves, 2 days
Carex aquatilis Sedge	Mechanical grazing	Total phenolics and proanthocyanidins increase 30-40%, and 40-50% respectively in undamaged foliage, 1 year
Betula pubescens Birch	Mechanical leaf damage	Total phenolics increase in neighboring leaves, 2 days

Source: Gerald A. Rosenthal and Daniel H. Janzen, Herbivores, Their Interaction with Secondary Plant Metabolites, *Academy Press 1979. p 19-21.*

In another case, when beet plants were infested by beet flies, within 24 days the mortality of the beet flies increased between 29 and 100 percent. The ability of beets to respond to infestation and in some cases kill all of the infesting insects shows the extreme effectiveness of some secondary plant metabolites and the plant's ability to defend itself[19] (see Table 6).

RISK V. RISK ANALYSIS FOR CROP PROTECTION

Current government policy only rarely regulates naturally occurring chemicals in the food supply. In only two cases has the Food and Drug Administration (FDA) directly regulated naturally occurring carcinogens. In the first instance, the FDA banned the chemical safrole, which had been used as a natural flavoring for root beer, after it was determined that safrole was carcinogenic in high-dose rat studies.

The second case of federal regulation of a natural chemical is aflatoxin. The FDA has established a standard of 20 part per billion of aflatoxin for a variety of foods susceptible to aflatoxin poisoning, most commonly peanuts.[20] In setting its aflatoxin standard the FDA engaged in a type of risk-versus-risk analysis.

The FDA recognized that it would be virtually impossible to eliminate aflatoxin from the food supply. To do so would have required the massive use fungicides which present other equally hazardous long term health effects as well as sacrifice portions of the food supply. The question which faced the agency was what level of aflatoxin contamination should be tolerated to maximize human health. Whether or not the agency came to the correct answer is difficult to determine. However, the fact that the agency considered the health effects of both aflatoxin and prophylactic fungicides and their effects on the food supply increases the chance of establishing a better health standard.

It is conceivable that restricting the use of technology might induce plants to produce more of their own chemical defenses, which may ultimately increase human health risks.

This type of risk-versus-risk analysis could benefit other regulatory programs affecting agriculture and the production of food. Current pesticide regulation, for example, does not take into account the potential of pesticides applied on crops to inhibit the formation of the plant's own secondary metabolites.

More importantly, as biotechnology and the ability to manipulate the genetic code of plants increases, the effects of secondary plant metabolites may increase geometrically. Recently the Environmental Protection Agency released its proposed rule on plant pesticides.[21]

The EPA is proposing to "exempt from Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) regulation those pesticides that are normally a component of (not new to) the plant." By failing to take into consideration the array of harmful chemicals that plants currently produce to defend themselves, federal policy may be focusing on only one side of the human health equation.

Technology, both chemical and genetic, when properly used should both protect the crop and keep the quantity of chemical harmful to humans to a minimum. It is conceivable that restricting the use of technology might induce plants to produce more of their own chemical defenses, which may ultimately increase human health risks.

A FINAL NOTE

More than fifty years after discovering the fertility problems in sheep in Australia, ranchers continue to struggle with clover disease. Despite decades of research and the attempted introduction of more benign types of clover, clover disease is still responsible for the loss of 1 million lambs every year.

Had clover disease struck herds of sheep in the middle ages or even in the 17th century, the source of this infertility would likely have been blamed on a witch. And no doubt if the infertility continued the shepherds would have rounded up some socially unpopular woman with few friends to defend her, held a mock trial, called in religious authorities to absolve their collective conscience and then burned her at the stake.

Although science has done much to dispel myths and witches, there still exists in humans the urge to blame the socially unpopular for the ills of humanity. In the modern sense it is easy for us to blame a witch, chemical or otherwise. But the responsible and far more laborious task is to determine what actually is causing the real problems that society faces.

ABOUT THE AUTHOR

Jonathan Tolman is an Environmental Policy Analyst at CEI. His most recent work has been focused on water pollution and agricultural issues. He is the author of "Federal Agricultural Policy: A Harvest of Environmental Abuse," and "Gaining More Ground: An Analysis of Wetland Trends in the United States." Prior to working with CEI, he was Associate Producer of the weekly television show *TechnoPolitics*. In 1991, Jonathan served as Special Assistant for the President's Council on Competitiveness, focusing on environmental and natural resource regulation. In 1992, he worked for the White House as an environmental analyst in the Office of Policy Development. Prior to coming to Washington, Jonathan worked as an Environmental and Chemical Analyst for Kennecott Copper Corp. in Salt Lake City, Utah. He received his bachelor of Sciences degree in political science from the University of Utah.

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[17]Safe, p. 349.

[18]Gerald A. Rosenthal and Daniel H. Janzen, *Herbivores, Their Interaction with Secondary Plant Metabolites* (New York: Academic Press 1979), p. 14.

[19]*Ibid*, p. 22.

[20]"Aflatoxin Controls Effective, GAO Tells Congress," *Food Chemical News*, May 27, 1991.

[21]Environmental Protection Agency, "Plant Pesticides Subject to the Federal Insecticide, Fungicide and Rodenticide Act and the Federal Food Drug and Cosmetic Act, Proposed Policy," *Federal Register*, November 23, 1994, p. 60496.